Exponent®

Center for Health Sciences

Expert Report of Ellen T. Chang, Sc.D.

In the matter of

State of Minnesota, et al., vs. 3M Company

Court File No. 27-CV-10-28862

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Prepared for

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Abbreviations

ADHD	Attention deficit/hyperactivity disorder
ALP	Alkaline phosphatase
ALT	Alanine aminotransferase
AST	Aspartate aminotransferases
ATSDR	Agency for Toxic Substances and Disease Registry
BMI	Body mass index
CDC	Centers for Disease Control and Prevention
COPD	Chronic obstructive pulmonary disease
DNA	Deoxyribonucleic acid
eGFR	Estimated glomerular filtration rate
Et-PFOSA-AcOH	2-(*N*-ethyl-perfluorooctane sulfonamido) acetate
GFR	Glomerular filtration rate
GGT	Gamma glutamyl transferase
HDL	High-density lipoprotein
HOMA-beta	Homeostatic model assessment of beta cell function
HOMA-IR	Homeostatic model assessment of insulin resistance
IARC	International Agency for Research on Cancer
Ig	Immunoglobulin
LDL	Low-density lipoprotein
Me-PFOSA-AcOH	2-(*N*-methyl-perfluorooctane sulfonamido) acetate
MMR	Measles, mumps, and rubella
NRC	National Research Council
NTP	National Toxicology Program
PFAS	Perfluoroalkyl and polyfluoroalkyl substances
PFBA	Perfluorobutanoic acid
PFBS	Perfluorobutane sulfonate
PFC	Perfluorochemical
PFDA	Perfluorodecanoic acid
PFDS	Perfluorodecanoic sulfonate
PFdoDA	Perfluorododecanoic acid
PFHpA	Perfluoroheptanoic acid
PFHpS	Perfluoroheptane sulfonate
PFHxA	Perfluorohexanoic acid
PFHxS	Perfluorohexane sulfonate
PFNA	Perfluorononanoic acid
PFOA	Perfluorooctanoic acid, also called “C8”
PFOS	Perfluorooctane sulfonate

PFOSA	Perfluorooctane sulfonamide
PFPA	Perfluoropentanoic acid
PFteDA	Perfluorotetradecanoic acid
PFtrDA	Perfluorotridecanoic acid
PFunDA	Perfluoroundecanoic acid
RNA	Ribonucleic acid
RR	Relative risk
SEER	Surveillance, Epidemiology, and End Results
T3	Triiodothyronine
T4	Thyroxine
TSH	Thyroid-stimulating hormone
U.K.	United Kingdom
U.S.	United States
U.S. EPA	United States Environmental Protection Agency
VLDL	Very low-density lipoprotein
WHO	World Health Organization
WMA	World Medical Association

1.0 Introduction and Scope

I have been asked by counsel for the defendant in this matter, 3M Company, to summarize the state of the epidemiologic evidence on the human health effects of perfluoroalkyl and polyfluoroalkyl substances (PFAS), especially perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorobutanoic acid (PFBA), and perfluorobutane sulfonate (PFBS),[1] and to opine on whether the existing evidence from epidemiologic studies is sufficient to establish causal associations between exposure to specific PFAS and the development of specific adverse human health outcomes.

PFAS are synthetic fluorinated chemicals that have been used in a broad range of industrial and commercial surfactant and polymer applications since the late 1940s. Applications of PFAS include textile stain, soil, and water repellents; grease-proof food-contact paper; non-stick cookware; aqueous film-forming foams for extinguishing petroleum-product-based fires (Buck et al. 2011). PFOA and PFOS, which previously were the most widely produced PFAS and have been the most extensively studied in the scientific literature, are no longer manufactured in the United States (U.S.), with a few exceptions for limited industrial purposes (U.S. EPA 2017b). This reduction process began in 2000, when 3M Company announced a voluntary phase-out of PFOA, PFOS, and PFOS-related products; this process was mostly completed by the end of 2002 (U.S. EPA 2000, 3M Company 2017). Subsequently, in 2006, the U.S. Environmental Protection Agency (EPA) invited eight major PFAS companies (including 3M Company) to participate in its global PFOA Stewardship Program, which involved making a commitment to reduce by 95% facility emissions and product content levels of PFOA, precursor chemicals that can break down to PFOA, and related higher homologue chemicals, and complete removal of these chemicals by 2015 (U.S. EPA 2017a).

According to the Complaint, the State of Minnesota alleges that Defendant 3M Company caused pollution of Minnesota ground and surface water with PFAS, and that PFAS (referred to in the Complaint as perfluorochemicals, or PFCs) "pose serious risks to human health and the environment." With respect to particular human health outcomes related to PFAS exposure, the Complaint states that "a recent study by 3M of its employees suggested a positive association 'between PFOA exposure and prostate cancer, cerebrovascular disease, and diabetes.'" Other specific human health outcomes allegedly associated with or caused by PFAS exposure are not identified in the Complaint.

[1] PFOA, PFOS, PFBA, and PFBS are the four specific PFAS listed in the Amended Complaint dated January 18, 2011 ("Complaint"). For simplicity, I refer throughout this report to specific perfluoroalkyl carboxylic and sulfonic acids and their salts (e.g., ammonium perfluorooctanoate, the ammonium salt of PFOA) synonymously.

In the State of Minnesota's amended Responses to 3M Company's Interrogatories No. 18 and 19, dated August 28, 2017, the State alleges that "at least 89 scientific studies published after June 11, 2012 (the date of the State's original responses), [document] the adverse human health effects of PFCs." Among the outcomes claimed to be associated with exposure to certain PFAS are hereditary prostate cancer,[2] ulcerative colitis,[3] cerebral palsy in boys,[4] decreases in the effectiveness of certain vaccines, including those against mumps, rubella, and diphtheria,[5] and decreased lung function among children with asthma.[6]

[2] The reference cited in the State's amended Responses to Interrogatories is Hardell et al. 2013.

[3] The reference cited in the State's amended Responses to Interrogatories is Steenland et al. 2013.

[4] The reference cited in the State's amended Responses to Interrogatories is Liew et al. 2014.

[5] The references cited in the State's amended Responses to Interrogatories are Stein et al. 2016 and Grandjean et al. 2016.

[6] The reference cited in the State's amended Responses to Interrogatories is Qin et al. 2017.

2.0 Summary of Opinions

My main opinions in this matter are as follows:

- The weight of epidemiologic evidence regarding a potential causal relationship between a specific exposure and a specific human health outcome should be assessed based on the entire body of relevant literature, taking into account study quality and considerations including the strength, consistency, temporality, exposure-response gradient, and plausibility of a causal effect.
    - Plaintiffs' experts Dr. Philippe Grandjean and Dr. Jamie DeWitt selectively cite epidemiologic studies to corroborate their opinions, thereby omitting a large body of scientific evidence that does not support a causal effect of exposure to PFOA, PFOS, or other PFAS on adverse human health outcomes.
- The overall weight of the relevant epidemiologic evidence is not sufficient to demonstrate causal associations between exposure to specific PFAS, including but not limited to PFOA, PFOS, PFBA, and PFBS, and the development of specific adverse health outcomes in humans, whether in general communities, PFAS-contaminated communities, or occupational settings.
    - Health outcomes for which causal associations with PFAS exposure have not been established include health endpoints considered by the C8 Science Panel (defined in Section 6.0) to have "probable links" to PFOA exposure (namely, testicular cancer, kidney cancer, diagnosed high cholesterol, thyroid disease, pregnancy-induced hypertension, and ulcerative colitis); the additional endpoints listed in the Complaint and State's amended Responses to Interrogatories, including prostate cancer and hereditary prostate cancer, cerebrovascular disease, diabetes, cerebral palsy in boys, decreased effectiveness of certain vaccines, and decreased lung function among children with asthma; and other health endpoints, including other cancers, body size, fetal growth, lipid levels, overall mortality, and conditions affecting various other organs and organ systems, including bone and connective tissue, cardiovascular, immune, hematological, renal (kidney), hepatic (liver), metabolic, neurodevelopmental, neurological, male and female reproductive, respiratory, and thyroid.
    - The existing published epidemiologic evidence base is insufficient to establish causal associations between specific PFAS and specific human health outcomes because associations reported in the scientific literature are inconsistent, and because many studies are methodologically unreliable for demonstrating causal relationships. These problems include cross-sectional or retrospective design, error-prone assessment of exposures and/or health outcomes, inadequate assessment of and control for confounders and underlying physiological

influences, potential bias due to differential study participation or data collection, and multiple hypothesis testing. Because of these and other limitations, confounding, bias, chance, and in some cases reverse causality cannot be excluded as plausible explanations for observed associations.

- Due to their inability to detect individual-level associations, which may differ substantially from group-level associations, ecologic studies cannot demonstrate causal effects.
    - Plaintiff's expert Dr. David Sunding's ecologic analyses of associations between residence in certain communities in Washington County, Minnesota, and various health outcomes, including low birth weight, premature birth, birth rate, cancer incidence, and cancer mortality, are not a valid scientific basis on which to conclude that exposure to PFAS in drinking water caused adverse human health effects.

This report summarizes my work performed to date for this matter, including evaluation of the epidemiologic evidence base and related material, and presents the findings resulting from that work. The findings presented herein are made to a reasonable degree of scientific certainty. I reserve the right to supplement this report and to expand or modify opinions based on review of additional material as it becomes available.

3.0 Qualifications

I am a Senior Managing Scientist at Exponent, Inc., an international science and engineering consulting company, where I work as an epidemiologist in the Center for Health Sciences. I am also a member of the Stanford Cancer Institute, a National Cancer Institute Designated Comprehensive Cancer Center. I earned my undergraduate degree at Harvard College in 1998 and my doctorate degree (Doctor of Science, Sc.D.) in epidemiology with a minor in biostatistics from the Harvard School of Public Health in 2003. I completed a post-doctoral fellowship in medical epidemiology and biostatistics at the Karolinska Institute in Stockholm, Sweden, in 2005.

Prior to joining Exponent, I was a research scientist at the Cancer Prevention Institute of California, where I conducted original research studies on cancer epidemiology and performed cancer surveillance research at a National Cancer Institute Surveillance, Epidemiology, and End Results (SEER) population-based cancer registry. I was also the Chief Epidemiologist at the Asian Liver Center at Stanford University, where I conducted community-based research on hepatitis B and liver cancer awareness, detection, prevention, and management. From 2005 to 2016, I was a Consulting Assistant Professor in the Division of Epidemiology, Department of Health Research and Policy at the Stanford University School of Medicine.

My main research interests are the epidemiology, surveillance, and prevention of cancer and other chronic diseases. I have conducted epidemiologic studies of a wide range of exposures in association with risk of cancer and other chronic diseases, including PFAS, air pollution, occupational exposures, infections, immunological biomarkers, medication use, reproductive factors, physical activity, body size, diet and nutrition, alcohol consumption, tobacco smoking, family structure, personal and family medical history, and genetic variation. As a consultant, I have worked on numerous matters involving the evaluation of potential health risks to communities from exposure to environmental contaminants.

I have published more than 160 peer-reviewed scientific articles and reviews and 11 book chapters; these include two systematic literature reviews on the epidemiology of PFOA and PFOS exposure in relation to risk of cancer and immunological health conditions in humans (Chang et al. 2014, Chang et al. 2016). My publications are listed in my *curriculum vitae*, which is attached to this report as Appendix B. Cases in which I have testified in the past four years are listed in Appendix C. My employer, Exponent, is compensated at an hourly rate of $300 for my work on this matter.

4.0 Documents Reviewed

In addition to the references cited in the body of this report and listed in section 13.0 (References Cited), and materials listed in section 14.0 (Other Documents and Information Considered), I reviewed the following case-specific materials.

- Amended Complaint, State of Minnesota, et al., vs. 3M Company, dated January 19, 2011
- Plaintiff State of Minnesota's Amended Responses to Defendant 3M Company's Interrogatories No. 18 & 19, dated August 28, 2017
- Declaration of Gary Berscheid (STATE_07072844), dated June 2, 2017
- Declaration of Melissa Furch (STATE_07103400), dated June 2, 2017
- Deposition Transcript of Larry Zobel, M.D., August 22, 2017
- Deposition Transcript of Helen Goeden, Ph.D., August 23, 2017
- Deposition Transcript of Jeffrey Mandel, M.D., August 24, 2017
- Deposition Transcript of Jessica Nelson, Ph.D., August 30, 2017
- Expert Report of Jamie C. Dewitt, Ph.D., dated September 22, 2017
- Expert Report of Philippe Grandjean, M.D., D.M.Sc., dated September 22, 2017
- Expert Report of Jessica Schmor, R.N., CCS, CPC, CHCAF, AHFI, CLNC, dated September 22, 2017
- Expert Report of David L. Sunding, Ph.D., dated September 22, 2017
- Minnesota Department of Health birth data, STATE_07507028–07507088 and STATE_07513050–07513089
- Minnesota Department of Health death records and aggregated cancer incidence data, STATE_07507028–07507088, STATE_07512741–07513049, and STATE_07513090–07513464
- 89 studies and other documents on PFCs and health effects, STATE_07513465–07516558
- Deposition Transcript of Jessica Schmor, R.N., October 5, 2017
- Deposition Transcript of Jamie C. DeWitt, Ph.D., October 10, 2017
- Deposition Transcript of Philippe Grandjean, M.D., DMSc, October 24, 2017
- Draft Deposition Transcript of Dr. David L. Sunding, Ph.D., October 31, 2017

5.0 Introduction to Epidemiology

5.1 Basic Epidemiologic Principles

Epidemiology is the scientific study of the distribution and determinants of diseases in populations. Epidemiologic research is required to measure disease occurrence, to identify the causes of specific health outcomes in humans, to assess the contribution of various causal factors to the occurrence of diseases that can be induced by multiple agents, and to determine exposure-response relationships between causes and human health effects.

This introductory section identifies and defines some technical terms that are used by epidemiologists to describe the design, results, and interpretation of epidemiologic studies. Many of these terms are used later in this report to describe the results of epidemiologic studies pertaining to this matter.

5.1.1 Measures of Disease Frequency

Epidemiologists use several standard measures to report the frequency of occurrence (incidence or prevalence) of disease, death, or another health outcome in a population; these measures are defined below.

Incidence: The occurrence of a new health event in an individual, or the risk of developing a specific health outcome during a given time period among individuals without a history of that health outcome; incidence is influenced by factors that cause a specific health outcome.

Prevalence: The existence of new and pre-existing health events, or the risk of having a specific health outcome at a moment or period in time; prevalence is influenced by factors that cause a specific health outcome and factors that affect the duration of the outcome (e.g., survival and treatment).

Incidence rate: The rate at which new health events arise in a population, i.e., the number of individuals who newly develop a specific health outcome in a given population during a given time interval, divided by the average number of persons at risk (i.e., without a history) of that health outcome in the population multiplied by the length of the specified time period.

Mortality rate: The rate at which deaths from all causes or from a specific cause occur in a population, i.e., the number of all or cause-specific deaths that occur in a given population during a given time interval, divided by the average number of living persons in the population multiplied by the length of the specified time period.

Prevalence proportion: The percentage of people in a defined population who have a specific health outcome at a given time, i.e., the number of existing cases of a specific health outcome in a given population at a given moment or interval in time, divided by the total number of persons in the population at the time or midway through that time period.

5.1.2 Epidemiologic Study Designs

Epidemiologists use a variety of study designs to evaluate hypotheses regarding associations between specific exposures and health outcomes; these study designs are described below.[7]

Proportionate mortality or morbidity study: A comparison of the proportion of deaths (or disease, in a morbidity study) due to a specific cause between a group defined by a particular exposure, such as an occupation, and an unexposed referent group; this type of study generally is not relied upon for causal inference because conclusions can be invalid if comparison populations have different distributions of causes of death/disease for reasons (generally unknown) that are not related to the exposure of interest.

Ecologic study: A comparison between populations or groups, rather than individuals; this type of study generally is not relied upon for causal inference because associations observed at the group level may not be valid at the level of individual persons.

Cross-sectional study: A comparison of the prevalence of a specific health outcome across levels of a specific exposure in study subjects (or vice versa), with the exposure and outcome both measured at a given time, providing a "snapshot" of the association between the exposure and the health outcome at one time; this type of study cannot establish causal relationships because exposures and health outcomes are assessed simultaneously, such that the temporal sequence of a cause preceding an effect cannot be demonstrated.

Case-control study: A comparison of specific past exposures between study subjects with the health outcome of interest ("cases") and a suitable group of subjects without the disease ("controls"); in this type of study, exposure assessment usually (but not always) occurs after onset of the health outcome in cases, i.e., retrospectively.

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[7] A *case report or case series* is a description of an individual or individuals with a specific exposure or health outcome. Case reports and case series are not analytic epidemiologic studies because they lack a comparison group and, therefore, cannot provide estimates of relative risk or risk differences. They can generate a hypothesis that an exposure may be related to a health outcome, but cannot test an association and cannot be used to establish causality due to the absence of a comparison group.

Cohort study: A comparison of incidence or mortality rates of a specific health outcome between study subjects with various levels of a specific exposure who are observed over time, often for years; in this type of study, exposure assessment may be based on information collected prior to onset of the health outcome (prospectively) or reconstructed from existing information collected prior to the onset of the health outcome in most or all subjects (retrospectively).

Randomized controlled trial: A comparison of health outcomes between participants who are randomly (by chance) assigned to receive one of two or more specific interventional exposures; this type of study generally is used to test the efficacy of a drug, medical device, surgical procedure, or public health intervention in a well-defined patient population; however, randomized controlled trials are logistically and/or ethically infeasible for many exposures of interest, and generally are restricted to narrowly defined exposures, short time periods, and highly selected populations.

5.1.3 Measures of Association

Relative risk (RR) is a general term that refers to the ratio of the probability of a health outcome in subjects who are exposed, compared with those who are not (or, in a case-control study, the probability of exposure in cases compared with controls). An RR equal to 1.0 signifies that the probability of the health outcome is equal in exposed and unexposed subjects; it can be referred to as "no association" or a "null association." An RR greater than 1.0 signifies that the probability of the health outcome is higher in exposed than unexposed subjects; it can be referred to as a "positive association." An RR less than 1.0 signifies that the probability of the health outcome is lower in exposed than unexposed subjects; it can be referred to as a "negative association" or an "inverse association." Measures of RR include odds ratios, prevalence ratios, hazard ratios, rate ratios, risk ratios, and standard incidence or mortality ratios, some of which are described below. Other statistical measures that typically accompany measures of risk are also described in this section.

Odds ratio: A measure of RR in a case-control study that compares the odds (probability divided by 1 – probability) of exposure vs. non-exposure between cases and controls.

Standardized mortality ratio: A measure of RR from a retrospective cohort study that compares the number of observed deaths from all causes or a specific cause in exposed study subjects to the number of expected deaths based on mortality rates (usually specific to age, sex, and calendar period) in a reference population (often the general population in a defined geographic area).

Standardized incidence ratio: A measure of RR from a retrospective cohort study that compares the number of observed cases of a specific health outcome in exposed study subjects to the number of expected cases based on incidence rates (usually specific to age, sex, and calendar period) in a reference population (often the general population in a defined geographic area).

Confidence interval: A measure of variability or precision, i.e., a margin of error around a point estimate; with repeated sampling of the same underlying study population and in the absence of bias, contains the value of the point estimate of interest with a frequency no less than the stated confidence level (usually set at 95% by convention); the range of results expected 95% of the time if samples for new studies were repeatedly drawn from the underlying study population. By convention, a "statistically significant" result is one where the 95% confidence interval for an RR excludes the null value of 1.0; it does *not* provide the range in which the correct or true point estimate lies.

P value: The probability, in the absence of bias, of observing the result of a hypothesis test or a more extreme result if the null hypothesis is true (typically, that the exposure and health outcome of interest are not associated); the probability that the observed difference or a more extreme difference occurred due to chance. By convention, a "statistically significant" result is one where $p < 0.05$; a p value does *not* denote the probability that the study result is correct or true.

5.2 Interpretation of Epidemiologic Associations

The detection of a statistical association, even a statistically significant one, between a specific exposure and a specific health outcome in an epidemiologic study does not necessarily indicate that the exposure caused the outcome. Causal inference based on statistical associations must take into account potential explanations for a spurious (non-causal) association. These alternative explanations, which include bias, confounding, and chance, are defined below.

Bias: Any methodological deviation from the truth in the design, conduct, or analysis of a study that can lead to invalid results; also referred to as systematic error.

Selection bias: A systematic difference between study participants and non-participants in characteristics that are associated with the exposure and health outcome of interest; in a case-control study, this bias results from a difference in study participation or data completeness between cases and controls due to reasons related to the exposure under investigation; in a cohort study, this bias results from a difference in study follow-up or data completeness between exposed and unexposed subjects due to reasons related to the health outcome under investigation.

Information bias: A systematic difference in data quality or validity between comparison groups; this category includes *recall bias*, a systematic difference in accuracy or completeness of recollected or reported information (e.g., exposure history) between comparison groups (e.g., cases and controls); *interviewer bias*, a systematic difference in accuracy or completeness of data collection by study staff between comparison groups; and *response bias*, a systematic difference in the voluntary completion of survey data between comparison groups.

Confounding: A distortion of the estimated exposure-outcome association due to the influence of an additional factor, referred to as a confounder, that is associated with the exposure of interest, independently associated with the health outcome of interest, and not on the causal pathway (i.e., not an intermediate) between the exposure and the outcome of interest; a confounder is an alternative risk factor, but not necessarily a cause, that can create spurious associations between an exposure and an outcome if not measured in adequate detail and sufficiently controlled by statistical adjustment or study design restrictions.

Chance: Random error resulting from variability in sampling a finite number of study subjects from a complete underlying population or from imprecise measurement of exposures, outcomes, or confounders. The probability of chance as an explanation for a statistical association can be reduced, but not completely eliminated, by increasing study size, improving accuracy and precision of study measures, or increasing the number of measures taken.

A well-known contemporary example of confounding is that of a spurious protective association, reported in several large, carefully conducted epidemiologic studies, between combined (estrogen and progesterone) menopausal hormone therapy and heart disease. This example helps to illustrate the pitfalls of drawing causal conclusions based largely on modest statistical associations from observational epidemiologic studies. The reported inverse association, bolstered by a plausible biological hypothesis, was interpreted as causal and led postmenopausal hormone therapy to become one of the most prescribed therapies in the U.S. (Herrington and Howard 2003). However, subsequent large randomized trials, in which potential confounders are distributed equally between treatment groups, thus eliminating confounding, showed no protective effect of menopausal hormone therapy on heart disease. A likely explanation for the earlier findings in observational studies is residual confounding by lifestyle factors (Barrett-Connor 2004, Kuller 2004, Lawlor et al. 2004, Petitti 2004, Vandenbroucke 2004). That is, women who took menopausal hormone therapy were more likely to be affluent and have healthier lifestyles, and consequently had lower rates of heart disease, than women who did not take menopausal hormone therapy. Without controlling adequately for such lifestyle factors, a lower risk of heart disease might improperly be attributed to menopausal hormone therapy. Moreover, combined menopausal hormone therapy is known

to cause breast and endometrial cancers (IARC 2012), illustrating the potential public health risks of interpreting confounded associations as causal.

Besides minimizing the possibility of bias, confounding, and chance as explanations for a given statistically significant association observed in an individual epidemiologic study, establishing whether an association is causal also requires an evaluation of the overall weight of the scientific evidence from relevant epidemiologic studies, taking into account evidence from relevant toxicological and mechanistic studies. A set of general guidelines for evaluating causality was formulated by Sir Austin Bradford Hill (Hill 1965); these guidelines or "viewpoints" are commonly used by epidemiologists and accepted in the scientific community and in legal settings (Cole 1997, Rothman et al. 2008, NRC 2011). Hill's nine considerations are described briefly below.

Strength: Magnitude of the observed association between the exposure and the health outcome of interest; all else being equal, strong associations are less likely than weak associations to be due to confounding or bias.

Consistency: Repeated observation of an association between the exposure and the health outcome of interest by different investigators across different study settings; repetition helps to reduce the probability of chance as an explanation for an observed association.

Specificity: Limitation of an observed association to a single exposure and a single health effect; absence of specificity does not necessarily reduce the likelihood of causality.

Temporality: Sequence by which a cause must precede an effect in time.

Biological gradient: Exposure-response trend by which occurrence of the health outcome increases in accordance with greater exposure.

Plausibility: Credibility of a causal relationship based on current knowledge in toxicology, biology, and other fields; depends on present scientific evidence, which is subject to change.

Coherence: Accordance of a causal relationship with the known natural history and biology of the health outcome of interest; depends on present scientific evidence, which is subject to change.

Experiment: Evidence from studies with controlled or quasi-controlled exposures, such as interventions or preventive actions; such evidence often is absent or not applicable.

Analogy: Comparability to similar exposure-outcome associations; often can be postulated to support or oppose a causal relationship, and does not necessarily enhance or reduce the likelihood of causality.

These guidelines are not intended to be used in a checklist fashion to determine whether an observed association is likely to be causal. As specified by Hill, "None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*. What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question – is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?"[8] Hill also indicated that these guidelines were intended for the evaluation of *statistically significant* associations: "Our observations reveal an association between two variables, perfectly clear-cut and *beyond what we would care to attribute to the play of chance*. What aspects of that association should we especially consider before deciding that the most likely interpretation of it is causation?" (Hill 1965) (emphasis added). Hill cautioned that "formal tests of significance … contribute nothing to the 'proof' of our hypothesis," yet a clear association must exist before the relationship can be hypothesized to be causal.

Throughout the remainder of this report, observed statistical associations are evaluated in terms of whether bias, confounding, and chance can sufficiently be excluded as likely non-causal explanations, and the overall weight of the epidemiologic evidence on associations between PFAS exposure and specific health outcomes is considered in accordance with the framework of the Hill guidelines.

[8] Logically, a cause must precede an effect in time; therefore, a temporal sequence whereby an exposure precedes a health outcome is necessary for causation.

6.0 The C8 Science Panel and Its "Probable Link" Reports

The former C8 Science Panel was a group of three epidemiologists commissioned by the West Virginia Circuit Court in 2005, as part of a class action settlement in a lawsuit involving PFOA releases from DuPont's Washington Works plant in Parkersburg, West Virginia, to conduct a community study in the Mid-Ohio Valley to evaluate whether there is a "probable link" between PFOA exposure and any human disease (C8 Science Panel 2017). A "probable link" in this legal context was defined as a "more likely than not" connection between PFOA exposure and a particular human disease among class members; it was not conceived to denote a necessarily causal relationship between PFOA and disease (C8 Science Panel 2012a). Nevertheless, the C8 Science Panel's statements often are cited inaccurately as if they were definitive scientific conclusions in favor of causal effects of PFOA on human diseases. Therefore, this section addresses the appropriate interpretation of the "probable link" reports in light of their basis on epidemiologic studies that did not establish causal associations.

The C8 Science Panel issued 17 "probable link" reports addressing 47 human diseases, and reached the conclusion that there was a "probable link" between PFOA exposure and six diseases: diagnosed high cholesterol, ulcerative colitis, thyroid disease, kidney cancer, testicular cancer, and pregnancy-induced hypertension (C8 Science Panel 2012a). For ulcerative colitis and pregnancy-induced hypertension, the Panel's conclusions on epidemiology were based exclusively on their research study in the Mid-Ohio Valley community, since no other relevant epidemiologic studies on these outcomes had been published at that time. Thus, the consistency of these associations across study populations could not be evaluated. For the other health endpoints, the Panel acknowledged that other epidemiologic studies outside of the Mid-Ohio Valley yielded different results. The Panel also recognized that, "given the many diseases [they were] studying, some may appear to be associated with exposure simply through chance, but [they had] to judge these associations individually and acknowledge the uncertainty inherent in making these judgments" (C8 Science Panel 2012c, 2012e).

The community study conducted by the C8 Science Panel, called the C8 Health Project, has methodological limitations that prevent a causal interpretation of its findings. First, several analyses—including those that served as the basis for several "probable link" conclusions (e.g., Steenland et al. 2009; Frisbee et al. 2010; Knox et al. 2011; Vieira et al. 2013) —were cross-sectional in nature, meaning that exposures and health outcomes were assessed simultaneously. As discussed earlier, some observed cross-sectional relationships may be due to reverse causation (i.e., an effect of a health endpoint or its treatment, such as cancer or chemotherapy, on behavioral patterns that influence PFOA exposure, or on the uptake, distribution, and/or

excretion of PFOA). Other observed cross-sectional relationships may be the result of factors affecting both the health endpoint and the underlying pharmacokinetics of PFOA, such as glomerular filtration rate (discussed further below). Thus, results of such studies cannot on their own establish causal relationships.

Second, many of the estimated associations (including those that served as the basis for "probable link" conclusions) relied on estimated, rather than measured, cumulative serum PFOA levels, which had an unknown degree of estimation error for years prior to study baseline in 2005–2006. Estimates of past PFOA exposure were based on PFOA emission and dispersion data, individual residential history and water consumption, and a physiologically-based pharmacokinetic model for PFOA (Shin et al. 2011a, Shin et al. 2011b). A comparison with measured serum PFOA concentrations in 2005–2006 revealed an overall correlation coefficient of 0.67 between predicted and observed median concentrations, indicating a substantial degree of estimation error (Shin et al. 2011b). The correlation coefficient was 0.82 (where 0 indicates no linear correlation, 1.0 indicates a perfect positive linear correlation, and -1.0 indicates a perfect negative linear correlation) for people who had lived and worked in one of six public water districts for 5 years before serum sampling and provided information on their water consumption; however, the correlation coefficient was 0.32 for those who had not lived and worked in one of the water districts for 5 consecutive years prior to sampling. Since serum PFOA levels were not measured prior to 2005–2006, the validity of the model for estimating past PFOA exposure could not be evaluated, but the model could have been poorer if demographic, residential, occupational, and water-consumption information reported by study subjects was less accurate for past years than for the present. Error in exposure estimation could have differed systematically by disease status if residential relocation patterns, water consumption, or other model covariates were associated with health characteristics; this pattern would have resulted in an unpredictable direction and magnitude of bias.

Third, health outcomes could have been misclassified substantially in some of the C8 Health Project studies (again including several that served as the basis for "probable link" conclusions), because those studies relied at least in part on self-reported health data (e.g., Lopez-Espinosa et al. 2012; Barry et al. 2013; Steenland et al. 2013; Winquist and Steenland 2014a, 2014b). Outcome classification error could have differed by PFOA exposure status if individuals who lived in residential districts known to have higher PFOA levels in drinking water were more likely to report certain health outcomes or, in studies where positive self-reports were validated through medical records review, if they were more likely to consent to such review, thereby resulting in overestimated associations.

Fourth, selection bias due to unequal participation rates, both at baseline and in follow-up questionnaires and tests, could have biased results from the C8 Health Project. The estimated participation rate in the original C8 Health Project was 80% (81% for adults) (Frisbee et al.

2009). Among participating adults, 74% consented to further study contact, and 82% of those (61% of original participants) completed one or two follow-up surveys in 2008–2011, when health outcomes such as cancer, autoimmune disease, and thyroid disease were ascertained (Barry et al. 2013, Steenland et al. 2013). Systematic differences in participation rates by exposure and health status could have led to biased estimates of association.

Fifth, although some of the analyses in the C8 Health Project included statistical adjustment for several covariates, uncontrolled or residual confounding still could have occurred due to incomplete control of crudely classified covariates (e.g., smoking, which generally was classified simply as current/former/never or yes/no) or lack of adjustment for other factors potentially associated with PFOA exposure and the health outcomes of interest (e.g., diet and occupation).

Finally, especially in light of the numerous hypotheses tested, including many that lacked strong *a priori* hypotheses to support an association with PFOA exposure, chance is a potential explanation for any statistically significant associations detected in the C8 Health Project. Only after extensive research, with attempts at replication of results and exclusion of alternative explanations, can one determine whether a particular result is probably explained by chance. Associations of PFOA with diagnosed high cholesterol, ulcerative colitis, thyroid disease, kidney cancer, testicular cancer, and pregnancy-induced hypertension across published epidemiologic studies are described in subsequent sections of this report. Given the inconsistent findings between the C8 Health Project and other, independent epidemiologic studies, as well as the sparse research on pregnancy-induced hypertension and ulcerative colitis, chance cannot reliably be excluded as a plausible explanation for any of the reported statistical associations.

In 2010, the three epidemiologists who comprised the C8 Science Panel published a review of the epidemiologic evidence on human health effects of PFOA, including studies of lipids, uric acid, cardiovascular disease, cerebrovascular disease, diabetes, cancer, immune function, thyroid function, sex hormones, liver function, kidney function, and reproductive and developmental outcomes (Steenland et al. 2010a). At that time, the C8 Science Panel concluded that the overall epidemiologic literature on PFOA was “limited in volume and quality.” They noted, “Many studies are cross-sectional in nature, making causal inference difficult. Other studies are small and have too few outcome events to draw firm conclusions.” Regarding the C8 Health Project, the authors wrote: “Although there are methodologic challenges in this setting as well, particularly in reconstructing historical exposure levels and in ascertaining health end points accurately, these studies should provide new evidence complementary to that which has been generated to this point.” Thus, the C8 Science Panel acknowledged the limitations of cross-sectional data collection, the ever-present potential for confounding, misclassification in retrospective exposure estimation, and potentially inaccurate or incomplete outcome ascertainment.

In summary, the associations designated by the C8 Science Panel as "probable links" are based in large part on results from the C8 Health Project, in which observed statistical associations can plausibly be explained by confounding, bias, or chance. Moreover, the associations are inconsistently detected across the available epidemiologic studies of PFOA, including those published after the "probable link" reports were issued in 2012—as discussed subsequently in this report. Therefore, these associations cannot reliably be interpreted as demonstrating causal relationships between PFOA and human health outcomes.

7.0 Systematic Literature Search for Epidemiologic Studies of PFAS and Human Health

As a basis for their opinions in this matter, Dr. Grandjean and Dr. DeWitt (to the extent that she relies upon epidemiologic evidence) selectively cite studies that found statistically significant associations between exposure to PFOA, PFOS, or other PFAS and risk of various human health outcomes, thereby omitting numerous studies that found non-significant results or even statistically significant associations in an inverse (protective) direction. Likewise, within studies that found a combination of significant and non-significant associations, they selectively present significant results, thereby omitting a multitude of hypothesis tests that indicated no significant relationship between PFAS exposure and various health endpoints.

A reliable scientific opinion on whether causation has been established for a given exposure-outcome association should be based on the entirety of the relevant scientific evidence, rather than a selected, non-representative subset of that evidence. In general, the peer-reviewed, published scientific literature on a given exposure-outcome association comprises a publicly available, transparent, replicable basis on which to evaluate the body of scientific evidence. (It should be noted, however, that the tendency to favor publication of statistically significant positive findings over null findings has created a body of published literature that is skewed toward positive associations (Ioannidis 2005, Kavvoura et al. 2007, Ioannidis 2008, Ioannidis et al. 2011, Kivimaki et al. 2014).)

Dr. Grandjean and Dr. DeWitt do not describe how they searched for and identified the studies that they cite in their reports. To identify the relevant epidemiologic literature on PFAS and human health published in peer-reviewed scientific journals, I used the following search terms to conduct a systematic literature search in PubMed, a publicly available resource developed and maintained by the U.S. National Library of Medicine that provides access to over 26 million citations for biomedical literature:

> *(PFOA OR perfluorooctanoate OR perfluorooctanoic OR PFOS OR perfluorooctane OR perfluorooctanesulfonic OR perfluorooctanesulphonic OR PFBA OR perfluorobutanoic OR perfluorobutanoate OR perfluorobutyrate OR perfluorobutyric OR PFBS OR perfluorobutane OR perfluorobutanesulfonic OR perfluorobutanesulphonic OR nonafluorobutanesulphonic OR PFAS OR perfluoroalkyl OR polyfluoroalkyl OR perfluorinated OR perfluorochemical*)*
>
> *AND*

(health OR disease OR illness* OR cancer* OR death* or mortality OR risk OR worker* OR employ* OR cohort OR case-control OR cross-sectional OR epidemiol* OR human* OR humans OR person* OR persons OR infants OR newborn* OR neonat* OR children OR child* OR adolescents OR adults OR men OR women OR subjects OR communit*).*

This literature search was designed to provide the basis for a comprehensive, non-selective review of the entirety of the available epidemiologic literature on PFOA and human health published in scientific journals.[9] I did not include unpublished studies because they cannot be searched comprehensively or systematically.

From a total of 3,573 articles identified using these search terms as of August 2017, as well as from examination of the reference lists of relevant articles, I identified relevant studies based on whether they evaluated exposure to specific PFAS in association with any specific human health outcome, including incidence, prevalence, and mortality, and reported a measure of RR or sufficient data to calculate an RR. I focused on original epidemiologic studies that analyzed and reported on their own data, rather than review articles that summarized the results of previously published studies. I did not include animal or mechanistic studies because they are outside the scope of this report, which focuses on human epidemiology.

Tables 1–19 of this report (Appendix A) briefly summarize the results of epidemiologic studies of exposure to PFAS in association with body size, bone and connective tissue conditions, cancer, cardiovascular outcomes, fetal growth, immune and hematological conditions, kidney outcomes, lipid levels, liver outcomes, metabolic conditions, all-cause mortality, neurodevelopmental outcomes in children, other neurological conditions, pregnancy-related hypertension, female reproductive outcomes, male reproductive outcomes, respiratory outcomes, thyroid conditions, and various other health outcomes. Studies are summarized by study design, study setting, age group, maximum study size, PFAS types evaluated, health outcomes for which any statistically significant associations were detected (focusing on the most highly adjusted reported results, that is, associations adjusted for the most confounders, where statistical significance is defined, in accordance with scientific convention in epidemiology, based on a p value < 0.05), and health outcomes for which no statistically significant associations were detected. Although the tables do not fully capture the methods and results of each study, and they omit much of the information that I considered in evaluating and interpreting each study, they help to illustrate the consistency of statistical findings across the available epidemiologic literature. Throughout this report, I use the words “significant” and

[9] These search terms were intended to identify all relevant articles; however, there is a possibility that some relevant articles may have been missed by this literature search.

"significantly" to refer only to statistical significance; I do not use these words to imply importance.

Within each of the health outcome categories, it is important to evaluate associations with each specific health endpoint separately, unless a direct correspondence or shared physiological mechanism has been established between endpoints (e.g., height and weight are directly related to body mass index (BMI), which is defined as $\frac{weight\ (kg)}{height\ ^2(m^2)}$; chronic bronchitis and emphysema are both strongly associated with tobacco smoking, and often coexist). Thus, significant associations with two different health endpoints within the same category cannot necessarily be interpreted as being consistent with each other, especially in the absence of data demonstrating a shared causal pathway.

8.0 Epidemiology of PFAS and Specific Human Health Outcomes

8.1 Epidemiology of PFAS and Body Size

A causal effect of exposure to PFOA, PFOS, or other PFAS on body size in humans has not been established by the available published epidemiologic studies. I identified 38 published studies that reported the association between specific PFAS and body size, including BMI, waist circumference, overweight or obesity, and other anthropometric measures in various study populations and age groups (Olsen et al. 1998, Olsen et al. 1999, Olsen et al. 2000, 2003a, So et al. 2006, Olsen and Zobel 2007, Sakr et al. 2007a, Costa et al. 2009, Lin et al. 2009, Rylander et al. 2009, Andersen et al. 2010, Nelson et al. 2010, Eriksen et al. 2011, Shankar et al. 2011a, Halldorsson et al. 2012, Maisonet et al. 2012, Shankar et al. 2012, Wang et al. 2012, Andersen et al. 2013, Vested et al. 2013, Lin et al. 2013a, Barry et al. 2014, Timmermann et al. 2014, Cariou et al. 2015, Kataria et al. 2015, Mattsson et al. 2015, Høyer et al. 2015b, Alkhalawi et al. 2016, Ashley-Martin et al. 2016, Braun et al. 2016, Domazet et al. 2016, Jaacks et al. 2016, Wang et al. 2016, Christensen et al. 2016a, Chen et al. 2017, Hartman et al. 2017, Karlsen et al. 2017, Mora et al. 2017) (Table 1 in Appendix A). Four of these studies evaluated body size in infants (≤ 12 months), 10 in children (~1–12 years), five in adolescents (~13–19 years), and 25 in adults (≥ 20 years). Nineteen studies were cross-sectional, one was a retrospective cohort study (in which past exposure information was estimated after the occurrence of the outcome), and 18 included at least a prospective study design component (i.e., exposure information was collected prior to the occurrence of the outcome). Most studies (n = 26) were conducted in general community settings where PFAS exposure would be expected to be at background levels. Three were conducted at least partially in fishing communities[10]; one was conducted in a PFAS-contaminated community (in this case, the Mid-Ohio Valley[11]), where PFAS exposure was expected to be higher; and eight were conducted at least in part in occupational settings (with one study also including residents of the Mid-Ohio Valley community, and another study also including residents of the surrounding PFAS-contaminated community in Changshu City, Jiangsu Province, China), where PFAS exposure would have been the highest.

To illustrate the anticipated discrepancy in exposure levels among study settings, Figures 1 and 2 show median or geometric mean serum PFOA and PFOS levels, respectively, in chemical

[10] Fishing communities may have higher exposure to PFAS (as well as other persistent organic pollutants) because seafood is a major source (Christensen et al. 2016a).

[11] Other PFAS-contaminated communities investigated in epidemiologic studies of other health outcomes included communities in Changshu City, Jiangsu Province, China; Guiyu Town, Shantou City, Guangdong Province, China; and Shenyang City, Liaoning Province, China.

plant workers; a PFOA-contaminated community (the Mid-Ohio Valley region where the C8 Health Project was based); Washington County, Minnesota; and the general U.S. population (Olsen et al. 2003b, Olsen and Zobel 2007, Sakr et al. 2007a, Sakr et al. 2007b, ATSDR 2008, Frisbee et al. 2009, Olsen et al. 2012b, Landsteiner et al. 2014, CDC 2017). Typical exposure levels in occupational settings are one to two orders of magnitude greater than those in PFAS-contaminated community settings, which in turn are roughly one order of magnitude greater than background levels in general communities. Thus, any adverse health effects of PFAS exposure would be anticipated to be most apparent in occupational settings and least apparent in general community settings.

The body size metric studied most frequently in relation to PFAS exposure was BMI, whether as a continuous variable or dichotomized at overweight (≥ 25 kg/m^2) or obesity (≥ 30 kg/m^2). Among infants and children, several studies assessed height and weight z-scores (i.e., standard deviation scores, where the value is the number of standard deviations below or above a reference mean or median value).

In the four studies of infants, all based in general community settings, PFOA, PFOS, and other PFAS (perfluorohexane sulfonate (PFHxS), measured in one study) were not consistently significantly associated with body size measures, including weight, weight z-score, length, length z-score, BMI, and ponderal index (g/cm^3). Most reported associations were statistically non-significant. PFOA was significantly associated with smaller body size among boys but not girls in the Danish general population (Andersen et al. 2010), but not in studies based in China, Germany, and Taiwan (So et al. 2006, Alkhalawi et al. 2016, Chen et al. 2017). PFOS was significantly associated with lower weight and BMI among girls but not boys in the Taiwan general population (Chen et al. 2017), but not in other studies (So et al. 2006, Andersen et al. 2010, Alkhalawi et al. 2016).

In the 10 studies of children, including eight studies in general community settings (Maisonet et al. 2012, Andersen et al. 2013, Timmermann et al. 2014, Braun et al. 2016, Wang et al. 2016, Chen et al. 2017, Hartman et al. 2017, Mora et al. 2017) and two based at least partly in fishing communities (Høyer et al. 2015b, Karlsen et al. 2017), PFOA, PFOS, and other PFAS (PFHxS, perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluoroundecanoic acid (PFunDA), perfluorododecanoic acid (PFdoDA)) were not consistently significantly associated with body size measures such as BMI, height, weight, waist circumference, skinfold thickness, and body fat percentage. Most reported associations were statistically non-significant, and two studies reported no significant associations between PFOA or PFOS and body size measures (Andersen et al. 2013, Timmermann et al. 2014). Whereas some studies found that levels of PFOA, PFOS, and other PFAS were significantly associated with measures of smaller body size in a minority of associations tested (Wang et al. 2016, Hartman et al. 2017), others found some significant associations with measures of larger body size (Maisonet et al. 2012, Høyer et al.

2015b, Braun et al. 2016, Mora et al. 2017), and still others found an assortment of positive and negative significant associations with body size measures depending on the specific PFAS evaluated (Chen et al. 2017, Karlsen et al.).

The five studies of body size in adolescents also yielded inconsistent findings, with predominantly statistically non-significant associations. Reported statistically significant associations included associations of PFOA, PFOS, and other PFAS (PFHxS, PFNA, or combinations of PFAS) with smaller body size measures among adolescents in general communities in the U.S. and Taiwan (Lin et al. 2009, Nelson et al. 2010, Lin et al. 2013a, Kataria et al. 2015); and both positive and inverse associations among adolescents in the general population of Odense, Denmark (Domazet et al. 2016).

Among the 25 studies of adults, associations with measures of body size—most often BMI, as well as overweight or obesity, waist circumference, skinfold thickness, and gestational weight gain for pregnant women— also were inconsistent. Most associations with PFOA, PFOS, and other PFAS (PFHxS, perfluoroheptanoic acid (PFHpA), perfluoroheptane sulfonate (PFHpS), perfluorooctane sulfonamide (PFOSA), 2-(*N*-methyl- or *N*-ethyl-perfluorooctane sulfonamido) acetate (Me- and Et-PFOSA-AcOH), PFNA, PFDA, PFunDA, and PFdoDA) were statistically non-significant, and 11 studies reported no significant associations between PFOA, PFOS, or other PFAS and adult body size measures in general community, contaminated community, and occupational settings (Olsen et al. 1998, Olsen et al. 2000, So et al. 2006, Costa et al. 2009, Rylander et al. 2009, Shankar et al. 2011a, 2012, Wang et al. 2012, Vested et al. 2013, Barry et al. 2014, Cariou et al. 2015). In the remaining studies, reported significant associations of PFAS with body size were positive (Olsen et al. 1999, Olsen et al. 2003a, Olsen and Zobel 2007, Sakr et al. 2007a, Halldorsson et al. 2012, Mattsson et al. 2015, Ashley-Martin et al. 2016, Jaacks et al. 2016), inverse (Lin et al. 2009, Eriksen et al. 2011, Lin et al. 2013a, Christensen et al. 2016a), or a mixture of both (Nelson et al. 2010, Domazet et al. 2016).

Two recent literature reviews on the epidemiologic evidence for associations between PFAS exposure and health outcomes in children concluded that no consistent association has been observed with childhood body size. One review, written by authors from U.S. EPA, concluded, "The evidence for effects on weight or BMI in children across PFAS is mixed with PFOA most frequently associated with overweight status in females but some PFOA studies also show null results" (Rappazzo et al. 2017). In the other review, regarding the relationship between PFAS exposure and childhood growth and obesity, the authors stated: "Because of the few number of studies in children and inconsistencies in findings, we have classified evidence as 'insufficient'" (Vrijheid et al. 2016).

Overall, no consistent associations were demonstrated between exposure to any specific PFAS and any measure of body size in any age group, and no clear pattern of association was evident

by study setting (i.e., stronger associations in occupational environments than in fishing or contaminated communities, and stronger associations in the latter than in general communities). Many estimated associations were adjusted for few or no confounders. In particular, estimated associations with any given PFAS generally were not adjusted for the potential confounding influence of other PFAS or other persistent organic pollutants, which often were correlated with each other. Consequently, positive associations with one PFAS often were accompanied by positive associations with other PFAS in the same study, while inverse associations with multiple PFAS tended to cluster within a study. That is, without adjusting for confounding by other PFAS and persistent organic pollutants, separate associations with each specific substance could not be distinguished. (This issue of collinearity between various PFAS also affects many other studies described in this report, especially those conducted in general community settings.)

Another shortcoming in most of these studies, as well as most others described in this report, is that when PFAS levels were measured in serum, plasma, or whole blood, this exposure was measured at only one point in time. That single measurement may not have been representative of long-term or etiologically relevant exposure to PFAS, thereby resulting in misclassification of exposure. This problem is particularly acute in cross-sectional studies, in which exposure levels are susceptible to reverse causality, since body size can influence dietary habits, which can in turn affect PFAS exposure (Domingo and Nadal 2017). Moreover, levels of some PFAS in serum, plasma, or whole blood may not reflect levels in organ-specific tissues (Perez et al. 2013). To the extent that reliance on circulating PFAS levels results in misclassification of more etiologically relevant tissue-specific PFAS levels, estimated associations with various health outcomes could be biased in either direction (i.e., overestimation or underestimation) (Thomas 1995, Weinberg et al. 1995, Jurek et al. 2005, Jurek et al. 2008, Ogburn and VanderWeele 2012).

Nevertheless, the lack of consistent associations remains after restriction to studies with a prospective design, where PFAS exposure was measured prior to measurement of body size. No single measure of body size stands out as having a unique association with PFAS exposure; instead, associations of a given PFAS with various body size measures tend to be in the same direction (positive, inverse, or null). The overall lack of consistent findings, combined with methodological limitations of the available studies, leads to the conclusion that a causal effect of PFOA, PFOS, or other PFAS on body size has not been established based on published epidemiologic studies.

Figure 1. Median (or geometric mean) serum levels of PFOA (1,000 ng/mL = 1 µg/mL = 1 part per million) measured among workers at the DuPont plant in Parkersburg, West Virginia, or 3M plants in Cottage Grove, Minnesota, Decatur, Alabama, and Antwerp, Belgium; among community members in the Mid-Ohio Valley and Washington County, Minnesota; and among general population members or blood donors in the U.S.

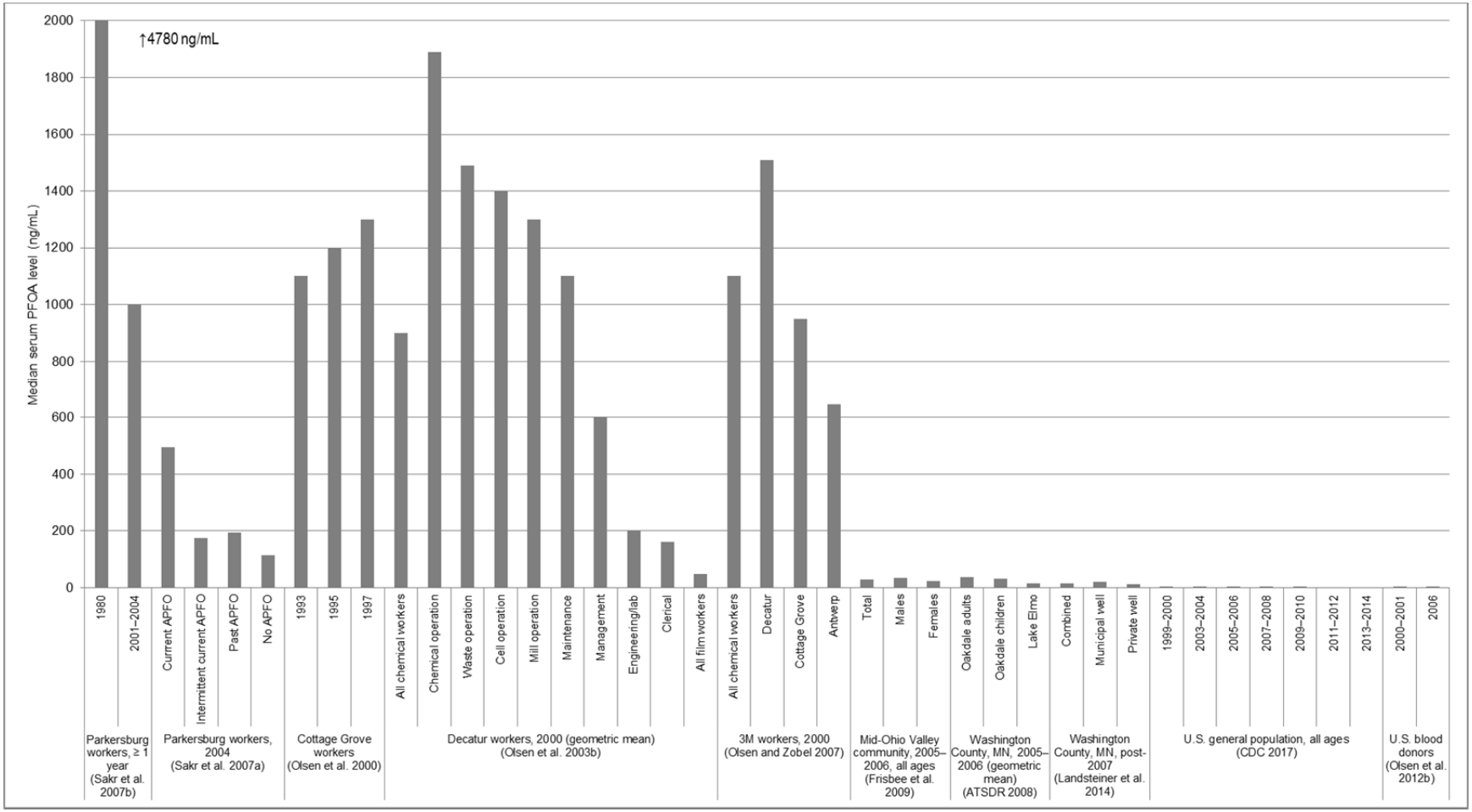
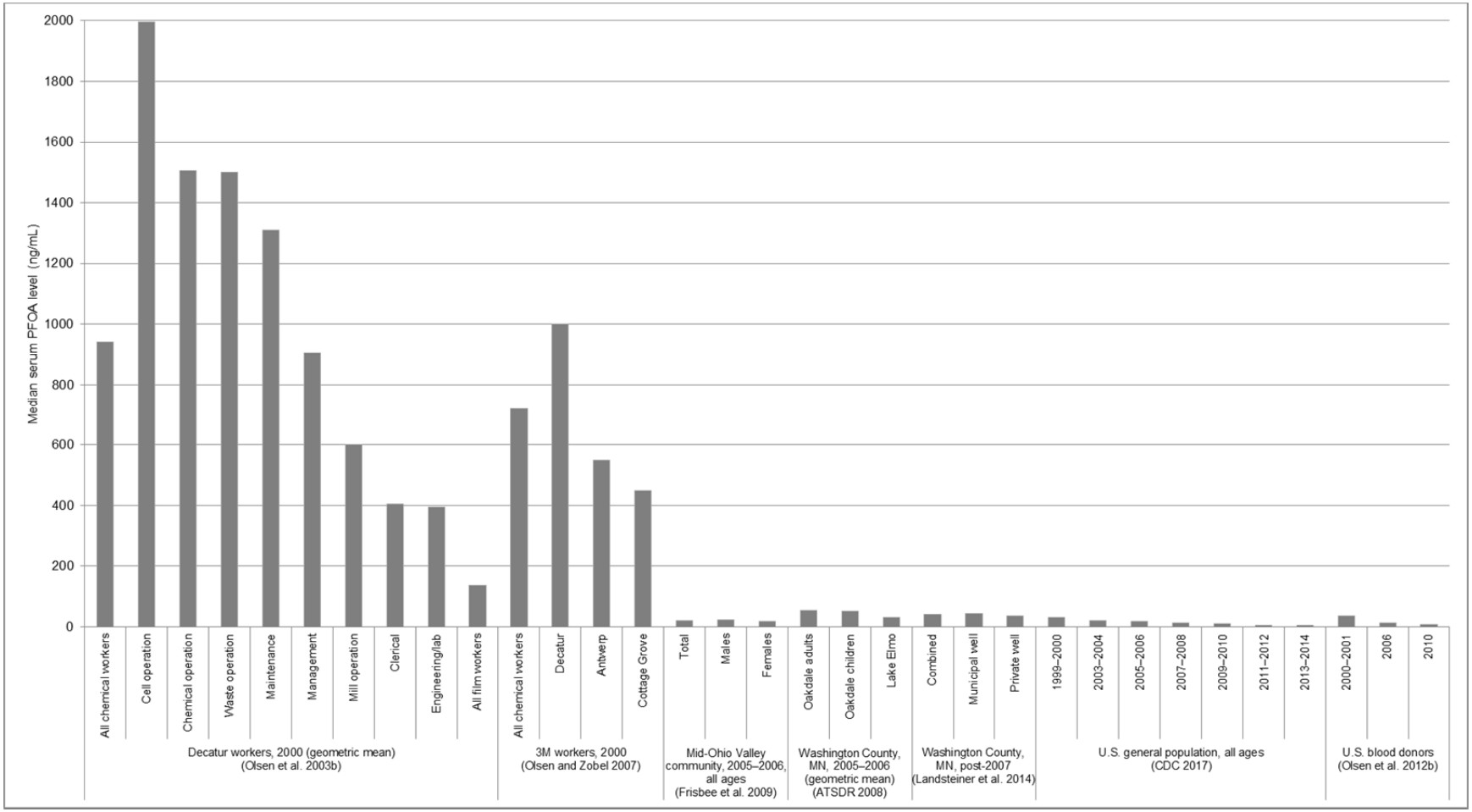


Figure 2. Median (or geometric mean) serum levels of PFOS (1,000 ng/mL = 1 µg/mL = 1 part per million) measured among workers at 3M plants in Cottage Grove, Minnesota, Decatur, Alabama, and Antwerp, Belgium; among community members in the Mid-Ohio Valley and Washington County, Minnesota; and among general population members or blood donors in the U.S.



8.2 Epidemiology of PFAS and Bone or Connective Tissue Conditions

A causal effect of exposure to PFOA, PFOS, or other PFAS on bone or connective tissue conditions in humans has not been established by the available published epidemiologic studies. I found six published studies that reported the association between specific PFAS and bone or connective tissue conditions, including osteoarthritis, bone mineral density, bone fracture, osteoporosis, and expression of genes related to parathyroid hormone, a hormone involved in bone remodeling, among adults in different study settings (Melzer et al. 2010, Innes et al. 2011, Lin et al. 2014, Galloway et al. 2015, Steenland et al. 2015, Khalil et al. 2016) (Table 2 in Appendix A). Five of these studies were cross-sectional, and one was a retrospective cohort study. Three studies were conducted in a general community with expected background PFAS exposure levels, two were conducted in the Mid-Ohio Valley, and one was conducted among PFOA plant workers in the Mid-Ohio Valley.

One of three studies that examined risk of osteoarthritis found a significant positive cross-sectional association with PFOA exposure in the Mid-Ohio Valley community, especially among non-obese adults and those under 55 years (Innes et al. 2011). However, a cross-sectional study in the general U.S. population found no positive exposure-response association between PFOA and osteoarthritis risk (Melzer et al. 2010), and an occupational retrospective cohort study reported no significant association (Steenland et al. 2015). The first study found a significant inverse association between PFOS exposure and osteoarthritis risk (Innes et al. 2011), whereas the second found no significant association with PFOS exposure (Melzer et al. 2010).

One of two cross-sectional studies of bone mineral density, bone fracture, and osteoporosis in the general U.S. adult population found some significant associations of PFHxS, PFOA, PFOS, and PFNA exposure with lower bone mineral density, especially in the total femur and lower femoral neck, as well as a higher risk of osteoporosis in women (Khalil et al. 2016). By contrast, the other study found generally non-significant associations between PFOA or PFOS exposure and total hip and lumbar spine bone mineral density (except between PFOS and lower lumbar spine bone mineral density among premenopausal women but not postmenopausal women or men), and no significant associations with all, hip, wrist, spine, or hip/wrist/spine fractures (Lin et al. 2014).

Only one study evaluated associations between PFOA or PFOS exposure and parathyroid hormone gene expression, which was examined among women in the Mid-Ohio Valley community (Galloway et al. 2015). Associations with expression of the parathyroid hormone gene, parathyroid hormone-like gene, and parathyroid hormone 2 gene were statistically non-significant, while PFOA and PFOS levels were significantly inversely associated with

expression of the parathyroid hormone 2 receptor gene. These findings have not been replicated elsewhere.

Overall, no consistent associations were detected in multiple studies of PFAS and various bone and connective tissue conditions among adults, and no pattern of association was observed by study setting. All but one of the studies were cross-sectional, resulting in temporal ambiguity regarding the sequence of the exposure and the health outcome, and observed associations could have been due to confounding, bias, or chance. In light of the lack of consistent findings, the small number of independent studies of each association, and the methodological problems of those studies, a causal effect of PFOA, PFOS, or other PFAS on bone or connective tissue conditions has not been established by published epidemiologic studies.

8.3 Epidemiology of PFAS and Cancer

A causal effect of exposure to PFOA, PFOS, or other PFAS on any type of cancer in humans has not been established by the available published epidemiologic studies. I retrieved 25 published studies that reported the association between specific PFAS and cancer, including several studies of multiple major site-specific cancer types in humans, and some studies of single cancer sites (Ubel et al. 1980, Gilliland and Mandel 1993, Alexander et al. 2003, Olsen et al. 2004, Alexander and Olsen 2007, Grice et al. 2007, Leonard et al. 2008, Eriksen et al. 2009, Lundin et al. 2009, Pirali et al. 2009, Vassiliadou et al. 2010, Bonefeld-Jørgensen et al. 2011, Steenland and Woskie 2012, Barry et al. 2013, Consonni et al. 2013, Vieira et al. 2013, Bonefeld-Jørgensen et al. 2014, Ghisari et al. 2014, Hardell et al. 2014, Innes et al. 2014, Raleigh et al. 2014, Steenland et al. 2015, Christensen et al. 2016a, Ghisari et al. 2017, Wielsøe et al. 2017) (Table 3 in Appendix A). These included three cross-sectional studies, six retrospective case-control studies, 12 retrospective cohort studies, and three prospective case-cohort or case-control studies. Six studies were set in a general community, four in a fishing community, two among residents of the contaminated Mid-Ohio Valley community, and 13 (one of which also included Mid-Ohio Valley community residents) in an occupational setting.

The cancer sites identified by the C8 Science Panel as having a "probable link" to PFOA exposure—namely, kidney cancer and testicular cancer (C8 Science Panel 2012b)—were found not to demonstrate a consistent, statistically significant positive association with exposure to PFOA in the studies that evaluated this association. (Associations between exposure to other specific PFAS and kidney cancer were not examined in any study. The association between PFOS exposure and testicular cancer mortality was evaluated in one retrospective occupational cohort study at the 3M chemical plant in Cottage Grove, Minnesota, and was found not to be statistically significant (Gilliland and Mandel 1993).)

The association between PFOA exposure and kidney cancer risk was examined in the following four populations, with some overlap among the latter three: workers at the 3M chemical plant in Cottage Grove, Minnesota (Lundin et al. 2009, Raleigh et al. 2014); workers at the DuPont chemical plant in Parkersburg, West Virginia (Leonard et al. 2008, Steenland and Woskie 2012, Barry et al. 2013); community members in the Mid-Ohio Valley area surrounding the DuPont plant (Barry et al. 2013, Vieira et al. 2013); and workers at six polytetrafluoroethylene production plants in Europe and North America, including the DuPont plant (Consonni et al. 2013). No significant association or exposure-response trend was observed between PFOA exposure and kidney cancer mortality (Lundin et al. 2009, Raleigh et al. 2014) or incidence (Raleigh et al. 2014) among workers at the Cottage Grove plant. A study of DuPont plant workers also found no significant association between PFOA exposure and kidney cancer mortality (Leonard et al. 2008), and the community/occupational study of the Mid-Ohio Valley area observed no significant association or exposure-response trend between PFOA exposure and kidney cancer incidence in analyses restricted to workers or combining community members and workers (Barry et al. 2013). By contrast, kidney cancer mortality was significantly positively associated with PFOA exposure in an analysis restricted to DuPont plant workers, using the same basis to estimate cumulative occupational PFOA exposure (Steenland and Woskie 2012). The discrepancy in results between Barry et al. (2013) and Steenland and Woskie (2012) could be due in part to the lack of equivalence between cancer incidence and cancer mortality, the latter of which is affected by factors, such as health care usage and disease severity and progression, that influence disease survival rather than disease development. However, the analysis of six polytetrafluoroethylene plants, including the DuPont plant, yielded no significant association between PFOA exposure and kidney cancer mortality (Consonni et al. 2013), whereas the two overlapping studies of the Mid-Ohio Valley community, not including plant workers, found a significant positive association between PFOA exposure and kidney cancer incidence (Barry et al. 2013, Vieira et al. 2013).

Overall, these findings are inconsistent, with no replication of the positive association between PFOA exposure and kidney cancer outside of the Mid-Ohio Valley/Parkersburg region. The latter association could have been confounded by co-exposure to tetrafluoroethylene, which has been found to cause kidney cancer in rodents (NTP 1997), although its association with kidney cancer in humans is not established (IARC 2016). Moreover, few other potential confounders, such as cigarette smoking, excess body weight, hypertension, and certain hereditary syndromes (Chow et al. 2010), were controlled in the analyses, and several studies classified PFOA exposure imprecisely, thereby limiting the ability to draw firm causal conclusions based on these results.

Associations between PFOA and testicular cancer in the same study populations also were inconsistent. Specifically, no statistically significant association or exposure-response trend was observed between PFOA exposure and testicular cancer or non-prostate male genital organ

cancer mortality among 3M Cottage Grove plant workers (Gilliland and Mandel 1993, Lundin et al. 2009). Likewise, no significant association or positive exposure-response trend with PFOA exposure was found for testicular cancer incidence or mortality in any of the three analyses of DuPont Parkersburg plant workers (Leonard et al. 2008, Steenland and Woskie 2012, Barry et al. 2013) or the analysis of six European and North American polytetrafluoroethylene plant workers (Consonni et al. 2013). A significant positive association between PFOA exposure and testicular cancer risk was detected in two analyses of the Mid-Ohio Valley community that used different exposure estimates (Barry et al. 2013, Vieira et al. 2013). Testicular cancer mortality, which has high survival rates, is a poor surrogate for testicular cancer incidence, which was examined in only two Mid-Ohio Valley studies (Barry et al. 2013, Vieira et al. 2013). Nevertheless, the inconsistent results and the lack of replication of the positive association outside of the Mid-Ohio Valley community, combined with methodological limitations including crude exposure classification and lack of control for potential confounders such as cryptorchidism, family history, body size, and socioeconomic status (Garner et al. 2005), prevent reliable conclusions about causality.

Associations between PFOA, PFOS, other PFAS (PFHxS, PFHpA, PFHpS, PFOSA, PFNA, PFDA, PFunDA, PFdoDA and some combined groups of PFAS) with cancer at other anatomical sites also have been inconsistently detected, and the majority of associations tested have yielded statistically non-significant results. For each statistically significant positive association detected, contradictory null or inverse associations have been reported in other studies. These include a significant positive association between PFOS exposure and bladder/other urinary organ cancer mortality in a study of workers at the 3M chemical plant in Decatur, Alabama, in one study (Alexander et al. 2003), but not in other studies conducted at the same plant (Olsen et al. 2004, Alexander and Olsen 2007) or in the general population of Denmark (Eriksen et al. 2009). Moreover, PFOA exposure was significantly inversely associated with bladder cancer incidence in two studies of DuPont Parkersburg plant workers (Barry et al. 2013, Steenland et al. 2015), and no significant association with PFOA exposure was detected in other studies (Gilliland and Mandel 1993, Leonard et al. 2008, Eriksen et al. 2009, Lundin et al. 2009, Steenland and Woskie 2012, Barry et al. 2013, Consonni et al. 2013, Vieira et al. 2013, Raleigh et al. 2014).

PFOA exposure was significantly positively associated with thyroid cancer incidence and mortality among workers at the DuPont Parkersburg plant in two studies (Leonard et al. 2008, Barry et al. 2013), but not among Mid-Ohio Valley community members (Barry et al. 2013, Vieira et al. 2013), 3M Cottage Grove plant workers (Lundin et al. 2009), or general community members in Pavia, Italy (Pirali et al. 2009).

Exposure to PFOA , PFOS, and several other PFAS was significantly associated with greater breast cancer risk in a retrospective case-control study of Greenland Inuit women (Bonefeld-

Jørgensen et al. 2011, Wielsøe et al. 2017), but these associations were not confirmed in a prospective case-cohort study of Danish women (Bonefeld-Jørgensen et al. 2014, Ghisari et al. 2017), among whom a significant positive association only with PFOSA exposure was not studied elsewhere. In other studies, associations of PFOA and PFOS exposure with breast cancer risk were statistically non-significant among Cottage Grove workers, Decatur workers, Parkersburg workers, and Mid-Ohio Valley residents (Gilliland and Mandel 1993, Alexander et al. 2003, Leonard et al. 2008, Lundin et al. 2009, Steenland and Woskie 2012, Barry et al. 2013, Vieira et al. 2013, Raleigh et al. 2014). A significant inverse association with PFOA exposure was detected among Mid-Ohio Valley residents and workers combined (Barry et al. 2013), and PFHxS exposure was associated with significantly lower breast cancer risk in Danish women (Ghisari et al. 2017). While significant interactions between PFOA and genetic polymorphisms in two xenobiotic and estrogen metabolizing genes were found in the latter study (Ghisari et al. 2017), which was prospective in design, they were not detected in the only other study to examine these interactions (Ghisari et al. 2014).

Longer duration of PFOA exposure and greater cumulative PFOA exposure were significantly associated with greater risk of prostate cancer mortality in two analyses of the 3M Cottage Grove plant (Gilliland and Mandel 1993, Lundin et al. 2009), but not in the most recent Cottage Grove analysis (Raleigh et al. 2014). The latter study also evaluated prostate cancer incidence and found no significant association with PFOA exposure (Raleigh et al. 2014). Prostate cancer mortality is a poor proxy for prostate cancer incidence because of high survival rates; therefore, associations with prostate cancer mortality may be influenced strongly by non-etiologic prognostic factors, such as health care access/usage and disease severity/progression. Moreover, no significant association between PFOA exposure and prostate cancer incidence was found in Denmark, Sweden, the Mid-Ohio Valley community, or Parkersburg plant workers (Eriksen et al. 2009, Barry et al. 2013, Vieira et al. 2013, Hardell et al. 2014, Steenland et al. 2015), and no significant excess of prostate cancer mortality was detected in other occupational cohorts exposed to PFOA (Leonard et al. 2008, Steenland and Woskie 2012, Consonni et al. 2013). PFOS exposure also was not significantly associated with prostate cancer incidence or mortality in occupational and general community studies (Olsen et al. 2004, Grice et al. 2007, Eriksen et al. 2009, Hardell et al. 2014). Exposure to PFHxS was associated with significantly greater risk of hereditary prostate cancer, but not sporadic or overall prostate cancer, in a Swedish case-control study (Hardell et al. 2014), but this association was not examined in other studies.

Analyses of associations between PFOS exposure and malignant melanoma yielded inconsistent positive (Olsen et al. 2004) and null findings at the 3M Decatur plant (Alexander et al. 2003, Grice et al. 2007), as did analyses of associations with PFOA exposure, which was significantly associated with increased melanoma risk in one Mid-Ohio Valley community study (Vieira et al. 2013), but not other studies conducted among community members and DuPont Parkersburg plant workers in the same region (Leonard et al. 2008, Barry et al. 2013, Steenland et al. 2015)

A significant positive association between PFOA exposure and lung cancer risk in the Mid-Ohio Valley community (Vieira et al. 2013) was contradicted by significant inverse associations between PFOA exposure and lung cancer in polytetrafluoroethylene workers (Consonni et al. 2013) and DuPont Parkersburg plant workers (Leonard et al. 2008), and statistically null associations in other studies (Gilliland and Mandel 1993, Lundin et al. 2009, Barry et al. 2013, Steenland et al. 2015).

Two studies found a significant inverse association between PFOA exposure and colon or colorectal cancer (Consonni et al. 2013, Innes et al. 2014) that was not confirmed in other populations (Gilliland and Mandel 1993, Barry et al. 2013, Vieira et al. 2013, Steenland et al. 2015). Other isolated significant positive associations (with malignant mesothelioma (Steenland and Woskie 2012) and brain and uterine cancers (Vieira et al. 2013)) and a significant inverse association with stomach cancer (Leonard et al. 2008) were not replicated elsewhere.

In summary, no consistent associations have been shown between exposure to PFOA, PFOS, other PFAS and any type of cancer, and no pattern of association emerged across occupational, contaminated-community, and general-community settings. The few observed statistically significant associations could plausibly be due to confounding, since most studies did not adjust adequately for other site-specific cancer risk factors; bias from selective participation, differential exposure misclassification, or other sources; or chance. Overall, the available published epidemiologic evidence does not demonstrate a causal effect of PFOA, PFOS, or other PFAS on risk of any specific cancer type in humans.

This conclusion is consistent with that reached by the International Agency for Research on Cancer (IARC 2016), which classified the weight of evidence in both humans and animals for the carcinogenicity of PFOA as "limited," and overall classified PFOA as "possibly carcinogenic to humans (Group 2B)."[12] According to IARC's classification system, "limited evidence of carcinogenicity" in humans indicates that a significant positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the IARC Working Group to be credible, but chance, bias, or confounding could not be ruled out with reasonable confidence. "Limited evidence of carcinogenicity" in experimental animals means that the data suggest a carcinogenic effect but are limited for making a definitive evaluation because, for example, the evidence of carcinogenicity is limited to a single experiment; there are unresolved questions regarding the adequacy of the design,

[12] During his deposition, Dr. Grandjean erroneously stated that "PFOA has been evaluated by IARC and it has been found to be a probable human carcinogen on the basis of the evidence" (80;12–14). On the contrary, IARC (2016) classified PFOA as a *possible*, not *probable*, human carcinogen. Dr. Grandjean's subsequent statement that "it is my opinion that [PFOA and PFOS] can cause cancer in humans at elevated exposures as seen in Minnesota" (81;10–12) thus deviates substantially from IARC's determination that PFOA is not an established human carcinogen, as well as the conclusions of U.S. EPA (2016a, b, c, d) and the Agency for Toxic Substances and Disease Registry (ATSDR 2015) that PFOA and PFOS are not established human carcinogens.

conduct, or interpretation of the studies; the agent increases the incidence of only benign neoplasms or lesions of uncertain neoplastic potential; or the evidence of carcinogenicity is restricted to studies that demonstrate only promoting activity in a narrow range of tissues or organs. Classification as "possibly carcinogenic to humans (Group 2B)," on a scale that also includes "carcinogenic to humans (Group 1)," "probably carcinogenic to humans (Group 2A)," "not classifiable as to its carcinogenicity to humans (Group 3)," and "probably not carcinogenic to humans (Group 4)," is used for agents for which there is "limited evidence of carcinogenicity" in humans and less than "sufficient evidence of carcinogenicity" in experimental animals. Thus, IARC's classification of PFOA as "possibly carcinogenic to humans" falls well short of a conclusion that PFOA has a causal effect on human cancer.

In a recent review of the human, animal, and mechanistic evidence on PFOS and cancer, following the IARC evaluation process, the authors concluded that there was "inadequate evidence of carcinogenicity" in humans and "inadequate evidence of carcinogenicity" in animals; thus, the authors proposed that PFOS should be categorized according to IARC criteria as "not classifiable as to its carcinogenicity (Group 3)" (Arrieta-Cortes et al. 2017).

Earlier reviews (including mine) of the epidemiologic evidence on the carcinogenicity of PFOA and PFOS reached similar conclusions of no established causal relationship with cancer risk (Health Council of the Netherlands 2013, Chang et al. 2014). In 2015, the Agency for Toxic Substances and Disease Registry (ATSDR) stated in its draft toxicological profile for PFAS: "There is limited information on whether perfluoroalkyls can cause cancer in humans" and "Although several studies have found significant increases in cancer risk, the results should be interpreted cautiously since most studies did not control for potential confounding variables (particularly smoking), the number of cancer cases was low, and a causal relationship between perfluoroalkyls and cancer cannot be established from these studies" (ATSDR 2015).

Likewise, U.S. EPA in 2016 classified the scientific evidence of carcinogenic potential for PFOA and PFOS as "suggestive" (U.S. EPA 2016d, b, c, a), indicating that the strength of the evidence for causality fell short of U.S. EPA's stronger categories of "carcinogenic to humans" and "likely to be carcinogenic to humans" (U.S. EPA 2005). (The other available categories are "inadequate information to assess carcinogenic potential" and "not likely to be carcinogenic to humans.") According to U.S. EPA (2005), the use of the descriptor "suggestive evidence of carcinogenic potential" is appropriate when "the weight of evidence is suggestive of carcinogenicity; a concern for potential carcinogenic effects in humans is raised, but the data are judged not sufficient for a stronger conclusion. This descriptor covers a spectrum of evidence associated with varying levels of concern for carcinogenicity, ranging from a positive cancer result in the only study on an agent to a single positive cancer result in an extensive database that includes negative studies in other species." Thus, U.S. EPA's classification of the scientific

database on PFOA and PFOS as providing "suggestive evidence of carcinogenic potential" is weaker than a conclusion that PFOA and PFOS have a causal effect on cancer in humans.

An important distinction should be made between these agencies' uses of classifications such as "possibly" or "probably" carcinogenic to humans and having "suggestive evidence" or being "likely" to be carcinogenic to humans, and typical uses of the words "possibly," "probably," and "likely" in common parlance or in a probabilistic sense. As U.S. EPA (2005) states: "Although the term 'likely' can have a probabilistic connotation in other contexts, its use as a weight of evidence descriptor does not correspond to a quantifiable probability of whether the chemical is carcinogenic. This is because the data that support cancer assessments generally are not suitable for numerical calculations of the probability that an agent is a carcinogen."

8.4 Epidemiology of PFAS and Cardiovascular or Cerebrovascular Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on cardiovascular or cerebrovascular outcomes in humans has not been established by the available published epidemiologic studies. I identified 26 published studies that reported the association between specific PFAS and cardiovascular or cerebrovascular outcomes, including blood pressure, hypertension, indicators of carotid artery atherosclerosis, heart disease, stroke, overall cardiovascular, cerebrovascular, or circulatory disease incidence or mortality, and other endpoints, in a variety of study populations and age groups (Gilliland and Mandel 1993, Alexander et al. 2003, Olsen et al. 2004, Anderson-Mahoney et al. 2008, Leonard et al. 2008, Lundin et al. 2009, Sakr et al. 2009, Melzer et al. 2010, Nolan et al. 2010, Shankar et al. 2011a, Min et al. 2012, Shankar et al. 2012, Steenland and Woskie 2012, Consonni et al. 2013, Simpson et al. 2013, Lin et al. 2013a, Raleigh et al. 2014, Winquist and Steenland 2014a, Geiger et al. 2014b, Kataria et al. 2015, Mattsson et al. 2015, Steenland et al. 2015, Lin et al. 2016, Christensen et al. 2016a, Bao et al. 2017, Lind et al. 2017c) (Table 4 in Appendix A). Two studies were conducted in adolescents, while 24 were conducted in adults (including two studies that also enrolled adolescents). Thirteen of the studies were cross-sectional, 12 were retrospective cohort studies, and one was a prospective nested case-control study. The studies of adolescents both were conducted in general community settings. Among adults, eight studies were set in the general community, one was set in a fishing community and three in contaminated communities, and 12 were set in occupational cohorts, including two that also encompassed residents of the surrounding community.

Cardiovascular endpoints varied widely across studies; blood pressure and/or hypertension were the outcomes evaluated most frequently. Among adolescents, two cross-sectional studies based in the U.S. general population found no significant association between PFOA, PFOS, or the sum of PFOA, PFOS, PFHxS, and PFNA and blood pressure or hypertension (Geiger et al.

2014b, Kataria et al. 2015). The other two studies were cross-sectional analyses based in the same population of adolescents and young adults aged 12–30 years living around Taipei, Taiwan (Lin et al. 2013a, Lin et al. 2016). No significant association was found between exposure to PFOA, PFOS, or other PFAS (PFNA and PFunDA) and systolic blood pressure. Carotid artery intima-media thickness, an indicator of atherosclerosis, was significantly positively associated with PFOS exposure, especially in individuals with higher levels of certain endothelium and platelet microparticles originating from activated or apoptotic cells, which were interpreted as potential biomarkers of endothelial dysfunction (Lin et al. 2013a, Lin et al. 2016). However, carotid artery intima-media thickness was not significantly associated with exposure to PFOA or PFunDA, and significantly inversely associated with PFNA exposure (Lin et al. 2013a, Lin et al. 2016). Some microparticles but not others were significantly positively associated with PFOS exposure and inversely associated with PFOA, PFNA, and PFunDA exposure, and 8-hydroxy-2'-deoxyguanosine, which was interpreted as a potential biomarker of oxidative stress, was not significantly associated with exposure to any of the four PFAS evaluated. These biomarkers were not measured in any other studies, so the consistency of the observed associations could not be evaluated in other settings.

The only other PFAS study that evaluated carotid artery intima-media thickness, among adults aged 70 years in Uppsala, Sweden, found no significant association between PFOS exposure and carotid artery intima-media thickness, nor were PFHpA, PFHxS, PFOA, PFNA, PFDA, or PFunDA significantly associated with this outcome (Lind et al. 2017c). The few significant positive associations detected in the latter study—between PFOSA and greater carotid artery intima-media thickness, between PFNA and greater intima-media complex echogenicity (a measure of the structural composition of the carotid artery wall), and between PFunDA and greater number of carotid arteries with atherosclerotic plaques—were all seen in women but not in men, and were not replicated in any other studies.

Among adults, reported associations of PFOA, PFOS, and other PFAS (PFBA, PFBS, perfluoropentanoic acid (PFPA), PFHpA, perfluorohexanoic acid (PFHxA), PFHxS, PFOA and isomers, PFOS and isomers, PFNA, PFDA, perfluorodecanoic sulfonate (PFDS), PFunDA, PFdoDA, perfluorotridecanoic acid (PFtrDA), and perfluorotetradecanoic acid (PFteDA)) with blood pressure and hypertension included a mixture of significant positive (Anderson-Mahoney et al. 2008, Shankar et al. 2011a, Min et al. 2012, Bao et al. 2017), significant inverse (Anderson-Mahoney et al. 2008, Winquist and Steenland 2014a, Mattsson et al. 2015, Christensen et al. 2016a), and predominantly non-significant associations (Olsen et al. 2004, Anderson-Mahoney et al. 2008, Nolan et al. 2010, Shankar et al. 2012, Winquist and Steenland 2014a, Mattsson et al. 2015, Steenland et al. 2015, Christensen et al. 2016a, Bao et al. 2017) in general community, fishing/contaminated community, and occupational settings.

Occupational retrospective cohort studies that evaluated mortality from cardiovascular, cerebrovascular, or circulatory disease, or causes of death within these categories, such as coronary heart disease, ischemic heart disease, rheumatic heart disease, endocardial disease, hypertension with or without heart disease, and stroke, all found lower risk or no significant difference in risk in association with PFOA exposure (Gilliland and Mandel 1993, Leonard et al. 2008, Lundin et al. 2009, Sakr et al. 2009, Steenland and Woskie 2012, Consonni et al. 2013, Raleigh et al. 2014) or, in one study, PFOS exposure (Alexander et al. 2003).

For nonfatal cardiovascular disease, coronary heart disease, and stroke, estimated associations with PFOA, PFOS, and other PFAS (PFHxS, PFHpA, PFHpS, PFNA, PFDA, PFunDA, and PFdoDA) also mostly were statistically null, including in studies conducted in general communities (Melzer et al. 2010, Mattsson et al. 2015), a fishing community (Christensen et al. 2016a), the Mid-Ohio Valley community (Nolan et al. 2010), PFOA workers (Steenland et al. 2015), and PFOS workers (Olsen et al. 2004). Exceptions that were not replicated elsewhere were a significant positive association between PFHpA exposure and coronary heart disease, with no exposure-response trend, in rural Sweden (Mattsson et al. 2015); a significant positive association between PFOA exposure and cardiovascular disease, peripheral arterial disease, or both in the general U.S. population (Shankar et al. 2012); a significant positive association of PFOA exposure with stroke (Simpson et al. 2013), but a significant inverse association with coronary artery disease, among Mid-Ohio Valley community residents and Parkersburg plant workers combined (Winquist and Steenland 2014a); and a significant positive association between residence in the Mid-Ohio Valley region and risk of self-reported undefined "cardiovascular problems" (Anderson-Mahoney et al. 2008).

Overall, most associations between exposure to PFOA, PFOS, and other PFAS and cardiovascular and cerebrovascular outcomes were statistically non-significant, and results did not vary systematically by study setting. Scattered significant positive associations were counterbalanced by significant inverse associations, and many of these were not independently replicated in more than one study population. Many of the studies were cross-sectional in design, raising concerns about reverse causality, and most did not adjust rigorously for numerous cardiovascular and cerebrovascular disease risk factors, including diet, body size, physical inactivity, tobacco smoking, alcohol consumption, socioeconomic status, and personal and family medical history (WHO 2017). Selection bias, including the healthy worker effect, which probably led to lower cardiovascular and cerebrovascular disease mortality in employed PFOA and PFOS workers than in the general population, was another methodological concern. Taking together these limitations with the inconsistent and generally statistically null findings, the available published epidemiologic evidence does not establish a causal effect of PFOA, PFOS, or other PFAS on risk of cardiovascular or cerebrovascular conditions in humans.

8.5 Epidemiology of PFAS and Fetal Growth

A causal effect of exposure to PFOA, PFOS, or other PFAS on fetal growth in humans has not been established by the available published epidemiologic studies. I identified 46 published studies that reported the association between specific PFAS and fetal growth, as measured by birth weight, birth length, head circumference, chest circumference, gestational age, ponderal index, and related outcomes in newborn infants, as well as categories of these measures, such as low birth weight and small for gestational age (Inoue et al. 2004, Apelberg et al. 2007, Fei et al. 2007, Grice et al. 2007, Monroy et al. 2008, Fei et al. 2008b, Nolan et al. 2009, Stein et al. 2009, Washino et al. 2009, Andersen et al. 2010, Fromme et al. 2010, Hamm et al. 2010, Kim et al. 2011a, Kim et al. 2011b, Chen et al. 2012, Maisonet et al. 2012, Wu et al. 2012, Savitz et al. 2012a, Whitworth et al. 2012a, Savitz et al. 2012b, Antignac et al. 2013, Darrow et al. 2013, Kishi et al. 2013, Lee et al. 2013, Lien et al. 2013, Morken et al. 2014, Cariou et al. 2015, Kishi et al. 2015, Robledo et al. 2015, Alkhalawi et al. 2016, Bach et al. 2016a, Callan et al. 2016, de Cock et al. 2016, Govarts et al. 2016, Kwon et al. 2016, Wang et al. 2016, Ashley-Martin et al. 2017, Berg et al. 2017, Chen et al. 2017, Kobayashi et al. 2017, Lauritzen et al. 2017, Minatoya et al. 2017, Shi et al. 2017, Starling et al. 2017, Valvi et al. 2017, Lind et al. 2017a) (Table 5 in Appendix A). Fetal growth studies were mostly prospective in design, with maternal PFAS exposure measured during pregnancy in 32 studies. Nine studies were cross-sectional (and six of the prospective cohort studies included a cross-sectional component) because they relied on PFAS levels measured in cord blood at birth. Five studies used retrospective measures or estimates of PFAS exposure. Other than six studies conducted in contaminated communities (including five in the Mid-Ohio Valley), one in a fishing community, and one in an occupational setting, the remaining 38 studies were conducted in general community settings.

The interpretation of associations between PFAS and measures of fetal growth is complicated because these associations are susceptible to confounding by maternal physiological mechanisms, such as glomerular filtration rate (GFR, i.e., the flow rate of fluid being filtrated by the kidneys), glucose metabolism, and plasma volume expansion, as well as maternal nutrition, which can produce spurious, non-causal associations with fetal growth (Savitz 2007, Morken et al. 2014, Verner et al. 2015). Such relationships with shared physiological mechanisms can distort results even in studies with prospective exposure assessment; that is, the bias is not limited to cross-sectional studies. In particular, circulating PFAS levels are dependent on GFR, since these chemicals are eliminated by the kidneys (Han et al. 2012). GFR, which generally increases by about 50% during the first half of pregnancy and declines slightly during the second half of pregnancy, has in turn been shown to be inversely associated with fetal growth (Verner et al. 2015). Maternal plasma volume also typically expands during pregnancy, and is associated with circulating PFAS levels and fetal growth (Salas et al. 1993, Salas et al. 2006). With one exception (Morken et al. 2014), none of the available studies adjusted for maternal estimated GFR (eGFR) or plasma volume expansion, leaving nearly all of

the results susceptible to confounding by these factors. The direction of bias would have been toward an association between higher PFAS levels and lower fetal growth, since both of these changes are potential consequences of lower maternal GFR and lower maternal plasma volume expansion. Indeed, the one study that reanalyzed the association between PFOA exposure and birth weight in a prospective Danish cohort found that the observed association was attenuated by 66% after adjustment for maternal eGFR (Morken et al. 2014). This confounding effect would have been magnified in studies that measured maternal PFAS levels later in pregnancy (Verner et al. 2015). Additionally, the single measurement of PFAS used in nearly all studies, and the variability across studies in the timing of exposure assessment, limits the ability of these studies collectively to capture the true relationship between PFAS exposure during gestation and fetal growth.

Although several statistically significant associations with measures of reduced fetal growth were detected among the 46 available studies, most associations with PFOA, PFOS, and other PFAS (PFHxA, PFHxS, PFHpA, PFHpS, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, PFDA, PFunDA, PFdoDA, PFtrDA, PFteDA, and combinations of PFAS) were statistically non-significant, and some significant associations were in the opposite direction toward increased fetal growth. Focusing on associations with birth weight, the outcome most commonly studied, significant associations between PFOA exposure and lower birth weight or dichotomized low birth weight were found in three studies conducted in contaminated communities (Nolan et al. 2009, Wu et al. 2012, Savitz et al. 2012b), but not in four other studies conducted in such communities or a fishing community (Stein et al. 2009, Savitz et al. 2012a, Darrow et al. 2013, Valvi et al. 2017). In general community settings, PFOA exposure also was significantly associated with lower/low birth weight in several studies (Apelberg et al. 2007, Fei et al. 2007, Andersen et al. 2010, Maisonet et al. 2012, Kwon et al. 2016, Minatoya et al. 2017, Starling et al. 2017), but not in most studies (Monroy et al. 2008, Washino et al. 2009, Fromme et al. 2010, Hamm et al. 2010, Kim et al. 2011a, Kim et al. 2011b, Chen et al. 2012, Whitworth et al. 2012a, Kishi et al. 2013, Lee et al. 2013, Lien et al. 2013, Morken et al. 2014, Cariou et al. 2015, Kishi et al. 2015, Robledo et al. 2015, Bach et al. 2016a, Callan et al. 2016, de Cock et al. 2016, Govarts et al. 2016, Wang et al. 2016, Ashley-Martin et al. 2017, Berg et al. 2017, Kobayashi et al. 2017, Shi et al. 2017, Lind et al. 2017a), and two studies found inconsistent results between boys and girls and by country (Chen et al. 2017, Lauritzen et al. 2017).

For PFOS, significant associations with lower/low birth weight also were reported in multiple studies (Apelberg et al. 2007, Chen et al. 2012, Maisonet et al. 2012, Lien et al. 2013, Kwon et al. 2016, Chen et al. 2017), and several other studies found inconsistent results between boys and girls and/or by study location (Washino et al. 2009, Andersen et al. 2010, Kishi et al. 2013, Kishi et al. 2015, Bach et al. 2016a, Lauritzen et al. 2017), including one that found a significant association with greater birth weight in boys (de Cock et al. 2016). However, most

studies found no significant association of birth weight with PFOS exposure (Inoue et al. 2004, Fei et al. 2007, Monroy et al. 2008, Hamm et al. 2010, Kim et al. 2011a, Kim et al. 2011b, Whitworth et al. 2012a, Lee et al. 2013, Cariou et al. 2015, Robledo et al. 2015, Alkhalawi et al. 2016, Callan et al. 2016, Govarts et al. 2016, Ashley-Martin et al. 2017, Berg et al. 2017, Kobayashi et al. 2017, Minatoya et al. 2017, Shi et al. 2017, Starling et al. 2017, Lind et al. 2017a). Associations with other PFAS and with other measures of fetal growth were less often studied, similarly inconsistent, and mostly statistically null (Monroy et al. 2008, Hamm et al. 2010, Kim et al. 2011a, Kim et al. 2011b, Chen et al. 2012, Maisonet et al. 2012, Antignac et al. 2013, Kishi et al. 2013, Lee et al. 2013, Lien et al. 2013, Cariou et al. 2015, Robledo et al. 2015, Alkhalawi et al. 2016, Bach et al. 2016a, Callan et al. 2016, Kwon et al. 2016, Wang et al. 2016, Ashley-Martin et al. 2017, Berg et al. 2017, Shi et al. 2017, Starling et al. 2017, Valvi et al. 2017, Lind et al. 2017a).

A recent systematic literature review of the epidemiologic evidence on PFAS and human fetal growth supported the conclusion that a causal relation between PFOA, PFOS, or other PFAS exposure and reduced birth weight has not been demonstrated (Bach et al. 2015c) (emphasis added):

> While high PFOA and PFOS exposures in pregnancy were associated with lower average birth weights in human newborns in most studies, not all results were statistically significant. *The existing data is insufficient to confirm or reject a certain association between PFASs exposure and fetal growth*. Knowledge on the influence of PFASs other than PFOS and PFOA on fetal growth is sparse and needs to be investigated in future studies.

A similar conclusion was reached in a 2016 literature review, in which the authors concluded that the level of evidence for an association between PFOA and birth weight was "moderate," while that for PFOS and other PFAS was "insufficient" (Vrijheid et al. 2016).

In summary, the available published epidemiologic studies of PFAS exposure and fetal growth yielded inconsistent and mostly statistically null results, and associations were not stronger in contaminated communities (or in the one occupational cohort) than in general communities. Although significant inverse associations between PFOA, PFOS, and other PFAS and indicators of fetal growth, especially birth weight, were detected in several studies, the validity of these findings is undermined by the high potential for confounding by maternal GFR and plasma volume expansion, which would be expected to introduce biased associations between maternal PFAS levels and reduced fetal growth (Morken et al. 2014, Verner et al. 2015). Given the mostly non-significant associations with birth weight and other indicators of fetal growth, as well as the inability of available studies to distinguish between a causal effect of maternal PFAS

and a spurious effect of underlying maternal physiology, the published epidemiologic evidence does not establish a causal effect of PFOA, PFOS, or other PFAS on human fetal growth.

8.6 Epidemiology of PFAS and Immune or Hematological Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on immune or hematological outcomes in humans has not been established by the available published epidemiologic studies. I reviewed 41 published studies that reported the association between specific PFAS and immune outcomes or hematological endpoints, including a wide variety of endpoints ranging from laboratory measures of blood cell counts, immune biomarkers, and anti-vaccine antibody levels to allergies, asthma, other atopic conditions, infections, cold symptoms, and autoimmune diseases (Emmett et al. 2006, Anderson-Mahoney et al. 2008, Leonard et al. 2008, Costa et al. 2009, Lundin et al. 2009, Melzer et al. 2010, Fei et al. 2010a, Lin et al. 2011, Wang et al. 2011, White et al. 2011, Shankar et al. 2011a, Grandjean et al. 2012, Okada et al. 2012, Dong et al. 2013, Granum et al. 2013, Kishi et al. 2013, Steenland et al. 2013, Humblet et al. 2014, Jiang et al. 2014, Looker et al. 2014, Okada et al. 2014, Osuna et al. 2014, Ashley-Martin et al. 2015, Genser et al. 2015, Smit et al. 2015, Steenland et al. 2015, Mogensen et al. 2015a, Buser and Scinicariello 2016, Conway et al. 2016, Dalsager et al. 2016, Kielsen et al. 2016, Zhu et al. 2016, Goudarzi et al. 2016a, Stein et al. 2016a, Stein et al. 2016b, Grandjean et al. 2017, Oulhote et al. 2017, Qin et al. 2017, Timmermann et al. 2017a, Goudarzi et al. 2017b, Zhou et al. 2017b) (Table 6 in Appendix A). Eight of these studies evaluated immune outcomes in newborns, infants, and/or young children up to about 4 years; 16 focused on children and/or adolescents; and the remaining 17 included mostly or exclusively adults. Seventeen studies were cross-sectional, four were retrospective case-control studies, four were retrospective cohort studies, and 16 were partly or entirely prospective cohort studies. Most studies (24) were conducted in general community settings, seven were conducted at least partially in fishing communities and six in PFAS-contaminated communities; and four were conducted in occupational settings.

Based on its evaluation of the epidemiologic evidence on PFOA exposure and autoimmune disease, the C8 Science Panel concluded in 2012 that ulcerative colitis, but not other autoimmune diseases, had a “probable link” to PFOA exposure (C8 Science Panel 2012c). At present, however, the association between exposure to PFAS and risk of ulcerative colitis has been evaluated only in the Mid-Ohio Valley region, including one study of community members and DuPont Parkersburg plant workers combined (Steenland et al. 2013) and one study of workers only (Steenland et al. 2015). The first study did not report results separately for community members and workers; therefore, the significant positive association between PFOA exposure and ulcerative colitis in both studies could have been driven largely by the same population of workers. Another limitation of these studies is that they obtained

information on ulcerative colitis (and other diseases) based on self-report, and validated only positive reports using medical records. Given that only 151 (25%) of 596 self-reported diagnoses of ulcerative colitis in the combined cohort were confirmed by medical records review (with ~75% consenting to medical record review and ~92% of those yielding an obtained medical record), self-reported data on ulcerative colitis appear to be highly error-prone (Steenland et al. 2013). Thus, the consistency of the positive association between PFOA exposure and ulcerative colitis observed in the Mid-Ohio Valley community/worker population has not been tested or established, and a causal interpretation of the observed association is precluded by methodological limitations of the existing overlapping studies, including a potentially high degree of misclassification of both estimated cumulative PFOA exposure (Shin et al. 2011b) and ulcerative colitis as a health outcome. Other autoimmune diseases, including rheumatoid arthritis, Crohn's disease, type 1 diabetes, lupus, and multiple sclerosis, also were either significantly inversely associated (in the case of type 1 diabetes), not significantly associated, or not consistently associated (in the case of rheumatoid arthritis) with PFOA exposure in the Mid-Ohio Valley population (Steenland et al. 2013, Steenland et al. 2015, Conway et al. 2016), and have not been studied elsewhere in relation to PFAS exposure. (Type 1 diabetes risk also was significantly inversely associated with PFOS, PFHxS, and PFNA exposure in one Mid-Ohio Valley cross-sectional study (Conway et al. 2016).) A single cross-sectional pilot study found predominantly non-significant associations of PFOA and PFOS exposure with autoantibodies against neural and non-neural antigens among children in a fishing community (Osuna et al. 2014).

Ten studies from six independent study populations reported associations between PFOA, PFOS, or other PFAS (PFBS, PFHxA, PFHxS, PFHpA, PFNA, PFDA, PFDA, PFunDA, PFdoDA, PFteDA, and combinations of these) and asthma in children, adolescents, and adults. Positive associations with PFOA were found among children and adolescents in the general communities of northern Taiwan (Dong et al. 2013) and the U.S. (Humblet et al. 2014), but not in a study of fishing and general communities in Europe (Smit et al. 2015). A mixed prospective and cross-sectional study of children and adolescents in the Faroe Islands found that PFOA exposure at age 5 years (but not during gestation or at 13 years) was associated with significantly greater risk of asthma (especially atopic asthma) at ages 5 years and 13 years among children who had not received the measles, mumps, and rubella (MMR) vaccination, but no such association was detected among MMR-vaccinated children (Timmermann et al. 2017a). In adults, a significant positive association but no exposure-response trend was observed in the general U.S. population (Melzer et al. 2010), and asthma risk was higher among residents of contaminated water districts in the Mid-Ohio Valley population than in national survey data (Anderson-Mahoney et al. 2008). By contrast, no significant association of PFOA exposure with risk of medicated asthma was observed among workers at the DuPont PFOA plant in Parkersburg, West Virginia (Steenland et al. 2015), nor was a significant excess of asthma

mortality observed among workers at the 3M PFOA plant in Cottage Grove, Minnesota (Lundin et al. 2009).

PFOS exposure was significantly associated with asthma risk among children and adolescents in northern Taiwan (Dong et al. 2013), but not the U.S. or Europe (Humblet et al. 2014, Smit et al. 2015), and the one study of PFOS and asthma in adults found no significant association (Melzer et al. 2010). The Faroe Islands study found that PFOS exposure at 5 years (but not during gestation or 13 years) was significantly associated with increased risk of atopic asthma at 5 years (but not 13 years) only among children who had not received the MMR vaccine (but not MMR-vaccinated children) (Timmermann et al. 2017a). Significant associations of asthma with other PFAS were observed only in two other studies (Dong et al. 2013, Timmermann et al. 2017a). One of these studies found some variations between asthmatics and non-asthmatics in associations of PFAS with biomarkers of atopy (e.g., eosinophils and immunoglobulin (Ig)E) or immune function (e.g., cytokines such as interleukins 2, 4, and 5), reproductive hormone levels, and lung function (Dong et al. 2013, Zhu et al. 2016, Qin et al. 2017, Zhou et al. 2017b), while the other found significant heterogeneity by MMR vaccination status (Timmermann et al. 2017a); these interactions have not been studied elsewhere. Thus, the data on PFAS exposure and asthma are inconsistent and sparse, and inference is limited in most cases by cross-sectional or retrospective exposure assessment, unvalidated outcome assessment, and potential confounding.

Ten studies examined the association of PFOA, PFOS, and other PFAS (PFHxA, PFHxS, PFHpA, PFNA, PFDA, PFunDA, PFdoDA, PFtrDA, and combinations of these) with various allergic/atopic outcomes among infants, children, and adolescents in general community or fishing community settings. Allergic/atopic outcomes evaluated in these studies included general allergy or allergic diseases, food allergy, allergic sensitization (as measured by specific IgE), wheeze, rhinitis or rhinoconjunctivitis, eczema, atopic dermatitis, and positive skin prick test. Overall, most associations tested were statistically null, and significant associations detected between specific PFAS and specific allergic/atopic outcomes, which may have distinct etiologies, were not consistent across studies, or within studies in some instances. For PFOA, a significant positive association with self-reported food allergy, especially to peanuts, tree nuts, or shellfish, among U.S. adolescents (Buser and Scinicariello 2016) was not supported by a non-significant association between PFOA exposure and specific-IgE-based food sensitization in the same study, as well as statistically null findings for PFOA exposure and food allergy (Okada et al. 2012, Kishi et al. 2013) and allergic sensitization to food or other antigens (Stein et al. 2016b) in other studies. A significant positive association between PFOA exposure and risk of rhinitis in U.S. children and adolescents in one study (Stein et al. 2016b) was offset by non-significant associations with rhinoconjunctivitis among Japanese children (Goudarzi et al. 2016a) and Faroese children and adolescents (Timmermann et al. 2017a). Associations between PFOA exposure and general allergy/allergic disease, wheeze, eczema, atopic dermatitis, and

positive skin prick test were non-significant or significantly inverse (Wang et al. 2011, Okada et al. 2012, Kishi et al. 2013, Humblet et al. 2014, Okada et al. 2014, Smit et al. 2015, Goudarzi et al. 2016a, Stein et al. 2016b, Timmermann et al. 2017a).

For PFOS, significant positive associations were found with self-reported food allergy, but not elevated food-specific IgE (Buser and Scinicariello 2016), and with elevated mold-specific IgE, but decreased specific IgE against plants, cockroach/shrimp, and any antigen (also including dust mites, pets, rodents, and food) (Stein et al. 2016b). Other observed associations with PFOS exposure were null or significantly inverse (Wang et al. 2011, Okada et al. 2012, Kishi et al. 2013, Humblet et al. 2014, Okada et al. 2014, Smit et al. 2015, Goudarzi et al. 2016a, Stein et al. 2016b, Timmermann et al. 2017a), as were associations with other PFAS, except for two associations (PFHxS and greater risk of self-reported food allergy (Buser and Scinicariello 2016); PFtrDA and greater risk of rhinoconjunctivitis, with no exposure-response trend (Goudarzi et al. 2016a)) that were not replicated elsewhere.

Various measures of infection, ranging from cold and influenza symptoms to hospitalization or mortality from infectious diseases, have been studied in relation to PFAS exposure. Findings have been heterogeneous and often inconsistent within and across studies, but mostly statistically null. Significant positive associations of PFOA exposure were detected with fever and nasal discharge, but not cough, diarrhea, or vomiting, in young children in the general community of Odense, Denmark (Dalsager et al. 2016); hospitalization for infectious diseases in Denmark among girls, but not boys, aged 0–10 years in the Danish general population (Fei et al. 2010a); and number of episodes of common cold and gastroenteritis at 0–3 years among Norwegian children (Granum et al. 2013). However, no significant association was observed between PFOA exposure and total infectious diseases at 4 years among children in Hokkaido, Japan (Goudarzi et al. 2017b); otitis media at 18 months among children in Sapporo, Japan (Okada et al. 2012, Kishi et al. 2013); cold or influenza among adults in the Mid-Ohio Valley community (Looker et al. 2014); or mortality from infectious and parasitic diseases among workers at the DuPont Parkersburg plant (Leonard et al. 2008). Most associations between PFOS exposure and infection were also statistically null (Fei et al. 2010a, Okada et al. 2012, Granum et al. 2013, Looker et al. 2014, Dalsager et al. 2016, Goudarzi et al. 2017b), outnumbering the few significant positive associations (Fei et al. 2010a, Dalsager et al. 2016, Goudarzi et al. 2017b). Isolated associations between other specific PFAS and infections were not replicated elsewhere.

Studies of PFAS exposure in relation to hematological metrics and immune biomarkers are difficult to interpret for several reasons, including the cross-sectional design of many of these studies; the intercorrelation of many outcomes, such that observed associations are not independent of each other; the fluctuation (often substantial) of most biomarkers within individuals over time, such that a single test may not be informative; and, often, comparison of

exposed and unexposed groups that both fall within the normal range of biomarker levels, such that any observed differences may not be clinically meaningful. In six studies of infants and children, associations of PFOA, PFOS, and other PFAS (PFHxA, PFHxS, PFHpA, PFNA, PFDA, PFunDA, PFdoDA, PFtrDA, PFteDA, and five PFAS combined) with a range of immune biomarkers were mostly statistically null. PFOA exposure was significantly associated with lower IgE levels among girls in one community, but not among boys and not in the other community, in a study based in Japan (Okada et al. 2012, Kishi et al. 2013). By contrast, PFOA exposure was significantly associated in the opposite direction, with higher total IgE levels, among boys but not girls in Taiwan (Wang et al. 2011) and among U.S. children and adolescents (Stein et al. 2016b), whereas no significant association between PFOA and total IgE was detected in Canada (Ashley-Martin et al. 2015) or the Faroe Islands (Timmermann et al. 2017a). Other associations reported in these studies were statistically null or, in the case of a significant positive association between PFAS exposure and basophil levels in the Faroe Islands fishing community (Oulhote et al. 2017) and a positive association between PFNA exposure and total IgE in the U.S. (Stein et al. 2016b), not replicated elsewhere.

Among adults, hematological and immune biomarker studies, all of which were cross-sectional in design, found mostly statistically null results in association with PFOA, PFOS, and other PFAS (PFHxA, PFHxS, PFHpA, PFOSA, PFNA, PFDA, PFunDA, PFdoDA, and combinations of these). Across studies conducted in general communities (Lin et al. 2011, White et al. 2011, Shankar et al. 2011a, Jiang et al. 2014, Stein et al. 2016a), in the Mid-Ohio Valley (Emmett et al. 2006, Genser et al. 2015), and in the Miteni PFOA production plant in Trissino, Italy (Costa et al. 2009), no significant association between a specific PFAS and a specific hematological or immune biomarker was observed in more than one study setting.

The relationships of exposure to PFOA, PFOS, and other PFAS (PFHxS, PFNA, PFDA, and combinations of these) with the vaccine immune response were assessed in four cross-sectional studies and four prospective cohort studies (two of which had some cross-sectional analyses) in four general community settings (Granum et al. 2013, Kielsen et al. 2016, Stein et al. 2016a, Stein et al. 2016b), one fishing community (three studies in the Faroe Islands (Grandjean et al. 2012, Mogensen et al. 2015a, Grandjean et al. 2017)), and one contaminated community (one study among the Mid-Ohio Valley residents (Looker et al. 2014)). Vaccine antigens against which antibody responses were measured included diphtheria, tetanus, influenza A/H3N2, influenza A/H1N1, influenza B, mumps, rubella, and measles.

Studies of the antibody-mediated immune response to vaccination share some of the same problems with interpretation as studies of hematological and immune biomarkers, because antibody levels fluctuate over time (Laserson et al. 2014), and observed differences may not translate to clinically detectable discrepancies in immune function. Additionally, changes in the immune response to one vaccine cannot necessarily be interpreted as being consistent with

changes in the immune response to another vaccine, because the particular components of the immune system involved in the response to each antigen may differ (Amanna et al. 2007, Granum et al. 2013). Indeed, epidemiologic studies that evaluated associations between exposure to PFAS and the immune response to multiple vaccines generally found heterogeneous associations by vaccine type, suggesting that the immune responses to different vaccine types should not be interpreted as a single health outcome.

Anti-tetanus antibody levels were evaluated in three studies, the most of any of the vaccine antigens studied; results were inconsistent across studies. Among Faroe Island children, an assortment of significant inverse associations and statistically null associations between PFOA, PFOS, PFHxS, PFNA, and PFDA exposure and anti-tetanus antibody levels were observed, depending on the ages at PFAS and antibody assessment. For PFOA, significant inverse associations with anti-tetanus antibody levels were observed between PFOA measured at 5 years of age and antibody levels measured at 7 years of age (Grandjean et al. 2012), but not between in most prospective and cross-sectional combinations of PFOA levels measured prenatally, at 7 years, and at 13 years of age and anti-tetanus antibody levels measured at 5, 7, and 13 years of age (Grandjean et al. 2012, Mogensen et al. 2015a, Grandjean et al. 2017). In the Faroese study population, PFOS exposure was significantly inversely associated with anti-tetanus antibody levels measured simultaneously at 5 years of age (Grandjean et al. 2012), whereas PFOS levels measured at 7 years of age were significantly positively associated with anti-tetanus antibody levels measured at 13 years of age in children who were deemed less likely to have a received a booster vaccine (Grandjean et al. 2017); no other PFOS-anti-tetanus-antibody combinations were significantly associated. A combination of significant inverse and mostly non-significant associations with anti-tetanus antibody levels also were observed for PFHxS, PFNA, and PFDA. By contrast, prenatal exposures to PFOA, PFOS, PFHxS, and PFNA all were non-significantly associated with anti-tetanus antibody levels in a Norwegian cohort of 0- to 3-year-olds (Granum et al. 2013). Also, no significant cross-sectional association was observed between PFOA, PFOS, PFHxS, or PFNA and post-vaccination antibody levels against tetanus among adults in Copenhagen, Denmark, although lower post-vaccination antibody increases were observed in association with PFunDA and PFdoDA exposure (Kishi et al. 2013).

Significantly lower levels of anti-diphtheria antibodies were observed in association with PFOA, PFOS, PFHxS, and PFDA levels in several prospective as well as cross-sectional age combinations among Faroe Island children at ages 5, 7, and 13 years (Grandjean et al. 2012, Mogensen et al. 2015a, Grandjean et al. 2017). For PFOA, these combinations included PFOA prenatally and at 5 years in association with antibodies at 7 years; PFOA at 7 years in association with antibodies at 7 and 13 years; and PFOA at 13 years in association with antibodies at 13 years. For PFOS, these combinations included PFOS prenatally in association with antibodies at 5 years; PFOS at 5 years in association with antibodies at 7 years; and PFOS at 7 years in association with antibodies at 7 and 13 years. A minority of associations between

anti-diphtheria-antibody levels and exposure to PFHxS, PFNA, and PFDA also were significantly inverse (Grandjean et al. 2012, Grandjean et al. 2017). However, while exposure to PFOS (as well as PFNA, PFDA, PFunDA, and PFdoDA) was significantly associated with a lower post-vaccination increase in anti-diphtheria antibodies among Copenhagen adults, exposure to PFOA (and PFHxS and PFHpA) was not (Kielsen et al. 2016). Thus, significant inverse associations of PFOS, PFNA and PFDA exposure with the anti-diphtheria antibody response were observed in two study populations, but this relationship was not studied elsewhere.

Significant inverse cross-sectional associations of exposure to PFOA, PFOS, and PFHxS (but not PFNA) and anti-rubella antibody levels were observed in antibody-seropositive U.S. children and adolescents (Stein et al. 2016b). These associations, as well as a significant inverse association with PFNA exposure, also were observed in a prospective birth cohort of children up to age 3 years in Norway (Granum et al. 2013). PFOA and PFOS levels also were significantly associated with lower anti-mumps antibody levels in the U.S. study (Stein et al. 2016b), whereas anti-measles antibody levels were not significantly associated with exposure to PFOA, PFOS, PFHxS, or PFNA exposure in the Norwegian study (Granum et al. 2013) or the U.S. study (Stein et al. 2016b). Thus, these associations were replicated in two populations, but were not assessed in any other independent studies, particularly in large, prospective cohorts. Moreover, associations with anti-measles antibody levels were statistically null in both studies.

Post-FluMist-vaccination levels of anti-influenza A/H1N1 antibodies were not significantly associated with PFOA, PFOS, PFHxS, or PFNA exposure in an overall cross-sectional analysis of adults in New York City—and where significant associations were observed in subgroup analyses, they were in the direction of greater odds of seroconversion in relation to PFOS and PFNA exposure (Stein et al. 2016a). Anti-influenza A/H1N1 antibody levels also were not significantly associated with PFOA or PFOS levels in a cross-sectional analysis of Mid-Ohio Valley adults (Looker et al. 2014).

Finally, anti-influenza B antibody levels were not significantly associated with exposure to PFOA or PFOS in Mid-Ohio Valley adults (Looker et al. 2014) or in young Norwegian children, among whom PFHxS and PFNA exposure also were not significantly associated with anti-influenza B antibodies (Granum et al. 2013).

In summary, among published epidemiologic studies that examined a broad array of immune outcomes, associations between specific PFAS and specific immune endpoints were not consistently observed across multiple study populations, nor was any clear pattern of association detected by study setting in accordance with average exposure levels. Many outcomes were evaluated in a small number of studies, providing an insufficient scientific basis on which to make a determination of causality. Confounding, bias, and chance could not be

ruled out as plausible explanations for the statistically significant associations observed. Numerous genetic, demographic, behavioral, infectious, medical, and environmental risk factors, such as nutrition, occupation, physical exercise, stress, smoking, and urbanization, have been identified for immune disorders (Miller et al. 2012, Beasley et al. 2015, Genuneit et al. 2017); these could potentially act as confounders if not adequately controlled in epidemiologic studies. Thus, the overall published epidemiologic evidence does not demonstrate a causal relationship between exposure to PFOA, PFOS, or other PFAS and risk of any specific immune outcome in humans.

Two 2016 reviews (including mine) of the epidemiologic evidence on the potential immunotoxicity of PFOA and PFOS reached similar conclusions that the available epidemiologic database was insufficient to demonstrate a causal immunotoxic effect (Chang et al. 2016, NTP 2016b). Specifically, the National Toxicology Program (NTP) classified both PFOA and PFOS as "presumed to be an immune hazard to humans," based on a "high" level of evidence of suppression of the antibody response in animal studies, and a "moderate" level of evidence from human studies. By not classifying PFOA and PFOS as "known" to be an immune hazard and not classifying the level of human evidence as "high," NTP stopped short of determining that PFOA and PFOS are known to cause immunotoxic effects in humans. (The other hazard identification categories available to NTP were "suspected," "not classifiable," and "not identified to be an immune hazard to humans.")

With respect to children in particular, a recent literature review concluded that the epidemiologic evidence thus far is "insufficient" to establish any association between PFAS exposure and childhood immune outcomes (Vrijheid et al. 2016). Likewise, another recent review of the epidemiologic literature conducted by authors from U.S. EPA did not reach a conclusion that a causal relationship has been demonstrated with any specific immune-related endpoint in children (Rappazzo et al. 2017):

> Studies of individual health outcomes are limited in number, therefore conclusions should be made with caution; current evidence potentially suggests that antibody response to vaccination and asthma may be influenced by PFAS. The studies of vaccine response were well done cohort study designs and despite the small number offer compelling evidence. The asthma studies are less consistent and include a broader range of study designs and quality. There is no evidence for relationships between PFAS and IgE levels, allergy, and infection.

8.7 Epidemiology of PFAS and Kidney Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on non-malignant kidney (renal) outcomes in humans has not been established by the available published epidemiologic studies.

Twenty-two published studies reported the association between specific PFAS and kidney outcomes, including uric acid level,[13] non-specific laboratory metrics such as blood urea nitrogen, creatinine, total protein, and albumin levels, eGFR, chronic kidney disease, and mortality from nephritis/nephrosis or chronic kidney disease, in a range of populations (Emmett et al. 2006, Grice et al. 2007, Anderson-Mahoney et al. 2008, Leonard et al. 2008, Costa et al. 2009, Lundin et al. 2009, Steenland et al. 2010b, Shankar et al. 2011a, 2011b, Steenland and Woskie 2012, Consonni et al. 2013, Geiger et al. 2013, Watkins et al. 2013, Lin et al. 2013a, Jiang et al. 2014, Raleigh et al. 2014, Gleason et al. 2015, Kataria et al. 2015, Steenland et al. 2015, Qin et al. 2016, Dhingra et al. 2016b, Dhingra et al. 2017) (Table 7 in Appendix A). One of these studies evaluated kidney function in children and adolescents up to age 18 years, three in adolescents, and 18 in adults or mixed age groups including children or adolescents as well as adults. Twelve studies were cross-sectional, nine were retrospective cohort studies (with two including some cross-sectional analyses), and one was a prospective cohort study (including some retrospective analyses). Eight studies were conducted in general community settings, six were conducted in the Mid-Ohio Valley community, and the remaining eight were conducted in occupational settings in the U.S. and Europe.

The cross-sectional design of many studies of PFAS and kidney outcomes is especially problematic because of the high potential for reverse causation, given that impaired kidney function (i.e., lower GFR) leads to reduced elimination and higher circulating levels of PFAS (Han et al. 2012, Dhingra et al. 2017). Thus, observed cross-sectional associations between PFAS exposure and higher levels of uric acid, lower eGFR, and a higher prevalence of chronic kidney disease are an expected consequence of reverse causation, and cannot reliably be interpreted as an indication of a causal effect of PFAS exposure on impaired kidney function.

Indeed, in the 14 cross-sectional analyses of PFAS exposures and kidney outcomes, significant associations between exposure to PFOA, PFOS, and/or other PFAS (PFBS, PFHxA, PFHxS, PFHpA, PFNA, PFDA, PFunDA, PFdoDA, PFteDA, and combinations of these) and greater levels of uric acid and/or risk of hyperuricemia (Costa et al. 2009, Steenland et al. 2010b, Shankar et al. 2011a, 2011b, Geiger et al. 2013, Gleason et al. 2015, Kataria et al. 2015, Qin et al. 2016), greater levels of non-specific laboratory markers of kidney function (Jiang et al. 2014), lower eGFR (Shankar et al. 2011a, Watkins et al. 2013, Kataria et al. 2015, Dhingra et al. 2017), and a higher prevalence of self-reported unspecified kidney disease (Anderson-Mahoney et al. 2008) were reported in the majority of studies.

[13] Although uric acid is a possible marker of cardiovascular or metabolic disease (Cain et al. 2010), it is discussed in this section because it is also a marker of potential chronic kidney disease, and because associations between serum or plasma PFOA and uric acid were highly susceptible to reverse causation due to underlying kidney function.

Focusing on the 10 available retrospective and prospective cohort analyses, in which PFOA exposure was assessed or estimated using pre-disease information on employment (in occupational cohorts) or PFOA emission and dispersion data, residential history and water consumption, and a physiologically-based pharmacokinetic model for PFOA (in the Mid-Ohio Valley), the only significant association reported was between PFOA exposure and greater risk of mortality from chronic renal disease among DuPont Parkersburg plant workers (Steenland and Woskie 2012). This single finding was counterbalanced by statistically null associations between PFOA exposure and nephritis/nephrosis mortality among North American and European polytetrafluoroethylene plant workers (including the Parkersburg plant workers) (Consonni et al. 2013), Parkersburg plant workers in an earlier analysis (Leonard et al. 2008), and 3M Cottage Grove plant workers (Lundin et al. 2009, Raleigh et al. 2014). Moreover, analyses of the association between PFOA exposure and eGFR and chronic kidney disease in these cohorts consistently showed no significant results (Watkins et al. 2013, Steenland et al. 2015, Dhingra et al. 2016b, Dhingra et al. 2017). The one retrospective cohort study of PFOS exposure also showed no significant association with nephrolithiasis (Grice et al. 2007). (PFAS other than PFOA and PFOS have not been evaluated in relation to kidney outcomes in any published retrospective or prospective cohort studies.)

A recent U.S. EPA literature review did not find that a causal relationship has been established between exposure to PFAS and kidney outcomes in children (Rappazzo et al. 2017): “While all studies of PFAS and kidney function in children to date have been cross-sectional, results from these studies provide evidence for interesting potential associations between PFAS and renal function in children using multiple different markers of kidney function.”

In summary, based on epidemiologic studies that are less susceptible by design to reverse causation, the association between PFOA or PFOS exposure and non-malignant kidney outcomes was mostly or entirely statistically null, depending on the endpoint. No evident pattern of association was detected by study setting, whereby stronger associations might be expected in occupational cohorts, followed by contaminated communities and general communities. Because cross-sectional studies of PFAS exposure and kidney function are highly susceptble to reverse causation, their results cannot reliably be interpreted as providing evidence in support of an effect of PFAS exposure on kidney outcomes. Thus, taking together the generally non-significant findings from retrospective and prospective cohort studies and the high likelihood of spurious associations in cross-sectional studies, no causal effect of exposure to PFOA, PFOS, or other PFAS on non-malignant kidney outcomes has been demonstrated in the avaiable published epidemiologic studies.

8.8 Epidemiology of PFAS and Lipid Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on lipid outcomes in humans has not been established by the available published epidemiologic studies. I reviewed 41 published studies that reported the association between specific PFAS and lipid outcomes, including levels of total cholesterol, low-density lipoprotein (LDL) cholesterol, very low-density lipoprotein (VLDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, ratios of these measures, other lipids and lipoproteins, and hyperlipidemia (e.g., high cholesterol levels), in various study populations and age groups, especially adults (Gilliland and Mandel 1996, Olsen et al. 1999, Olsen et al. 2000, 2003a, Olsen et al. 2004, Emmett et al. 2006, Olsen and Zobel 2007, Sakr et al. 2007a, Sakr et al. 2007b, Costa et al. 2009, Lin et al. 2009, Steenland et al. 2009, Chateau-Degat et al. 2010, Frisbee et al. 2010, Nelson et al. 2010, Gump et al. 2011, Lin et al. 2011, MacPherson et al. 2011, White et al. 2011, Shankar et al. 2011a, 2012, Wang et al. 2012, Olsen et al. 2012a, Eriksen et al. 2013, Fisher et al. 2013, Fitz-Simon et al. 2013, Lin et al. 2013a, Fu et al. 2014, Timmermann et al. 2014, Geiger et al. 2014a, Winquist and Steenland 2014a, Kataria et al. 2015, Kishi et al. 2015, Mattsson et al. 2015, Skuladottir et al. 2015, Steenland et al. 2015, Maisonet et al. 2015b, Domazet et al. 2016, Christensen et al. 2016a, Starling et al. 2017) (Table 8 in Appendix A). Four of these studies evaluated lipid levels in children under 12 years, eight (including some that considered children separately) examined adolescents as a separate age group, and 34 studies focused on adults or mixed age groups including adults. Thirty studies were cross-sectional in design, three were retrospective cohort studies, and eight included at least a prospective component (including one phase I clinical trial and two studies that included some cross-sectional analyses). Twenty-two studies were conducted in general community settings, two were conducted in fishing communities, four took place in the Mid-Ohio Valley community, and twelve were occupational studies (one of which also included residents from the surrounding contaminated community).

Approximately three quarters of the studies of PFAS exposure and lipid outcomes were cross-sectional. As with other health outcomes, the cross-sectional design prevents a causal interpretation of study results, especially in light of plausible reverse-causal associations. Shared underlying physiological mechanisms that affect circulating PFAS levels and lipid levels could also influence results, leading to spurious associations, even in prospective studies. Specifically, alternative explanations for observed positive associations between serum PFAS and serum lipid levels include a common underlying physiological mechanism (Frisbee et al. 2010), such as shared gut receptors; an effect of total cholesterol, LDL, and non-HDL cholesterol on decreased kidney function (Schaeffner et al. 2003, Morita et al. 2010); and confounding by numerous demographic, behavioral, and environmental factors that affect lipid levels (Thelle 1990), such as high-fat diets, which increase circulating lipid levels and also could be associated with higher exposure to PFAS from fast-food packaging (Buck et al. 2011). Cross-sectional studies—as well as other epidemiologic studies of PFAS and lipids, given that

most measured lipid levels only once—also are hampered by outcome misclassification, given that lipid levels can vary, sometimes substantially, within individuals over short and long time scales (Hegsted and Nicolosi 1987, Smith et al. 1993, Tolfrey et al. 1999). Additional methodological concerns in these studies are whether analyses of LDL cholesterol and/or triglycerides were restricted to fasting blood specimens, and whether analyses of cholesterol levels were restricted to individuals not taking lipid-lowering medications. Some analyses were not restricted to individuals with non-fasting specimens or those not taking lipid-lowering medications, or it was unclear whether such restrictions were implemented. Not doing so would have led to outcome misclassification with an unpredictable influence on results, given likely correlations between misclassification error and potential confounders such as socioeconomic status and health care access.

Even the results of prospective studies can be biased by non-causal physiological factors, such as body size and diet, that may affect PFAS clearance at background exposure levels (Longnecker 2006):

> Because the [pollutants] found in greatest concentration in human blood have half-lives on the order of years, small differences in clearance could, with sufficient time, account for substantial differences in blood levels … The impressive capabilities of modern analytical chemistry are being applied with increasing frequency in epidemiologic studies. With lower exposures being measured, it seems opportune to bear in mind that a great proportion of variation in measured levels among subjects may be accounted for by differences in metabolism and excretion.

In 2012, the C8 Science Panel issued a "probable link" evaluation of heart disease, and concluded that PFOA exposure had a "probable link" with diagnosed high cholesterol, but not other cardiovascular outcomes (C8 Science Panel 2012e). This conclusion was based largely on a significant positive cross-sectional association of PFOA (and PFOS) exposure with risk of high total and LDL cholesterol (total cholesterol ≥170 mg/dL, LDL cholesterol ≥ 110 mg/dL) among children and adolescents < 18 years in the Mid-Ohio Valley community (Frisbee et al. 2010), as well as a significant positive cross-sectional association of PFOA (and PFOS) exposure with risk of high total cholesterol (≥ 240 mg/dL) among adults in the same community (Steenland et al. 2009). After 2012, in a retrospective cohort analysis of the combined population of the Mid-Ohio Valley community and DuPont Parkersburg plant workers, cumulative PFOA exposure was significantly associated with greater risk of high cholesterol treated with medication, more so among men than women; however, PFOA exposure was significantly associated with *lower* risk of medicated high cholesterol in a prospective cohort analysis of outcomes diagnosed after study enrollment (Winquist and Steenland 2014a). In a

retrospective cohort study of plant workers alone, no significant association was observed between PFOA exposure and incidence of medicated high cholesterol (Steenland et al. 2015).

Likewise, in six other studies, no significant association was detected between exposure to PFOA or PFOS and risk of high cholesterol. These studies included a cross-sectional analysis of Wisconsin anglers (a fishing community), in which a significant association between PFHpS and greater risk of high cholesterol was not replicated elsewhere (and PFHxS, PFNA, PFDA, and PFunDA were not significantly associated with high cholesterol) (Christensen et al. 2016a); a cross-sectional analysis of the Canadian general adult population, in which a significant association between PFHxS and greater risk of high cholesterol was not replicated elsewhere (Fisher et al. 2013); and consistently statistically null associations between exposure to PFOA, PFOS, and other PFAS (PFHxS, PFNA, PFDA, PFunDA, or combinations of PFAS) and risk of high cholesterol in general communities (Fu et al. 2014, Kataria et al. 2015) and PFOA or PFOS workers (Olsen et al. 2003a, Olsen et al. 2004). Thus, the combined epidemiologic evidence does not show a consistent association between exposure to PFOA, PFOS, or other PFAS and diagnosed high cholesterol, especially accounting for the problematic interpretation of the results of cross-sectional studies, and most studies published after the C8 Science Panel's "probable link" conclusion on diagnosed high cholesterol found no significant association with PFOA exposure.

Other studies that examined associations between PFAS exposure and lipid outcomes evaluated as continuous measures found inconsistent results. Among children, the lone prospective cohort study found that PFOA exposure was significantly associated with higher levels of total and LDL cholesterol only within the lowest tertile of prenatal PFOA exposure among 7-year-old girls in the general population of Avon, United Kingdom (U.K.) (Maisonet et al. 2015b). PFOA exposure was not significantly associated with total, LDL, or HDL cholesterol or triglycerides at higher prenatal PFOA exposure levels, and PFOS was not significantly associated with these endpoints at any prenatal exposure level. Heterogeneous findings were observed across the three cross-sectional studies of PFAS and lipids in children. A Mid-Ohio Valley study found significant associations between PFOA exposure and higher levels of total and LDL cholesterol and triglycerides, and between PFOS exposure and higher levels of total, LDL, and HDL cholesterol, among children under age 12 (Frisbee et al. 2010). However, these findings were countered by statistically null associations of PFOA and PFOS (as well as PFHxS, PFOSA, and PFNA, but not PFDA, which was significantly positively associated) with total and LDL cholesterol among children aged 9–11 years in Oswego County, New York. Among 8- to 10-year-old children in the general Danish population, PFOA and PFOS exposures were significantly associated with higher triglyceride levels only among overweight, not normal-weight, children (Timmermann et al. 2014).

Among adolescents in the prospective cohort study in Avon, U.K., PFOA and PFOS were inconsistently associated with total and LDL cholesterol levels by tertile of prenatal PFOA/PFOS exposure: significant positive associations were observed in the lowest or second tertile, whereas significant inverse associations were observed in the highest tertile of prenatal exposure (Maisonet et al. 2015b). This pattern—with an adverse effect at lower exposure levels and a beneficial effect at higher exposure levels—is not readily explained biologically. The other prospective cohort study of adolescents, based in Odense, Denmark, found no significant association between PFOS or PFOS exposure at ages 9 or 15 years and triglyceride levels at 15 years (Domazet et al. 2016). In cross-sectional studies of adolescents, significant positive associations of PFOA and PFOS exposure with higher levels of total cholesterol, LDL cholesterol, and/or triglycerides in studies of contaminated and general communities (Frisbee et al. 2010, Geiger et al. 2014a, Zeng et al. 2015) were countered by statistically null associations in another general community setting (Nelson et al. 2010). Isolated significant positive associations of PFBS and PFNA exposure with cholesterol levels (Zeng et al. 2015), and a significant inverse associations of PFHxS exposure with cholesterol (Nelson et al. 2010) and PFNA exposure with low HDL (Lin et al. 2009), were not replicated elsewhere.

In contrast to the relatively sparse data on PFAS exposure and lipid levels in children and adolescents, dozens of studies have been conducted among adults, although most used a cross-sectional design. Among the few prospective studies, associations between PFOA exposure and levels of total and/or LDL cholesterol or triglycerides were a mixture of significant positive (Olsen et al. 2003a, Sakr et al. 2007b, Fitz-Simon et al. 2013, Mattsson et al. 2015), inverse (MacPherson et al. 2011, Olsen et al. 2012a), and statistically null findings (Domazet et al. 2016). Likewise, associations of total and/or LDL cholesterol levels with PFOS exposure included significant positive (Fitz-Simon et al. 2013, Mattsson et al. 2015) and null findings (Olsen et al. 2012a), with a significant association with higher HDL cholesterol levels in the latter study. Results from cross-sectional studies do not lend clarity, as associations between PFOA exposure and levels of total or LDL cholesterol and/or triglycerides have been significantly positive (Olsen et al. 2003a, Sakr et al. 2007a, Costa et al. 2009, Steenland et al. 2009, Nelson et al. 2010, Shankar et al. 2011a, Fu et al. 2014, Mattsson et al. 2015, Skuladottir et al. 2015), significantly inverse (Lin et al. 2013a), null (Gilliland and Mandel 1996, Olsen et al. 2000, Emmett et al. 2006, Lin et al. 2009, Lin et al. 2011, White et al. 2011, Shankar et al. 2012, Wang et al. 2012, Fisher et al. 2013, Kishi et al. 2015, Starling et al. 2017), or heterogeneous by subgroup (Eriksen et al. 2013).

Associations with PFOS exposure likewise have been a variety of significantly positive (Steenland et al. 2009, Chateau-Degat et al. 2010, Shankar et al. 2011a, Mattsson et al. 2015, Skuladottir et al. 2015), significantly inverse (Kishi et al. 2015), null (Lin et al. 2009, Lin et al. 2011, Wang et al. 2012, Fisher et al. 2013, Lin et al. 2013a, Fu et al. 2014, Domazet et al. 2016, Starling et al. 2017), and internally heterogeneous findings (Olsen et al. 1999, Nelson et al.

2010, Eriksen et al. 2013). The generally close tracking of results for PFOA and PFOS illustrates the problem of distinguishing independent associations with multiple correlated PFAS, which were not mutually adjusted in these studies. Other PFAS besides PFOA and PFOS, including PFHxS, PFHpA, PFHpS, PFOSA, PFNA, PFDA, PFunDA, and PFdoDA, have been examined far less often in relation to lipid levels among adults, and scattered significant associations have not been observed consistently.

A recent U.S. EPA literature review stated that "there is evidence for positive associations between PFAS and dyslipidemia" in children, but did not conclude that the available epidemiologic evidence was sufficient to demonstrate a causal effect (Rappazzo et al. 2017).

In summary, the published epidemiologic literature is inconsistent with regard to the association between exposure to PFOA, PFOS, or other PFAS and lipid outcomes, including absolute lipid levels and dichotomized high cholesterol. No clear pattern of association was observed across occupational, contaminated-community, and general-community settings. Important methodological limitations precluded a causal interpretation of the significant associations observed in some studies. These limitations include the simultaneous measurement of circulating PFAS and lipid levels in most studies, enabling reverse causation or confounding by uncontrolled risk factors or shared physiological mechanisms; as well as the dearth of studies with repeated outcome measures and the lack of restriction, in most studies, to fasting blood specimens among subjects not taking cholesterol-lowering medications. Thus, as a whole, the published epidemiologic evidence does not establish a causal relationship between exposure to PFOA, PFOS, or other PFAS and altered lipid levels or diagnosed high cholesterol.

8.9 Epidemiology of PFAS and Liver Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on non-malignant liver (hepatic) outcomes in humans has not been established by the available published epidemiologic studies. I identified 29 published studies that reported the association between specific PFAS and liver outcomes, including non-specific biomarkers of liver function or damage (e.g., alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma glutamyl transferase (GGT), and bilirubin), liver cirrhosis, cholelithiasis or cholecystitis, any unspecified or chronic liver disease, and other endpoints, all in adults or mixed-age populations including adults (Gilliland and Mandel 1996, Olsen et al. 1999, Olsen et al. 2000, Alexander et al. 2003, Olsen et al. 2003a, Olsen et al. 2004, Emmett et al. 2006, Grice et al. 2007, Olsen and Zobel 2007, Sakr et al. 2007a, Sakr et al. 2007b, Anderson-Mahoney et al. 2008, Leonard et al. 2008, Costa et al. 2009, Lundin et al. 2009, Lin et al. 2010, Melzer et al. 2010, Gallo et al. 2012, Steenland and Woskie 2012, Wang et al. 2012, Olsen et al. 2012a, Consonni et al. 2013, Fisher et al. 2013, Yamaguchi et al. 2013, Fan et al. 2014, Jiang et al. 2014, Gleason et al. 2015, Steenland et al. 2015, Darrow et al. 2016) (Table 9 in Appendix A).

Seventeen studies were cross-sectional, nine were retrospective (with one including some cross-sectional analyses), and three were prospective (with cross-sectional analyses in one). The distribution of study settings was skewed toward occupational environments, where 19 studies were based (including two that also enrolled subjects from the surrounding contaminated community); four studies were based in the Mid-Ohio Valley community only, and six were based in general community settings.

Again, the cross-sectional design of most studies of liver outcomes makes their results susceptible to bias from reverse causality, and confounding by shared biological pathways could affect studies of cross-sectional, retrospective, or prospective design. Confounding also could occur from failure to control for influences on liver outcomes, such as diet and exercise (Africa et al. 2016). Because the liver plays a key role in PFAS elimination (Han et al. 2012), decrements in liver function—like those in kidney function—can introduce a spurious, non-causal association between higher PFAS levels and impaired liver function or liver disease. Cross-sectional and other studies that rely on liver biomarkers measured at a single point in time also suffer from outcome misclassification, since liver enzyme levels can vary within individuals in the short term, even fluctuating between levels considered clinically normal and abnormal (Rochling 2001, Lazo et al. 2008). Many studies found that the majority of subjects had liver biomarker levels within the normal laboratory reference range; thus, observed differences in such biomarkers between exposure groups could have been clinically unremarkable.

Associations of PFOA and PFOS exposure with liver outcomes in general community studies, all of which were cross-sectional, were not consistent. PFOA and PFOS were significantly positively associated with elevated levels of ALT and AST, but not GGT, in a Japanese study (Yamaguchi et al. 2013); both were significantly associated with elevated GGT and total bilirubin levels, and PFOA (but not PFOS) also was significantly associated with elevated ALT and AST levels, in a U.S. study (Gleason et al. 2015); and PFOA was associated with elevated levels of ALT and GGT, but not total bilirubin, while PFOS was associated with significantly lower levels of GGT and total bilirubin, but not ALT, in another U.S. general population study (Lin et al. 2010). By contrast, PFOA and PFOS were not significantly associated with ALT, AST, or total bilirubin in a study based in Tianjin, China (Jiang et al. 2014), or with liver disease in U.S. and Canadian studies (Melzer et al. 2010, Fisher et al. 2013). Exposure to PFHxS and PFNA also was significantly associated with higher levels of total bilirubin in one study (Lin et al. 2010) and bilirubin or selected liver enzymes in another study (Gleason et al. 2015), but not in a third (Jiang et al. 2014). Significant positive associations between other PFAS (PFHxA, PFHpA, PFDA, and PFDoDA) and total bilirubin (Jiang et al. 2014) were not replicated in other general-community studies.

Associations also were inconsistent in studies of residents of contaminated communities, including the Mid-Ohio Valley and Changshu City, Jiangsu Province, China. In the lone retrospective cohort study in a contaminated community, set in the Mid-Ohio Valley and also including DuPont Parkersburg plant workers, PFOA exposure was associated with a significant increase in ALT levels but a significant decrease in direct bilirubin levels, and no significant difference in GGT, enlarged/fatty/cirrhotic liver, or any liver disease (Darrow et al. 2016). In cross-sectional studies, PFOA exposure was variously associated with a significantly lower risk of abnormal AST (and not significantly associated with ALT, ALP, GGT, bilirubin, or self-reported liver disease) (Emmett et al. 2006); a significantly higher prevalence of self-reported unspecified "liver problems" in subjects aged 18–34 years, but not older adults (Anderson-Mahoney et al. 2008); a significant increase in levels of ALT and GGT, and risk of abnormal ALT and GGT levels, but not direct bilirubin (Gallo et al. 2012); no significant difference in ALT or AST levels (Wang et al. 2012); and no significant difference in risk of Gilbert syndrome phenotype, a disorder of bilirubin processing without other liver disease (Fan et al. 2014). The study of "liver problems" was particularly unreliable due to probable selection bias favoring inclusion of participants with perceived health problems (participation rate ≈ 2.5%), reliance on self-reported health outcomes with no clinical confirmation, a medico-legal context (class action litigation concerning PFOA health effects) that favored over-reporting of adverse health conditions, the absence of an appropriate comparison group from the same source population, the lack of information on potential confounders, and the crude classification of PFOA exposure based on broad geographic area of residence (Anderson-Mahoney et al. 2008). Isolated significant associations between PFOS and higher levels of ALT and direct bilirubin but not GGT (Gallo et al. 2012), and between PFHxA, PFNA, and PFDA and higher risk of Gilbert syndrome phenotype, as well as statistically null associations with PFPA, PFHxS, PFHpA, PFunDA, and PFdoDA (Fan et al. 2014), were not replicated in other studies based in contaminated communities with intermediate PFAS exposure levels.

PFOA and PFOS were the only PFAS studied in relation to liver outcomes in occupational settings. In the three prospective analyses of PFOA exposure, significant associations were observed with lower total bilirubin and higher AST levels, but not levels of ALT, ALP, or GGT, in DuPont Parkersburg workers (Sakr et al. 2007b); lower AST levels and, in combination with PFOS, lower total bilirubin levels, but not levels of ALT or ALP, in 3M Cottage Grove and Decatur workers (Olsen et al. 2012a); and not with any liver enzymes tested (ALT, AST, ALP, and GGT) among 3M Decatur and Antwerp workers (Olsen et al. 2003a). In the six retrospective cohort studies of PFOA workers (including the analysis that also covered Mid-Ohio Valley community residents), no significant excess was observed for any liver disease, non-hepatitis liver disease, enlarged liver/fatty liver/cirrhosis, or mortality from liver cirrhosis or chronic liver disease (Leonard et al. 2008, Lundin et al. 2009, Steenland and Woskie 2012, Consonni et al. 2013, Steenland et al. 2015, Darrow et al. 2016). The remaining five cross-sectional studies yielded inconsistent positive, inverse, and mostly statistically null associations

between PFOA exposure and liver biomarker levels, with no clear indication of a pattern of elevated biomarkers among studies or across time periods, geographic locations, or BMI groups within studies (Gilliland and Mandel 1996, Olsen et al. 2000, 2003a, Sakr et al. 2007a, Costa et al. 2009).

For PFOS, results in occupational studies also were not consistent, but mostly null. The two prospective cohort studies, conducted among Decatur and Antwerp plant workers (Olsen et al. 2003a) and Cottage Grove and Decatur workers (Olsen et al. 2012a), respectively, were fairly consistent in finding non-significant associations between PFOS exposure and liver outcomes. All results for longitudinal analyses of PFOS exposure in association with ALT, AST, ALP, and GGT in the first study were statistically null (although some significant positive associations were observed in cross-sectional analyses) (Olsen et al. 2003a), and all prospective associations with ALT, AST, ALP, and total bilirubin also were statistically null, except for a significant association of increases in both PFOA and PFOS exposure with lower total bilirubin level (Olsen et al. 2012a). One retrospective occupational cohort study among 3M Decatur plant workers found that PFOS was significantly associated with greater risk of episodes of care for biliary tract disorders and acute and chronic cholelithiasis among long-term chemical workers, but not for liver disorders or non-alcoholic liver cirrhosis (Olsen et al. 2004). The other two retrospective occupational cohort studies, also set in Decatur, Alabama, found no significant excess of cholelithiasis, cholecystitis, liver disease, or liver cirrhosis mortality among PFOS workers (Alexander et al. 2003, Grice et al. 2007). The lone cross-sectional analysis found significant associations between PFOS and lower total and direct bilirubin levels in some analyses, but non-significant associations with ALT, AST, ALP, and GGT (Olsen et al. 1999).

Overall, most associations tested between exposure to PFOA, PFOS, and other PFAS and liver outcomes were statistically non-significant, and significant associations with specific liver endpoints were offset by statistically null findings in most other studies, and often by null findings in subgroups within the same studies that reported sporadic positive associations. Associations did not follow a trend according to study setting, with stronger associations in more highly exposed populations. Nearly all associations with clinical liver disease were statistically null, suggesting that even if PFAS exposure were associated with changes in levels of liver enzymes, bilirubin, or other biomarkers (although such associations were not consistently observed), the differences were not clinically meaningful. Thus, based on the mostly statistically non-significant and otherwise inconsistent associations, the published epidemiologic evidence does not establish a causal relationship between PFOA, PFOS, or other PFAS exposure and liver outcomes in humans.

8.10 Epidemiology of PFAS and Metabolic Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on metabolic outcomes in humans has not been established by the available published epidemiologic studies. I identified 42 published studies that reported the association between specific PFAS and metabolic outcomes, including diabetes mellitus, metabolic syndrome, insulin resistance, glucose and insulin levels, homeostatic hormone levels, and other outcomes, in a range of study populations and age groups (Gilliland and Mandel 1993, Olsen et al. 2004, Anderson-Mahoney et al. 2008, Leonard et al. 2008, Costa et al. 2009, Lin et al. 2009, Lundin et al. 2009, MacNeil et al. 2009, Melzer et al. 2010, Nelson et al. 2010, Nolan et al. 2010, Lin et al. 2011, Shankar et al. 2011a, Halldorsson et al. 2012, Shankar et al. 2012, Steenland and Woskie 2012, Consonni et al. 2013, Fisher et al. 2013, Lin et al. 2013a, Jiang et al. 2014, Karnes et al. 2014, Lind et al. 2014, Raleigh et al. 2014, Timmermann et al. 2014, Kataria et al. 2015, Steenland et al. 2015, Zhang et al. 2015, Conway et al. 2016, Domazet et al. 2016, Lopez-Espinosa et al. 2016, Shapiro et al. 2016, Su et al. 2016, Zong et al. 2016, Christensen et al. 2016a, Christensen et al. 2016b, Kim et al. 2016b, Ashley-Martin et al. 2017, Fleisch et al. 2017, Minatoya et al. 2017, Starling et al. 2017, Valvi et al. 2017, Lind et al. 2017b) (Table 10 in Appendix A). Two of these studies evaluated metabolic outcomes in infants, four in children (including one that also included adolescents and adults, evaluated separately), five in adolescents evaluated as a separate group, and 36 in adults (≥ 20 years), including three studies of older adults aged ≥ 60 years. Twenty-three studies were cross-sectional, nine were retrospective, and ten included at least one component that was prospective in design (including one randomized controlled trial, one combined prospective cohort and cross-sectional study, and one combined prospective and retrospective cohort study). Twenty-four studies were set in general communities, three were based in fishing communities, six were conducted in the contaminated Mid-Ohio Valley community, and nine were occupational studies.

Conflicting results were observed in two prospective cohort studies of newborns, with a significant positive association between prenatal PFOS exposure and total adiponectin levels at birth in Hokkaido, Japan (Minatoya et al. 2017), but no significant association between prenatal PFOS exposure and newborn adiponectin levels in Canada (Ashley-Martin et al. 2017). Other results of these studies were consistently statistically null: PFOA exposure was not significantly associated with adiponectin levels in either study, nor was PFOA or PFOS exposure (or PFHxS exposure, which was measured only in the Canadian study) significantly associated with levels of leptin or, in the Japanese study, high-molecular-weight adiponectin at birth.

In contrast to the results from the Hokkaido newborn cohort, a prospective cohort study of 7-year-old children in Boston, Massachusetts, found significant inverse associations between exposure to PFOA, PFHxS, and PFDA and levels of leptin, as well as adiponectin for PFOA and PFHxS (and no significant association with exposure to PFOS or PFDA) (Fleisch et al.

2017). This study also found significant inverse cross-sectional associations, but not prospective associations, of PFOA, PFOS, PFHxS, PFNA, and PFDA levels with the homeostatic model assessment of insulin resistance (HOMA-IR) at 7 years. By contrast, PFOA and PFOS exposure were associated with higher cross-sectional HOMA-IR and insulin levels, but also higher HOMA for beta cell function (HOMA-beta), among overweight, but not normal-weight, children aged 8–10 years in Denmark (Timmermann et al. 2014). Adiponectin, leptin, and glucose levels were not significantly associated with PFOA or PFOS exposure in either overweight or normal-weight children in this study. In a cross-sectional study of 6- to 9-year-olds in the Mid-Ohio Valley, PFOA, PFOS, and PFNA exposure were associated with significantly lower insulin-like growth factor levels (Lopez-Espinosa et al. 2016). No significant cross-sectional association was detected between PFOA exposure and risk of type 2 diabetes among Mid-Ohio Valley children and adolescents aged < 20 years (Conway et al. 2016).

Associations of PFOA, PFOS, and other PFAS exposure (PFHxS, PFNA, and PFunDA) with metabolic outcomes also were inconsistent among adolescents. The only prospective cohort study of this topic, based in the general community of Odense, Denmark, found a significant inverse association of PFOA exposure at age 9 years with lower HOMA-beta at 15 years, but otherwise no significant associations of PFOA or PFOS exposure at 9 or 15 years with HOMA-IR, glucose, or insulin levels at 15 years (Domazet et al. 2016). The remaining four studies of adolescents, all cross-sectional in design, found heterogeneous results: a significant positive association of PFOA and PFOS exposure with HOMA-IR (Kataria et al. 2015), a significant inverse association between PFOA exposure and HOMA-beta among adolescent girls but not boys, and not with PFOS exposure (Nelson et al. 2010), and statistically null associations of PFOA and PFOS exposure with type 2 and unspecified diabetes, HOMA-IR, HOMA-beta, metabolic syndrome, and levels of glucose, insulin, leptin, and adiponectin (Lin et al. 2009, Conway et al. 2016). Associations with other PFAS (PFHxS, PFNA, or sums of PFAS) in these studies also were inconsistent and not independently confirmed (Lin et al. 2009, Nelson et al. 2010, Kataria et al. 2015).

Among adults, no consistent pattern of association between exposure to PFOA, PFOS, or other PFAS was observed. Significant positive associations between PFOA exposure and levels of insulin and leptin (but lower levels of adiponectin) were seen in 20-year-old women, but not in men, in a general-community prospective cohort study in Aarhus, Denmark (Halldorsson et al. 2012). A prospective cohort study in Michigan and Texas found a significant positive association between PFOA exposure and risk of gestational diabetes mellitus (Zhang et al. 2015), whereas a Canadian prospective cohort study did not (Shapiro et al. 2016); no significant association was seen between PFOA exposure and risk of gestational impaired glucose tolerance in the latter study. In the other four prospective cohort studies, no significant associations were seen between PFOA exposure and type 2 diabetes, metabolic syndrome, HOMA-IR, HOMA-beta, or levels of insulin or glucose (Karnes et al. 2014, Domazet et al.

2016, Kim et al. 2016b, Lind et al. 2017b). Likewise, in the five prospective cohort studies that measured PFOS exposure, significant positive associations were detected with HOMA-IR (continuous and dichotomized) and insulin levels among adults aged ≥ 60 years in Seoul, South Korea (Kim et al. 2016b), but no significant associations with PFOS exposure were observed in the other cohorts (Halldorsson et al. 2012, Zhang et al. 2015, Domazet et al. 2016, Shapiro et al. 2016, Kim et al. 2016b, Lind et al. 2017b). Significant associations with PFHxS exposure were observed in one study but not others (Shapiro et al. 2016), and with PFdoDA exposure in one study but not others (Kim et al. 2016b). No other PFAS (PFHxS, PFHpA, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, PFDA, PFunDA, and PFtrDA) were significantly associated with metabolic outcomes in prospective studies.

Among the 10 retrospective cohort studies of PFAS and metabolic outcomes—mostly occupational studies focused on PFOA or PFOS exposure and diabetes—the only significant associations detected with PFOA exposure were for diabetes mortality among Parkersburg plant workers (Leonard et al. 2008, Steenland and Woskie 2012) and among Cottage Grove plant workers (Lundin et al. 2009); however, the latter association was statistically non-significant in an updated analysis of the Cottage Grove cohort (Raleigh et al. 2014). Moreover, no significant association between PFOA exposure and diabetes incidence—a much more sensitive and specific indicator of disease etiology than diabetes mortality, which is strongly influenced by prognostic factors (Panzram 1987)—was seen among Parkersburg plant workers or in the broader Mid-Ohio Valley community (Karnes et al. 2014, Steenland et al. 2015). Diabetes mortality, episodes of medical care for diabetes, fasting glucose levels in non-diabetics, and gestational diabetes also were statistically unassociated with PFOA and/or PFOS exposure (and gestational diabetes also was statistically unassociated with PFHxS, PFNA, PFDA, and combined PFAS exposure) in other retrospective cohort studies (Gilliland and Mandel 1993, Olsen et al. 2004, Consonni et al. 2013, Karnes et al. 2014, Valvi et al. 2017).

The remaining 20 studies of metabolic outcomes in adults were cross-sectional, and therefore susceptible to bias from reverse causality and confounding by shared underlying physiological mechanisms that affect PFAS levels and metabolic changes. Additional confounders include the numerous risk factors for metabolic disorders, including demographic characteristics, diet, physical activity, and psychosocial factors (Abraham et al. 2007). Associations of type 2 or unspecified diabetes, pre-diabetes, metabolic syndrome, HOMA-IR, beta-cell impairment, proinsulin/insulin ratio, and levels of insulin, glucose, adiponectin, and glycohemoglobin with PFOA exposure included a small number of significant positive (Anderson-Mahoney et al. 2008, Lind et al. 2014) and significant inverse associations (Lin et al. 2009, MacNeil et al. 2009, Conway et al. 2016, Su et al. 2016, Starling et al. 2017), but predominantly statistically null findings (Costa et al. 2009, Lin et al. 2009, MacNeil et al. 2009, Melzer et al. 2010, Nelson et al. 2010, Nolan et al. 2010, Lin et al. 2011, Shankar et al. 2011a, 2012, Fisher et al. 2013, Lin et al. 2013a, Jiang et al. 2014, Lind et al. 2014, Zong et al. 2016, Christensen et al. 2016a,

Christensen et al. 2016b). Associations with PFOS exposure also were mostly statistically non-significant (Lin et al. 2009, Melzer et al. 2010, Nelson et al. 2010, Lin et al. 2011, Fisher et al. 2013, Lin et al. 2013a, Jiang et al. 2014, Lind et al. 2014, Zong et al. 2016, Christensen et al. 2016a, Christensen et al. 2016b, Starling et al. 2017), with a few significant positive (Lin et al. 2009, Shankar et al. 2011a, Su et al. 2016) and inverse associations (Conway et al. 2016). Associations with other PFAS evaluated, including PFHxA, PFHxS, PFHpS, PFOSA, PFNA, PFDA, PFunDA, and PFdoDA, with metabolic outcomes followed a similar pattern of isolated, generally unconfirmed statistically significant findings in either direction, and mostly statistically null results.

Recently, a U.S. EPA review of the epidemiologic literature on PFAS and cardiometabolic outcomes in children found that no clear association has been established with glucose metabolism: "Studies of glucose regulation in children have generally reported mixed effects, with limited agreement between studies" (Rappazzo et al. 2017).

In summary, the majority of published epidemiologic evidence on PFOA, PFOS, and other PFAS exposure and metabolic outcomes consists of statistically null findings, and scattered significant positive associations with a few endpoints were counterweighed by null or inverse results in other studies. Associations were not stronger in occupational settings than contaminated communities, or in the latter compared with general communities. Although most studies were cross-sectional in design, rendering them unreliable for causal inference, several prospective and retrospective cohort studies also mostly found no significant association between PFOA or PFOS exposure and risk of metabolic outcomes. Thus, given the statistically null findings for most metabolic outcomes and the inconsistent results for a few other endpoints, the available published epidemiologic evidence does establish a causal relationship between exposure to PFOA, PFOS, or other PFAS and metabolic outcomes in humans.

8.11 Epidemiology of PFAS and All-Cause Mortality

A causal effect of exposure to PFOA, PFOS, or other PFAS on all-cause mortality in humans has not been established by the available published epidemiologic studies. I identified eight published studies that reported the association between specific PFAS and all-cause mortality, all in retrospective occupational cohort studies of adults (Ubel et al. 1980, Gilliland and Mandel 1993, Alexander et al. 2003, Leonard et al. 2008, Lundin et al. 2009, Steenland and Woskie 2012, Consonni et al. 2013, Raleigh et al. 2014) (Table 11 in Appendix A). These included studies of workers at the 3M plant in Cottage Grove, Minnesota (Ubel et al. 1980, Gilliland and Mandel 1993, Lundin et al. 2009, Raleigh et al. 2014); workers at the 3M plant in Decatur, Alabama (Alexander et al. 2003); workers at the DuPont plant in Parkersburg, West Virginia (Leonard et al. 2008, Steenland and Woskie 2012); and workers at six North American and

European polytetrafluoroethylene plants, including the Parkersburg plant (Consonni et al. 2013).

None of the cohorts exhibited a significant excess of all-cause mortality overall. Although female workers at the Parkersburg plant had a higher risk of all-cause mortality compared with other DuPont workers in the U.S. geographic region, no significant difference was observed in comparison with the U.S. and West Virginia populations (Leonard et al. 2008), and no clear trends between PFOA exposure and mortality were observed among women in extended follow-up of this cohort (Steenland and Woskie 2012). No significant excess of mortality was observed among women in the Cottage Grove cohort (Gilliland and Mandel 1993). In fact, all-cause mortality was significantly lower in several cohorts of PFOA or PFOS workers compared with the general population (Gilliland and Mandel 1993, Alexander et al. 2003, Leonard et al. 2008, Lundin et al. 2009, Consonni et al. 2013, Raleigh et al. 2014). This difference probably was due largely to the healthy worker effect, that is, a tendency toward lower all-cause mortality rates among workers than in the general population, which includes severely ill and chronically disabled people who typically are excluded from employment (Porta et al. 2014).

In summary, based on the published epidemiologic data, no consistent or convincing significant excess of all-cause mortality was detected in association with exposure to PFOA or PFOS in occupational cohorts, which would be anticipated to have the highest exposure levels typically encountered. Thus, the available published epidemiologic evidence does not establish a causal relationship between exposure to PFOA or PFOS and overall risk of death in humans.

8.12 Epidemiology of PFAS and Neurodevelopmental Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on neurodevelopmental outcomes in humans has not been established by the available published epidemiologic studies. I identified 23 published studies that reported the association between specific PFAS and neurodevelopmental outcomes, including a range of behavioral, cognitive, psychomotor, and other neurological measures and disorders, among infants, toddlers (~13–24 months), and children (Fei et al. 2008a, Hoffman et al. 2010, Fei and Olsen 2011, Gump et al. 2011, Stein and Savitz 2011, Chen et al. 2013, Stein et al. 2013, Braun et al. 2014, Liew et al. 2014, Ode et al. 2014, Strom et al. 2014, Stein et al. 2014a, Donauer et al. 2015, Forns et al. 2015, Liew et al. 2015, Wang et al. 2015, Høyer et al. 2015a, Lien et al. 2016, Oulhote et al. 2016, Quaak et al. 2016, Vuong et al. 2016, Goudarzi et al. 2016b, Jeddy et al. 2017) (Table 12 in Appendix A). Specifically, seven studies evaluated neurodevelopmental outcomes in infants and/or toddlers, and the remaining 16 studies focused on children, mostly prior to adolescence. Most studies (19) included at least component with prospective exposure assessment, sometimes combined with a cross-sectional or retrospective cohort analysis; the remaining four studies were cross-sectional only. One study was conducted in the Faroe Islands fishing community and another

included subjects from the fishing community of Greenland; three studies were conducted in the Mid-Ohio Valley community; and the remaining 18 studies were conducted in general community settings with anticipated background exposure to PFAS.

Evaluating the consistency of epidemiologic studies of neurodevelopmental outcomes is challenging because a wide array of neurological tests are used in research studies, and the results of different tests of the same general domain or endpoint (e.g., attention deficit/hyperactivity disorder (ADHD) assessed according to a parent's report of a doctor's diagnosis of ADHD or use of ADHD medication, a computerized test of attention and impulsivity, or a parent or teacher's report of behavioral symptoms) may not be comparable. Moreover, some endpoints are assessed using different tools for different ages or languages/cultures, potentially making results non-comparable across these groups. Thus, for example, a significant positive association with a measure of inattention in toddlers may not necessarily translate to a positive association with ADHD in adolescents.

In the seven prospective cohort studies of infants and toddlers, no significant associations with exposure to PFOA, PFOS, or other PFAS (PFHxS, PFNA, and the sum of PFHxS, PFHpS, PFHA, PFOS, PFNA, PFDA, and PFunDA) were observed across a broad range of cognitive, behavioral, and psychomotor tests. None of the significant associations with PFOA exposure were independently replicated in other studies. These included significant associations of PFOA exposure with greater verbal comprehension score at 15 months to infants born to mothers > 30 years, lower vocabulary comprehension and production score at 15 months to infants born to mothers < 25 years, lower intelligibility score at 38 months, and greater communicative score at 38 months among infants born to mothers < 25 years, but not with these outcomes in other subgroups, and not with nonverbal communication or social developmental score at 15 months or language score at 38 months in an Avon, U.K., cohort (Jeddy et al. 2017); greater risk of a "hypotonic" profile, but not measures of attention, self-regulation, quality of movement, arousal, excitability, requiring special handling, lethargy, non-optimal reflexes, asymmetrical reflexes, hypotonicity alone, stress/abstinence, or an overall "difficult" profile, at 5 weeks in a Cincinnati, Ohio, cohort (Donauer et al. 2015); lower mental development, but not psychomotor development, at 6 months, and no difference in mental or psychomotor development at 18 months in a Hokkaido, Japan, cohort (Goudarzi et al. 2016b); and a lower score for externalizing problems in boys, but not in girls, and not an ADHD scale at 18 months in a Zwolle, Netherlands, cohort (Quaak et al. 2016). Three prospective cohorts of infants or toddlers found no significant associations of neurodevelopmental outcomes with PFOA exposure, including a Danish cohort study that measured developmental milestones for gross motor skills, fine motor skills, attention, cognition, or language at 18 months (Fei et al. 2008a); a northern Taiwan cohort study of overall, cognitive, language, gross motor, fine motor, social, or self-help development at 2 years (Chen et al. 2013); and a Norwegian study that examined a range of cognitive, psychomotor, and behavioral symptoms at 6, 12, and 24 months (Forns et al.

2015). Significant associations with PFOS included a mixture of higher verbal comprehension and lower vocabulary comprehension and production at 15 months, and lower language and intelligibility as well as higher intelligibility at 38 months, depending on maternal age subgroup, in the U.K. cohort (Jeddy et al. 2017); an assortment of higher and lower risk of meeting gross motor and language developmental milestones at 18 months in the Danish cohort (Fei et al. 2008a); a lower score on a test of overall development, but none of the components of that score, in the northern Taiwan cohort (Chen et al. 2013); and no significant association with any neurodevelopmental outcomes measured in the Cincinnati, Hokkaido, Norway, and Zwolle cohorts (Donauer et al. 2015, Forns et al. 2015, Quaak et al. 2016, Goudarzi et al. 2016b). Associations with other PFAS were assessed in only two cohorts, with inconsistent and mostly statistically null results (Quaak et al. 2016, Jeddy et al. 2017).

Results also were inconsistent and mostly statistically non-significant in studies of PFOA, PFOS, and other PFAS exposure in relation to neurodevelopmental outcomes in children. Each significant association with a specific neurodevelopmental outcome was counterbalanced by statistically null results in other studies, or else that outcome was not studied in other populations. For example, in prospective studies, PFOA exposure was variously associated with measures of significantly higher intelligence in the Mid-Ohio Valley community (Stein et al. 2013), but no significant difference in intelligence in central Taiwan (Wang et al. 2015); lower executive function rating among boys (but not girls) in the Mid-Ohio Valley community (Stein et al. 2014a), but no significant difference in executive function in Cincinnati, Ohio (Vuong et al. 2016); and significantly greater scores for problems on various subtypes of behavioral difficulties in two cohorts (e.g., peer relationship problems, autism screening, and internalizing problems in Faroe Island children (Oulhote et al. 2016), hyperactivity in Greenland children, and prosocial behavior problems in Ukraine children (Høyer et al. 2015a)), countered by significantly lower scores for problems evaluated using the same questionnaire in other cohorts (e.g., emotional symptoms and hyperactivity in Danish children (Fei and Olsen 2011), and internalizing problems, emotional symptoms, conduct problems, and peer problems in Taiwan children (Lien et al. 2016)). Exposure to PFOA (and PFOS) was significantly associated with greater risk of congenital cerebral palsy among boys, but not girls, in a prospective nested case-control study in Denmark (Liew et al. 2014), but cerebral palsy was not studied in other populations with respect to PFOA or PFOS exposure.

For prospectively assessed PFOS exposure, associations with neurodevelopmental outcomes also were mostly statistically null, and scattered significant associations with certain outcomes in some studies were not replicated elsewhere. For example, greater overall behavioral difficulties (and lower risk of abnormal emotional symptoms) were found in association with PFOS exposure among Greenland, Ukraine, and Poland children (Høyer et al. 2015a), but no such findings were observed using the same questionnaire in Faroe Island children (Oulhote et al. 2016), Taiwan children (Lien et al. 2016), or Danish children (Fei and Olsen 2011), among

whom a significant inverse association with prosocial behavior was countered by a significant positive association with prosocial behavior in the Taiwan cohort (Lien et al. 2016). Other PFAS were assessed less often than PFOA and PFOS in prospective studies, and also were not consistently associated with any specific neurodevelopmental outcomes.

Associations between PFOA exposure and ADHD, assessed in various ways, were significantly inverse for parent- or self-reported ADHD diagnosis among children and adolescents aged 5–18 years (Stein and Savitz 2011) and a computerized test for ADHD-type behaviors among children aged 6–12 years (Stein et al. 2013), but a variety of positive, inverse, and null, depending on child gender and parent- or teacher-provided data, using a survey for ADHD-like behaviors (Stein et al. 2014a) in the Mid-Ohio Valley community. In studies based in other communities, associations between PFOA exposure and ADHD or hyperactivity/inattention scores also were an assortment of some significantly positive (Hoffman et al. 2010, Fei and Olsen 2011, Liew et al. 2015, Høyer et al. 2015a) and significantly inverse results (Lien et al. 2016), counterbalanced by statistically null findings (Ode et al. 2014, Strom et al. 2014, Høyer et al. 2015a, Lien et al. 2016, Oulhote et al. 2016). Associations of PFOS exposure with ADHD or hyperactive/inattentive behaviors were mostly statistically null (Fei and Olsen 2011, Stein and Savitz 2011, Ode et al. 2014, Strom et al. 2014, Stein et al. 2014a, Liew et al. 2015, Høyer et al. 2015a, Lien et al. 2016, Oulhote et al. 2016), with a lone exception (Hoffman et al. 2010). Associations with other PFAS (PFHxS, PFHpS, PFNA, PFDA, and a sum of PFAS) also were mostly non-significant (Hoffman et al. 2010, Stein and Savitz 2011, Liew et al. 2014, Ode et al. 2014, Lien et al. 2016, Oulhote et al. 2016).

A recent review of the epidemiologic literature on PFAS and neurodevelopment conducted by U.S. EPA authors concluded that the two were not consistently associated: “Effects for observed neurological outcomes across studies are inconsistent; while some studies observe positive associations for both ADHD and neurodevelopment, there are also several studies that observe negative and null associations” (Rappazzo et al. 2017). Similarly, the authors of another literature review concluded that the evidence of an association between PFAS exposure and neurodevelopmental outcomes is “insufficient” due to “the few available studies and inconsistencies in their findings” (Vrijheid et al. 2016).

In summary, the published epidemiologic evidence on PFOA, PFOS, or other PFAS exposure and neurodevelopmental outcomes is inconsistent, and associations were not stronger in contaminated communities than general communities. Most studies found no significant association between PFAS exposure and various neurodevelopmental outcomes, including ADHD and various measures of inattentive and/or hyperactive behavior, as well as numerous other measures of cognitive, behavioral, psychomotor, and other related outcomes. Interpretation of the inconsistent findings is complicated by potential non-comparability of outcome measures among different study populations, as well as the measurement of PFAS

exposure at a single point in time that may not correspond to the etiologically relevant period or periods of susceptibility, if any, to neurodevelopmental effects. Thus, taking into account the inconsistency of the findings on neurodevelopmental outcomes. the generally statistically null results overall, and the potential for significant results to be explained by bias, chance, or confounding by sociodemographic, medical, behavioral, and environmental risk factors (Bellinger 2012), the published epidemiologic literature does not demonstrate a causal effect of exposure to PFOA, PFOS, or other PFAS on neurodevelopmental outcomes in infants, toddlers, or children.

8.13 Epidemiology of PFAS and Neurological Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on neurological outcomes in humans has not been established by the available published epidemiologic studies. I identified four published studies that reported the association between specific PFAS and neurological outcomes, including depressive symptoms, memory problems, and other cognitive limitations, in adults of all ages or older adults based on cutoffs of 50 years or higher (Gallo et al. 2013, Power et al. 2013, Berk et al. 2014, Shrestha et al. 2017) (Table 13 in Appendix A). All four studies were cross-sectional; two were conducted in general community settings, one was conducted in the Mid-Ohio Valley community, and one was conducted in an area in upstate New York, near the Hudson River, that was contaminated with polychlorinated biphenyls.

Exposure to PFOA, PFHxS, PFNA, and PFDA was significantly associated with lower risk of depressive symptoms in a general community study of U.S. adults aged ≥18 years, whereas exposure to PFOS, PFBS, PFHpA, PFOSA, Me-PFOSA-AcOH, Et-PFOS-AcOH, PFunDA, and PFdoDA was not significantly associated with depressive symptoms in this study (Berk et al. 2014). Depressive symptoms were not evaluated with respect to PFAS exposure in other studies.

The three other cross-sectional studies of neurological outcomes examined problems with memory and other cognitive performance among older adults. In the Mid-Ohio Valley, exposure to PFOA, PFOS, PFHxS, and PFNA was associated with significantly lower risk of short-term memory impairment, especially among subjects without diabetes, whereas the association was non-significant among diabetic subjects (Gallo et al. 2013). Similarly, in a study of the U.S. general population, exposure to PFOS, PFHxS, and PFNA (but not PFOA) was associated with significantly lower risk of problems due to difficulty remembering or periods of confusion, but this inverse association was observed only among diabetics not currently taking medication (Power et al. 2013); among medicated diabetics and non-diabetics, these associations were not significant. This study also found no significant associations between PFAS exposure and difficulties with activities of daily living due to senility, or performance on a digit-symbol substitution task designed to test cognitive performance. Finally, among older adults in the

Hudson River region, the majority of associations tested between PFOA and PFOS exposure and a series of 30 neuropsychological tests were statistically non-significant (Shrestha et al. 2017). Three significant associations with PFOA exposure were in a beneficial direction (higher California Verbal Learning Test t-score and lower perseverative error and perseverative response scores on the Wisconsin Card Sorting Test), as were the two significant associations with PFOS exposure (higher visual reproduction delayed recall score on the Wechsler Memory Scale, and higher Block Design Subtest score).

In summary, all four studies of neurological outcomes in relation to exposure to PFOA, PFOS, and other PFAS reported statistically significant associations in a beneficial direction toward lower risk of depressive symptoms and lower risk of impairment of memory and other cognitive performance in relation to higher PFAS exposure. No evident pattern was seen by study setting. However, due to the cross-sectional nature of these studies, as well as the potential for selection bias and uncontrolled confounding by diverse demographic, behavioral, and environmental risk factors (Baumgart et al. 2015), and the small number of studies with limited independent confirmation of findings, these results cannot reliably be interpreted as causal. Therefore, the available published epidemiologic evidence has not established a causal relationship between exposure to PFOA, PFOS, or other PFAS and human neurological outcomes in adults.

8.14 Epidemiology of PFAS and Pregnancy-Related Hypertension

A causal effect of exposure to PFOA, PFOS, or other PFAS on pregnancy-related hypertension in humans has not been established by the available published epidemiologic studies. I identified 10 published studies (including two separate studies reported in a single paper) that reported the association between specific PFAS and pregnancy-related hypertension, preeclampsia, and/or eclampsia[14] (Stein et al. 2009, Nolan et al. 2010, Savitz et al. 2012a, Savitz et al. 2012b, Darrow et al. 2013, Starling et al. 2014, Avanasi et al. 2016a, Avanasi et al. 2016b, 2016c) (Table 14 in Appendix A). All but one of these studies were conducted in the Mid-Ohio Valley community. Two studies were cross-sectional, one was a retrospective case-control study, five were retrospective cohort studies, one was a prospective nested case-control study, and one was a prospective cohort study.

In December 2011, the C8 Science Panel concluded that a "probable link" existed between PFOA exposure and pregnancy-induced hypertension (C8 Science Panel 2011). At the time, this conclusion was based on five studies in the Mid-Ohio Valley community (Stein et al. 2009,

[14] Preeclampsia is a complication of pregnancy characterized by hypertension and proteinuria after the 20th week of pregnancy. Eclampsia follows preeclampsia and is characterized by the onset of seizures.

Nolan et al. 2010, Savitz et al. 2012a, Savitz et al. 2012b) (including two studies described by Savitz et al. 2012a); thus, no independent confirmation of this association was available based on studies in other populations. At present, there remains no epidemiologic study outside of the Mid-Ohio Valley region that has detected a significant positive association between PFOA (or other PFAS) exposure and risk of pregnancy-related hypertension. This is because the one study from another region, a nested prospective case-control study of women in the Norway general population, found no significant association between exposure to PFOA, PFOS, PFHxS, PFHpS, PFNA, or PFDA and risk of preeclampsia, and a significant inverse association with exposure to PFunDA (Starling et al. 2014).

Even the studies in the Mid-Ohio Valley community did not consistently detect a significant positive association between PFOA or PFOS exposure and risk of pregnancy-related hypertension or preeclampsia/eclampsia. A retrospective study of pregnancies within the five years prior to PFOA and PFOS exposure measurement found no significant association between PFOA exposure and self-reported preeclampsia risk, while PFOS exposure was significantly positively associated with risk (Stein et al. 2009). The next study, a cross-sectional ecologic analysis of data from Ohio state birth records, showed no significant association between residence in the water district with the highest PFOA levels and risk of either pregnancy hypertension of eclampsia (Nolan et al. 2010). A retrospective case-control study based on Ohio and West Virginia state birth records revealed no significant difference in PFOA exposure between cases with pregnancy-related hypertension and controls without this condition (Savitz et al. 2012b). In the same paper, a separate cross-sectional analysis of linked birth records for C8 Health Project participants found a significant positive association between some categories of PFOA exposure—but not the highest quintile of exposure—and increased risk of pregnancy-related hypertension in some statistical models, but the results did not show a positive exposure-response gradient (Savitz et al. 2012b). In the same year, a retrospective cohort study of C8 Health Project participants found a combination of significant positive and statistically null associations between estimated PFOA exposure at the of pregnancy and risk of preeclampsia, with a significant positive association per 100-ng/mL increase in estimated serum PFOA, but a statistically nonsignificant difference in risk per interquartile increase in the natural logarithm of estimated serum PFOA, and no exposure-response trend across percentile categories (Savitz et al. 2012a). A prospective cohort study based on C8 Health Project participants – and the smallest study of this outcome—found significant positive associations between exposure to PFOA or PFOS and risk of pregnancy-induced hypertension during cohort-follow-up, based on linkage to state birth records (Darrow et al. 2013). Most recently, a series of sensitivity analyses of the retrospective cohort study (Savitz et al. 2012a) showed that uncertainty in estimated PFOA concentrations in drinking water (Avanasi et al. 2016a), geocoding of residential and workplace addresses (Avanasi et al. 2016b), and water ingestion rates and pharmacokinetic parameters (Avanasi et al. 2016c) resulted in little impact on the estimated association between PFOA exposure and preeclampsia risk, a moderate increase, and a modest attenuation,

respectively, with no significant association in the first and third studies, and a significant positive association in the second.

In summary, the association of exposure to PFOA (or PFOS, evaluated in three of nine studies) with pregnancy-related hypertension, preeclampsia, or eclampsia was inconsistent across multiple studies within the Mid-Ohio Valley population, and it was not confirmed in any independent study populations. In light of methodological limitations including error in PFOA exposure estimation, potential confounding by various behavioral and environmental risk factors (Sheppard and Khalil 2010), and temporal ambiguity in cross-sectional studies, the significant positive associations seen in the Mid-Ohio Valley population cannot reliably be interpreted as being causal. Therefore, the available published epidemiologic studies do not demonstrate a causal association between PFOA or PFOS exposure and pregnancy-related hypertension, preeclampsia, or eclampsia in women.

8.15 Epidemiology of PFAS and Female Reproductive Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on female reproductive outcomes in humans has not been established by the available published epidemiologic studies. I identified 54 published studies that reported the association between specific PFAS and female reproductive outcomes, including reproductive hormone levels, fertility or fecundity, pregnancy outcomes, menarche/menopause/menstrual cycle characteristics, endometriosis, and lactation (not including pregnancy-related hypertension, discussed in the preceding section), in girls and women in various populations (Olsen et al. 2004, Grice et al. 2007, Fei et al. 2009, Rylander et al. 2009, Stein et al. 2009, Nolan et al. 2010, Fei et al. 2010b, Christensen et al. 2011, Governini et al. 2011, Lopez-Espinosa et al. 2011, White et al. 2011, Knox et al. 2011b, Buck Louis et al. 2012, Fei et al. 2012, Vestergaard et al. 2012, Wu et al. 2012, Savitz et al. 2012a, Savitz et al. 2012b, Whitworth et al. 2012b, Buck Louis et al. 2013, Caserta et al. 2013, Kristensen et al. 2013, Darrow et al. 2014, Ding et al. 2014, Jorgensen et al. 2014, La Rocca et al. 2014, Lyngso et al. 2014, Taylor et al. 2014, Stein et al. 2014b, Bae et al. 2015, Barrett et al. 2015, Cariou et al. 2015, Jensen et al. 2015, Lewis et al. 2015, Tsai et al. 2015, Vélez et al. 2015, Bach et al. 2015a, Maisonet et al. 2015a, Bach et al. 2015b, Buck Louis et al. 2016, Campbell et al. 2016, Itoh et al. 2016, Lopez-Espinosa et al. 2016, Romano et al. 2016, Whitworth et al. 2016, Zhou et al. 2016, Dhingra et al. 2016a, Crawford et al. 2017, Dhingra et al. 2017, Lum et al. 2017, McCoy et al. 2017, Wang et al. 2017a, Zhou et al. 2017a, Timmermann et al. 2017b) (Table 15 in Appendix A). Six of these studies evaluated female reproductive outcomes in infants, seven in children or adolescents, and 43 in adult women (including two that evaluated adolescent girls separately). Twenty studies were cross-sectional, five were retrospective case-control studies, 11 were retrospective cohort studies (including one with a cross-sectional component), and 18 were prospective cohort or case-control studies (including one with a retrospective component). Thirty-seven studies were conducted in general community settings, three were conducted at

least partially in fishing communities, 12 were conducted in PFAS-contaminated communities, and two were conducted in the occupational setting of the 3M Decatur plant.

Studies of PFAS exposure in relation to female reproductive outcomes may yield spurious associations because of underlying physiological processes that affect circulating PFAS levels and reproductive status in women. For example, studies of the relationship between PFAS exposure and time to pregnancy (fecundity) are susceptible to bias, leading to overestimated positive associations, because past pregnancies tend to decrease circulating PFAS, such that multiparous women with longer time to pregnancy will have had more time to accumulate stored levels of PFAS (Glynn et al. 2012, Whitworth et al. 2012b). Likewise, the association between PFAS exposure and shorter duration of breastfeeding, especially among multiparous women, may be inflated because PFAS are excreted in breast milk (Fei et al. 2010b, Mondal et al. 2014). Moreover, studies using physiologically based pharmacokinetic modeling to evaluate extraneous influences on observed associations between PFAS exposure and female reproductive outcomes have found evidence of bias due to reverse causality, shared physiological mechanisms, or alternative risk factors for various female reproductive endpoints. For example, dilution of circulating PFAS levels through pubertal growth and a new route of excretion (menstruation) could explain part of an observed association between PFAS exposure and delayed age at menarche (Wu et al. 2015). Conversely, loss of PFAS excretion due to menopause could explain a substantial amount of an observed association between PFAS exposure and early menopause (Ruark et al. 2017). Another study found that confounding by oral conceptive use could explain a small amount of an observed association between PFAS exposure and greater risk of endometriosis (Ngueta et al. 2017). Finally, numerous potential environmental and behavioral risk factors for female reproductive outcomes have been identified, and could act as confounders of observed associations (Sharara et al. 1998). Thus, apparent associations between PFAS levels and these and other female reproductive outcomes may be driven by non-causal explanations.

Fecundity and fertility were evaluated in association with PFAS exposure in 20 studies, including five cross-sectional, three retrospective case-control, six retrospective cohort studies, and six prospective cohort studies. Six studies from four independent study populations examined the association between PFAS exposure and fecundity among primiparous women, thereby restricting the population to women among whom past pregnancies would not distort the relationship between circulating PFAS levels and subsequent time to pregnancy (Fei et al. 2012, Whitworth et al. 2012b, Jorgensen et al. 2014, Bach et al. 2015a, Bach et al. 2015b, Whitworth et al. 2016). Three studies of a Norwegian pregnancy cohort found conflicting results, with two analyses showing no significant association between exposure to PFOA, PFOS, or other PFAS (PFHxS, PFHpS, PFOSA, PFNA, PFDA, PFunDA, PFdoDA, and PFtrDA) and fecundability ratio (probability of pregnancy within 12 months) or subfecundity among primiparous women (Whitworth et al. 2012b, Whitworth et al. 2016), but another

showing inverse associations of PFOA and PFOS exposure with fecundability among both primiparous and multiparous women, although infertility was significantly positively associated with PFOA exposure only among multiparous women (Fei et al. 2012). Results from the other three study populations were mostly consistent with no association between PFOA, PFOS, or other PFAS exposure and fecundability among primiparous women. In Greenland, Ukraine, and Poland, PFOA exposure was significantly associated with higher fecundability ratio and not significantly associated with fertility, and exposure to PFHxS, PFOS, and PFNA were not significantly associated with either outcome, among primiparous women (Jorgensen et al. 2014). In a pregnancy cohort based in Aarhus, Denmark, no significant association was detected between exposure to PFOA, PFOS, or other PFAS and either fecundability ratio or infertility in nulliparous women (Bach et al. 2015a), whereas a Danish national pregnancy cohort showed heterogeneous associations, with no significant association between PFOA or PFOS exposure and fecundability ratio among primiparous women in one sample, but significant inverse associations in the other (Bach et al. 2015b).

These results are in line with the inconsistent, mostly statistically null results for the association between PFAS exposure and fecundity or fertility in studies that were not restricted to primiparous women. That is, most studies found no significant association between exposure to PFOA, PFOS, or other PFAS (PFHxS, PFHpS, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, PFDA, and combinations) and fecundity, infertility, or parity (Olsen et al. 2004, Rylander et al. 2009, Caserta et al. 2013, La Rocca et al. 2014, Cariou et al. 2015, Crawford et al. 2017, Lum et al. 2017). Among the remaining studies, significant positive associations with subfecundity and/or infertility were reported for PFOA in two analyses of the same Norwegian cohort (Fei et al. 2009, Ding et al. 2014) and in a Canadian cohort (Vélez et al. 2015); PFOS in the Norwegian cohort (Fei et al. 2009); PFHxS in the Canadian cohort (Vélez et al. 2015), countered by a significant association with greater fecundability ratio in the Danish cohort (Vestergaard et al. 2012); and PFOSA in a Michigan and Texas cohort (Buck Louis et al. 2013). A cross-sectional study in France found that exposure to PFHxS, but not PFOA, PFOS, or PFNA, was significantly associated with lower parity, but no significant association was found between exposure to any of these four PFAS and increased time to pregnancy (Cariou et al. 2015). Collectively, statistically significant associations comprised the minority of all associations tested, and were not confirmed in most studies.

Two studies found varied, mutually inconsistent associations with other fertility-related outcomes, such as oocyte fertilization rate, number of oocytes retrieved and transferred, follicle count, and percent fertilization, among women undergoing *in vitro* fertilization (Governini et al. 2011, McCoy et al. 2017). No further studies of *in vitro* fertilization were available to confirm these results.

Thirteen studies, including 10 cross-sectional studies and three prospective cohort studies, examined PFAS exposure in relation to levels of reproductive hormones, including estradiol, progesterone, prolactin, total and/or free testosterone, sex hormone binding globulin, follicle-stimulating hormone, luteinizing hormone, dehydroepiandrosterone, and anti-Müllerian hormone, in newborns, girls, and women. Because levels of both PFAS and reproductive hormones fluctuate over short and long time scales in girls and women, with different patterns by age (Nelson et al. 1980, De Hertogh et al. 1992), the one-time measurement of exposures and outcomes in these studies limits their ability to capture relationships accurately. The majority of observed associations between exposure to PFOA, PFOS, or other PFAS (PFBS, PFHxA, PFHxS, PFOSA, Me-PFOSA, PFNA, PFDA, PFunDA, PFdoDA, PFteDA, and combinations of PFAS) and female reproductive hormone levels were statistically non-significant.

Significant associations with PFOA exposure were observed in only two studies, one of which found a significant inverse association with sex hormone binding globulin level among Taiwanese girls aged 12–17 years (Tsai et al. 2015), and the other of which found a significant positive association, but no exposure-response trend, with total testosterone level in 15-year-old girls in Avon, U.K. (Maisonet et al. 2015a). However, these findings were offset by statistically null results for sex hormone binding globulin, total testosterone, and other reproductive hormone levels in relation to PFOA exposure in the same and other studies (Lopez-Espinosa et al. 2011, Knox et al. 2011b, Kristensen et al. 2013, Barrett et al. 2015, Lewis et al. 2015, Tsai et al. 2015, Maisonet et al. 2015a, Itoh et al. 2016, Lopez-Espinosa et al. 2016, Zhou et al. 2016, Crawford et al. 2017, McCoy et al. 2017).

In the same set of studies, PFOS exposure was significantly associated with lower follicular-phase estradiol level and luteal-phase progesterone level in nulliparous, but not parous, women in Tromsø, Norway (Barrett et al. 2015); lower estradiol levels in Mid-Ohio Valley women over age 42 years (but not ages 18–42 years) and peri-pubertal girls (Lopez-Espinosa et al. 2011, Knox et al. 2011b) and South Carolina women (based on PFOS in plasma, but not follicular fluid) (McCoy et al. 2017); lower total testosterone levels among girls in the Mid-Ohio Valley (Lopez-Espinosa et al. 2016) and Taipei, Taiwan (Tsai et al. 2015); greater total testosterone levels among adolescent girls in Avon, U.K. (Maisonet et al. 2015a); and lower progesterone and prolactin levels among newborn girls in Hokkaido, Japan (Itoh et al. 2016). Although four studies reported significant inverse associations with estradiol levels, more studies found no significant association between PFOS exposure and estradiol or estrogen (Kristensen et al. 2013, Tsai et al. 2015, Itoh et al. 2016, Lopez-Espinosa et al. 2016, Zhou et al. 2016), reflecting a general pattern of statistically null associations with reproductive hormone levels in females. Similarly, observed associations with other PFAS were mostly statistically non-significant, and the few significant findings were not confirmed across multiple studies (Lopez-Espinosa et al. 2011, White et al. 2011, Knox et al. 2011b, Kristensen et al. 2013, Barrett et al. 2015, Lewis et

al. 2015, Tsai et al. 2015, Maisonet et al. 2015a, Itoh et al. 2016, Lopez-Espinosa et al. 2016, Zhou et al. 2016, Crawford et al. 2017, McCoy et al. 2017).

Twelve studies of PFAS exposure and pregnancy outcomes mostly detected no significant results, and statistically significant associations were not consistently observed across studies. PFOA exposure was associated with a significantly increased risk of stillbirth in one cross-sectional study in a PFAS-contaminated community in China (Wu et al. 2012), but not significantly associated with pregnancy loss, miscarriage, stillbirth, or spontaneous abortion in other studies in general community, contaminated community, and occupational settings (Stein et al. 2009, Savitz et al. 2012a, Savitz et al. 2012b, Darrow et al. 2014, Jensen et al. 2015, Buck Louis et al. 2016). PFOS exposure was associated with a significantly increased risk of miscarriage among Mid-Ohio Valley women in one study (Darrow et al. 2014), but no significant association with pregnancy loss, miscarriage, stillbirth, or spontaneous abortion was seen in other studies set in general communities or PFOS plants (Olsen et al. 2004, Grice et al. 2007, Stein et al. 2009, Jensen et al. 2015, Buck Louis et al. 2016). No consistent associations were observed between exposure to other PFAS and pregnancy loss or miscarriage (Jensen et al. 2015, Buck Louis et al. 2016). Of the five studies (one cross-sectional and four retrospective cohort) that evaluated PFOA and/or PFOS exposure and birth defects, four found no significant associations (Olsen et al. 2004, Nolan et al. 2010, Savitz et al. 2012a, Savitz et al. 2012b) and one found a single significant association between PFOA exposure and greater risk of brain birth defects (but not gastrointestinal, kidney, craniofacial, eye, limb, genitourinary, or heart birth defects) that was not replicated elsewhere (Stein et al. 2014b).

Ten studies evaluated the relation between PFAS exposure and menstrual cycle outcomes, including age at menarche, age at menopause, and menstrual cycle length or regularity, but few independent studies evaluated any given outcome. Three studies in the Mid-Ohio Valley found significant associations between PFOA exposure and earlier age at menopause in some analyses, but results varied by exposure metric, subgroup of women, and statistical model (Knox et al. 2011b, Dhingra et al. 2016a, Dhingra et al. 2017). Only one independent study examined the same association, and it also found a significant positive association between PFOA exposure and earlier age at menopause in the general U.S. population (Taylor et al. 2014). This study additionally found significant positive associations between exposure to PFOS, PFHxS, and PFNA and earlier age at menopause. A significant positive association with PFOS exposure also was seen in a cross-sectional study of Mid-Ohio Valley women (Knox et al. 2011b), whereas the relation between PFHxS and PFNA exposure and age at menopause was not studied elsewhere.

Three studies of PFOA or PFOS exposure in relation to age at menarche reported varied results, including associations between PFOA exposure and later age at menarche in studies in the Mid-Ohio Valley (Lopez-Espinosa et al. 2011) and Aarhus, Denmark (Kristensen et al. 2013), but

not Avon, U.K. (Christensen et al. 2011); and an association between PFOS exposure and later age at menarche in the Mid-Ohio Valley (Lopez-Espinosa et al. 2011), but not the other two settings (Christensen et al. 2011, Kristensen et al. 2013). The U.K. study also found no significant association of age at menarche with exposure to PFHxS, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, PFDA, sulfonamide esters, sulfonates, or carboxylates. As discussed earlier, interpretation of observed associations between PFAS levels and age at menopause or menarche is complicated by the high potential for reverse causality or confounding by shared physiological processes (Wu et al. 2015, Ruark et al. 2017).

Menstrual cycle length and regularity, which also may affect PFAS levels rather than vice versa, were inconsistently associated with exposure to PFOA, PFOS, and other PFAS (PFBS, PFHxS, PFHpA, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, PFDA, PFunDA, and PFdoDA). For instance, PFOA exposure was significantly associated with shorter menstrual cycle length in a prospective cohort study in Michigan and Texas (Lum et al. 2017); longer cycle length in cross-sectional studies in Greenland, Ukraine, and Poland (Lyngso et al. 2014) and Shanghai, China (Zhou et al. 2017a); and no difference in cycle length in a prospective cohort study in Aarhus, Denmark (Kristensen et al. 2013). The same studies revealed a significant association between PFOS exposure and longer menstrual cycle in Shanghai (Zhou et al. 2017a) but not the other three settings (Kristensen et al. 2013, Lyngso et al. 2014, Lum et al. 2017); and no significant associations with other PFAS were detected in more than one study.

Four studies in general communities in the U.S., Italy, and China—two retrospective case-control and two cross-sectional in design—collectively found inconsistent and mostly statistically null associations between PFOA, PFOS, or other PFAS exposure and risk of endometriosis or endometriosis-related infertility. A significant positive association with PFOA exposure was observed in a U.S. study (Campbell et al. 2016), but not the other three (Buck Louis et al. 2012, Caserta et al. 2013, Wang et al. 2017a). For PFOS, a significant positive association in the same U.S. study (Campbell et al. 2016) was counterbalanced by statistically non-significant findings in Italy (Caserta et al. 2013) and Utah and California (Buck Louis et al. 2012), and a significant inverse association among women without other gynecologic pathology in Hangzhou, China (Wang et al. 2017a). Isolated significant positive associations with exposure to PFNA (Campbell et al. 2016) or PFBS (Wang et al. 2017a) were not replicated elsewhere, nor were significant inverse associations with exposure to PFHxS, PFHpA, PFNA, PFunDA, or PFdoDA (Wang et al. 2017a).

Three prospective cohorts and one cross-sectional study found significant associations between exposure to PFOA or PFOS (and, in one study, PFNA or PFDA (Timmermann et al. 2017b)) and shorter duration of breastfeeding or greater risk of stopping breastfeeding by 3 or 6 months (Fei et al. 2010b, Cariou et al. 2015, Romano et al. 2016, Timmermann et al. 2017b). The

authors of the cohort studies suggested that the associations could be due to an effect of PFAS exposure on reduced ability to lactate, but that confounding (e.g., by sociocultural, medical, behavioral, and nutritional influences on PFAS levels and breastfeeding, which may be terminated for reasons other than low milk production) also could be responsible for the observed relationships. Some authors also acknowledged that a spurious association between PFAS exposure and shorter duration of breastfeeding could arise because among multiparous women because PFAS are excreted in breast milk, leading to lower PFAS levels among women who breastfed longer with past and current children (Fei et al. 2010b). Thus, despite the observation of these associations across four studies, the results cannot reliably be interpreted as indicating causal relationships due to the high potential for reverse causation.

In a recent U.S. EPA review of the epidemiologic evidence on PFAS and indicators of pubertal onset, the authors stated, "The most consistent evidence is for later age at menarche associated with either PFOA or PFOS exposure or both"; however, they concluded that, overall, a consistent association was not established by the few available studies: "The six studies of pubertal onset indicators have generally mixed results and varied study design" (Rappazzo et al. 2017).

A recent systematic literature review on epidemiologic studies of PFAS and measures of human fertility reached the conclusion that a causal association between PFOA or PFOS exposure and reduced fecundability in women has not been demonstrated: "Neither in the male nor female studies did the studies with the highest average exposure levels demonstrate stronger findings. For PFOS and PFOA, the literature indicates a possible association with female fecundability mainly among parous women, which is likely to be spurious. The lack of association in most studies in nulliparous women and from pregnancy planner studies *failed to support a causal relationship* between PFAS exposure and fertility in women" (Bach et al. 2016b) (emphasis added).

In summary, based on the available published epidemiologic literature, the female reproductive outcomes evaluated were not consistently associated with exposure to PFOA, PFOS, or other PFAS, nor did any evident pattern of association emerge across study settings. Most associations with fertility/fecundity, reproductive hormone levels, pregnancy outcomes, and endometriosis were statistically non-significant, and the minority of associations that were statistically significant were not supported by other findings or not independently replicated, and susceptible to confounding or other bias. Observed associations with menstrual cycle characteristics and lactation were plausibly explained by non-causal mechanisms involving PFAS excretion in menstrual fluid and breast milk, respectively. Taking together the non-significant associations with most female reproductive outcomes, the sparse and largely unconfirmed associations with a few conditions, and the non-causal explanations for several observed associations, the weight of epidemiologic evidence does not establish a causal

relationship between exposure to PFOA, PFOS, or other PFAS and female human reproductive outcomes.

8.16 Epidemiology of PFAS and Male Reproductive Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on male reproductive outcomes in humans has not been established by the available published epidemiologic studies. I identified 28 published studies that reported the association between specific PFAS and male reproductive outcomes, including semen and sperm characteristics, infertility, levels of reproductive hormones and prostate-specific antigen, reproductive tract disorders, and other outcomes, in various groups of boys or men (Olsen et al. 1998, Olsen et al. 2004, Grice et al. 2007, Costa et al. 2009, Joensen et al. 2009, Lopez-Espinosa et al. 2011, Kvist et al. 2012, Raymer et al. 2012, Specht et al. 2012, Toft et al. 2012, Joensen et al. 2013, Vested et al. 2013, Jorgensen et al. 2014, Vesterholm Jensen et al. 2014, Bae et al. 2015, Buck Louis et al. 2015, Den Hond et al. 2015, Ducatman et al. 2015, Governini et al. 2015, La Rocca et al. 2015, Lenters et al. 2015, Lewis et al. 2015, Tsai et al. 2015, Itoh et al. 2016, Lopez-Espinosa et al. 2016, Toft et al. 2016, Zhou et al. 2016, Goudarzi et al. 2017a) (Table 16 in Appendix A). Seventeen studies were cross-sectional, three were retrospective case-control studies, three were retrospective cohort studies, and five were prospective cohort or nested case-control studies. Sixteen studies were conducted in general community settings, five were conducted in multiple communities including a fishing community (Greenland) and general communities (Ukraine and Poland), three were conducted in the Mid-Ohio Valley community, and four were conducted in occupational settings.

Levels of reproductive hormones, including testosterone (total, free, or bound), estradiol, sex hormone binding globulin, luteinizing hormone, follicle-stimulating hormone, prolactin, inhibin B, dehydroepiandrosterone, androstenedione, 17-hydroxyprogesterone, and ratios of hormones, were the male reproductive outcomes studied most frequently with respect to PFAS exposure. PFAS levels and reproductive hormone levels, which fluctuate over time depending in part on age (Couwenbergs et al. 1986, Plymate et al. 1989), were each measured only once in these studies; therefore, observed exposures and outcomes should be considered as assessed with error. Male reproductive hormone levels generally were not significantly associated with exposure to PFOA, PFOS, or other PFAS (PFBS, PFHxA, PFHxS, PFHpS, PFOA+PFOS, PFNA, PFDA, PFdoDA, and PFteDA) in four prospective studies, one retrospective case-control study, and twelve cross-sectional studies of these endpoints. Amid the mostly statistically null findings, a few significant associations were observed for PFOA exposure with greater sex hormone binding globulin levels in men in Poland but not Greenland, Ukraine, or all three areas combined (Specht et al. 2012); with greater luteinizing hormone and follicle-stimulating hormone levels in Danish men (Vested et al. 2013); with greater free testosterone and luteinizing hormone levels in North Carolina men (Raymer et al. 2012); with greater

prolactin and 17-hydroxyprogesterone levels in 3M Cottage Grove plant workers (Olsen et al. 1998); with greater estradiol levels in adolescent boys in Taiwan (Zhou et al. 2016); with lower total testosterone levels in Mid-Ohio Valley boys (Lopez-Espinosa et al. 2016); and with greater inhibin levels and lower dehydroepiandrosterone levels in newborn boys in Hokkaido and Sapporo, Japan, respectively (Itoh et al. 2016, Goudarzi et al. 2017a). However, these associations were not confirmed by statistically null results in the same and other studies (Olsen et al. 1998, Costa et al. 2009, Joensen et al. 2009, Raymer et al. 2012, Specht et al. 2012, Joensen et al. 2013, Vested et al. 2013, Den Hond et al. 2015, Lenters et al. 2015, Lewis et al. 2015, Tsai et al. 2015, Itoh et al. 2016, Zhou et al. 2016, Goudarzi et al. 2017a).

The same was true of the few significant associations with PFOS exposure. These included significant inverse cross-sectional associations with testosterone level among northern Taiwanese adolescent boys (Zhou et al. 2016), and with both testosterone and several ratios involving testosterone among Danish men (Joensen et al. 2013); significant inverse associations with total and free testosterone levels and estradiol levels in two cross-sectional Mid-Ohio Valley studies of boys (Lopez-Espinosa et al. 2011, Lopez-Espinosa et al. 2016); and a significant inverse association with follicle-stimulating hormone level among adolescent boys in Taipei, Taiwan (Tsai et al. 2015). However, these associations were counterbalanced by null results for these and other reproductive hormone levels in the same and additional studies of PFOS exposure (Joensen et al. 2009, Raymer et al. 2012, Specht et al. 2012, Joensen et al. 2013, Vested et al. 2013, Den Hond et al. 2015, Lenters et al. 2015, Lewis et al. 2015, Tsai et al. 2015, Zhou et al. 2016). Three studies of newborn boys found several significant associations between maternal PFOS exposure and levels of selected reproductive hormones, but the specific hormones and direction of association differed among the studies (e.g., a significant positive association with testosterone level in Danish boys, but no significant association with testosterone level and a significant inverse association with testosterone:estradiol ratio in Hokkaido boys; and conflicting results for androstenedione and dehydroepiandrosterone levels between Danish and Sapporo boys) (Itoh et al. 2016, Toft et al. 2016, Goudarzi et al. 2017a). Isolated significant associations were observed between exposure to other PFAS (PFHxA, PFHxS, PFNA, and PFDA) and estradiol or testosterone levels in Danish men (Joensen et al. 2013) and Taiwanese adolescent boys (Zhou et al. 2016), but these associations were not evaluated elsewhere.

Other male reproductive outcomes evaluated in several studies with respect to PFAS exposure were semen and sperm characteristics. In eight cross-sectional studies and one case-control study of men in general communities, with two studies also including men from a Greenland fishing community, associations between measures of semen quality or sperm morphology and exposure to PFOA, PFOS, and other PFAS (PFHxS, PFHpS, PFOA+PFOS, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, PFDA, PFunDA, and PFdoDA) were mostly statistically null, and the small number of statistically significant associations were inconsistent

across studies. With respect to PFOA exposure, the few observed significant associations with semen quality or sperm morphology were sometimes in a generally adverse direction (e.g., lower sperm concentration, total sperm count, and percentage progressive sperm (Vested et al. 2013); lower number and percent of morphologically normal sperm in relation to PFOA+PFOS exposure, but not PFOA exposure alone (Joensen et al. 2009)) and sometimes in a generally beneficial direction (e.g., higher percentage of motile sperm (Toft et al. 2012); greater sperm curvilinear velocity and acrosome area of sperm head, and lower percentage of sperm with coiled tail (Buck Louis et al. 2015)). Five studies found no significant associations of PFOA exposure with any measures of semen quality or sperm morphology (Raymer et al. 2012, Joensen et al. 2013, Den Hond et al. 2015, Governini et al. 2015, Lenters et al. 2015). Associations of PFOS exposure with semen and sperm characteristics also were predominantly statistically non-significant (Raymer et al. 2012, Joensen et al. 2013, Vested et al. 2013, Den Hond et al. 2015, Governini et al. 2015, Lenters et al. 2015), in the direction of a beneficial impact in one study (Buck Louis et al. 2015), in the direction of an adverse impact in another study (Joensen et al. 2009), and a mixture of beneficial and adverse directions in a third study (Toft et al. 2012). No significant associations between exposure to other PFAS and specific measures of semen quality or sperm morphology were consistently detected in more than one study.

Two studies of DNA fragmentation and markers of apoptosis (programmed cell death) in sperm cells found mostly statistically null results. One reported no significant associations with exposure to any PFAS (Lenters et al. 2015), whereas the other (an earlier study based in the same population) found a significant positive association between PFOA exposure and greater DNA fragmentation based on one assay but not another among Greenland men (but not among men in Ukraine, Poland, or overall); and a significant positive association between PFOS exposure and greater positivity of sperm cells for the Fas marker, but not the Bcl-xL marker, among men in Poland (but not Greenland, Ukraine, or overall) (Specht et al. 2012). A third study of DNA damage in sperm cells found significant positive associations of PFOA and/or PFOS exposure with some types of chromosomal aneuploidy and some markers of DNA fragmentation, but not with other, related outcomes tested (not specifically described by the authors) (Governini et al. 2015).

Four studies evaluated fertility or fecundability with respect to PFAS exposure in men. A retrospective case-control study in Italy found a significant positive association between PFOS levels in blood, but not semen, and risk of infertility among men in metropolitan areas, but not urban or rural areas, and no significant association with PFOA levels (La Rocca et al. 2015). By contrast, an occupational cohort study of mostly male workers at the 3M Decatur plant found no association between PFOS exposure and episodes of care for fertility or infertility management (Olsen et al. 2004). A retrospective case-control study in Belgium found no significant association between PFOA or PFOS exposure and male infertility (Den Hond et al. 2015).

Likewise, a retrospective cohort study in Greenland, Ukraine, and Poland found no significant association between PFOA or PFOS (or PFHxS) exposure in male partners and fecundability ratio; PFNA exposure was associated with significantly lower fecundability ratio in Greenland couples, but not those in Ukraine or Poland (Jorgensen et al. 2014).

Other male reproductive outcomes were evaluated in few studies, and significant associations with exposure to PFOA, PFOS, or other PFAS were not consistently detected across or within multiple study populations. For example, a cross-sectional study found a significant association between PFOS exposure and a significantly increased ratio of Y to X chromosomes in sperm among men in Greenland, Ukraine, and Poland combined, but a significantly decreased ratio among men in Greenland only, and no significant association with PFOA exposure (Kvist et al. 2012). By contrast, a later study in the same population reported no such association (Lenters et al. 2015), and a prospective cohort study in Michigan and Texas found no significant association between paternal exposure to PFOS (or to PFOA, PFOSA, Et-PFOSA-AcOH, or PFDA) and ratio of male to female births, although this study found significant associations of paternal Me-PFOSA-AcOH and PFNA exposure with a lower male-to-female ratio (decreased ratio of Y to X chromosomes) that did not follow an exposure-response trend (Bae et al. 2015). No significant association between prostate-specific antigen levels and exposure to PFOA, PFOS, or other PFAS was detected in Mid-Ohio Valley men (Ducatman et al. 2015), men in Greenland, Ukraine, and Poland (Lenters et al. 2015), or male PFOA production workers at the Miteni plant in Trissino, Italy (Costa et al. 2009). PFOS exposure also was not significantly associated with benign prostatic hyperplasia or prostatitis among workers at the 3M plant in Decatur, Alabama (Grice et al. 2007). Reproductive tract disorders (cryptorchidism, hypospadias, inguinal hernia, varicocele, testicular hydrocele, incarcerated hernia, phimosis, testicular torsion, chlamydia, gonorrhea, and epididymitis) were evaluated in a cohort of Danish men (Vested et al. 2013), a cohort of Danish newborn boys (Toft et al. 2016), and a case-control study of Danish and Finnish newborn boys (Vesterholm Jensen et al. 2014); none of these studies found any significant associations of male reproductive tract disorders with PFOA or PFOS exposure.

In a 2016 literature review of epidemiologic studies of PFAS exposure in association with human fertility, the authors concluded that a causal relationship with male reproductive outcomes has not been demonstrated (Bach et al. 2016b) (emphasis added):

> In men, the evidence regarding an association between exposure to PFASs and semen characteristics as well as reproductive hormones is sparse despite the fact that a relatively large amount of studies have investigated the topic. Even though a few male studies suggested some associations, this was based on the examination of a large number of exposure-outcome combinations, and there was little consistency regarding results for specific exposures and outcomes across

> studies. With respect to male reproduction, high impact adverse exposures usually affect more than one aspect of the reproductive system … PFOS or other PFASs might be weakly associated with lower testosterone levels or impaired sperm morphology, but *the lack of other consistent results regarding a large panel of outcomes limits the interpretation of this as causal*. Neither in the male nor female studies did the studies with the highest average exposure levels demonstrate stronger findings.

In summary, based on the published epidemiologic literature, most male reproductive outcomes evaluated, including specific measures of semen quality and morphology, levels of reproductive hormones, and various less commonly studied endpoints, were not significantly associated with exposure to PFOA, PFOS, or other PFAS, and most of the few statistically significant associations detected were counterbalanced by statistically null findings in other studies. No clear pattern of association was detected across occupational, contaminated-community, and general-community settings. For the small number of significant associations observed, causal inference is precluded by the typically cross-sectional study designs, limited control for confounding by a broad range of potential environmental and behavioral risk factors (Skakkebaek et al. 2016), and measurement of PFAS exposure and, in the case of reproductive hormone levels and other endpoints that fluctuate over time, measurement of the outcome at only one point in time. Thus, the available published epidemiologic evidence does not establish a causal relationship between PFOA, PFOS, or other PFAS exposure and male reproductive outcomes in humans.

8.17 Epidemiology of PFAS and Respiratory Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on non-malignant respiratory outcomes in humans has not been established by the available published epidemiologic studies. I identified 10 published studies that reported the association between PFOA or PFOS exposure and respiratory conditions, including mortality from overall or specific non-malignant respiratory disease, chronic obstructive pulmonary disease (COPD), chronic bronchitis, shortness of breath on stairs, and unspecified "lung disease," all in adults (Gilliland and Mandel 1993, Alexander et al. 2003, Anderson-Mahoney et al. 2008, Leonard et al. 2008, Lundin et al. 2009, Melzer et al. 2010, Nolan et al. 2010, Steenland and Woskie 2012, Consonni et al. 2013, Steenland et al. 2015) (Table 17 in Appendix A). Three studies were cross-sectional, including one set in the general U.S. population and two set in the Mid-Ohio Valley community; the other seven were retrospective occupational cohort studies. PFAS other than PFOA and PFOS were not evaluated in these studies.

In the six retrospective occupational cohort studies that evaluated mortality from non-malignant respiratory disease, none detected a significant excess (and some detected significant deficits) of

this endpoint or any subcategories (influenza/pneumonia, bronchitis/emphysema/asthma, bronchitis, emphysema, COPD, tuberculosis, or other respiratory disease) among PFOA or PFOS workers at the DuPont Parkersburg, 3M Cottage Grove, 3M Decatur, and North American and European polytetrafluoroethylene plants (Gilliland and Mandel 1993, Alexander et al. 2003, Leonard et al. 2008, Lundin et al. 2009, Steenland and Woskie 2012, Consonni et al. 2013). The seventh retrospective occupational cohort study, which examined COPD incidence rather than mortality, also found no significant excess among Parkersburg plant workers (Steenland et al. 2015).

The only study that reported a significant positive association between PFOA exposure and non-malignant respiratory disease—namely, chronic bronchitis and shortness of breath on stairs— was the cross-sectional study of Mid-Ohio Valley residents in which results were probably distorted by selection bias, confounding, over-reporting of adverse health outcomes, the lack of a suitable unexposed comparison group, and ecologic exposure classification (Anderson-Mahoney et al. 2008). The other two cross-sectional studies found no significant association of Mid-Ohio Valley water-district-level PFOA exposure with maternal "lung disease" (Nolan et al. 2010), no significant association of PFOA exposure with COPD, and a significant inverse association between PFOS exposure and COPD among general U.S. adults (Melzer et al. 2010).

In summary, published epidemiologic studies of PFOA or PFOS exposure and non-malignant respiratory outcomes, especially mortality from non-malignant respiratory diseases, consistently indicated no significant positive association, despite being based mostly in high-exposure occupational settings. Only one study reported significant positive associations of non-malignant respiratory disease with PFOA exposure that were of dubious validity and were not replicated elsewhere. The few observed significant deficits of non-malignant respiratory disease mortality among PFOA workers cannot reliably be interpreted as supporting a causal protective effect, due to potential bias from the healthy worker effect and lack of adjustment for confounding by tobacco smoking and other risk factors. Therefore, given the mostly statistically null findings and the lack of convincing positive associations, the published epidemiologic evidence does not show a causal effect of PFOA or PFOS exposure on non-malignant respiratory disease in humans.

8.18 Epidemiology of PFAS and Thyroid Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on non-malignant thyroid outcomes in humans has not been established by the available published epidemiologic studies. I identified 42 published studies that reported the association between specific PFAS and thyroid endpoints, including thyroid-related hormone, protein, or antibody levels, and clinical or subclinical thyroid diseases, in various study populations and age groups (Olsen et al. 1998,

Olsen et al. 2003a, Inoue et al. 2004, Olsen et al. 2004, Emmett et al. 2006, Olsen and Zobel 2007, Anderson-Mahoney et al. 2008, Dallaire et al. 2009, Pirali et al. 2009, Bloom et al. 2010, Melzer et al. 2010, Chan et al. 2011, Espino-Hernandez et al. 2011, Kim et al. 2011a, Knox et al. 2011a, Ji et al. 2012, Raymer et al. 2012, Lopez-Espinosa et al. 2012a, Lopez-Espinosa et al. 2012b, Audet-Delage et al. 2013, Jain 2013, Wang et al. 2013, Wen et al. 2013, Lin et al. 2013b, de Cock et al. 2014, Wang et al. 2014, Webster et al. 2014, Winquist and Steenland 2014b, Berg et al. 2015, Lewis et al. 2015, Shrestha et al. 2015, Steenland et al. 2015, Kato et al. 2016, Shah-Kulkarni et al. 2016, Webster et al. 2016, Yang et al. 2016, Kim et al. 2016a, Christensen et al. 2016b, Berg et al. 2017, Crawford et al. 2017, Li et al. 2017, Tsai et al. 2017) (Table 18 in Appendix A). Ten of these studies evaluated thyroid outcomes in infants, one in children, three in adolescents, and 34 in adults or mixed age groups comprising mostly adults. Twenty-six studies were cross-sectional, four were retrospective case-control studies, three were retrospective cohort studies (including one with a cross-sectional component), and nine were prospective cohort studies (including four with a cross-sectional component and two with a retrospective component). Most studies (26) were conducted in general community settings; four were conducted in fishing communities, six in the Mid-Ohio Valley community (including one study that also included Parkersburg plant workers), and one in a PCB-contaminated community; and five were conducted at least in part in occupational settings.

Most of the available epidemiologic studies were cross-sectional in design and tested associations between PFAS exposure and various thyroid hormones, especially thyroid-stimulating hormone (TSH), free or total thyroxine (T4), and free or total triiodothyronine (T3), measured simultaneously in serum or plasma. As with studies of other health endpoints, cross-sectional studies of thyroid outcomes are susceptible to reverse causality and confounding by shared underlying physiological mechanisms that influence levels of both PFAS and thyroid-related hormones, proteins, or antibodies. Many other potential behavioral and environmental influences on thyroid hormone levels and thyroid dysfunction could also confound observed associations (Bajaj et al. 2016). Moreover, nearly all of the cross-sectional studies relied on a single measure of thyroid biomarkers, which vary within individuals over short and long time periods (Feldt-Rasmussen et al. 1980, Nagayama et al. 1993, Andersen et al. 2003). Many studies reported that the vast majority of subjects had thyroid hormone levels within the laboratory reference range, thereby calling into question whether any observed differences in thyroid hormone levels could be interpreted as clinically adverse. Finally, several studies of thyroid-related biomarkers did not report whether individuals taking thyroid medications were excluded from analysis.

The C8 Science Panel concluded in 2012 that PFOA exposure had a “probable link” to thyroid disease (C8 Science Panel 2012d). This conclusion was based largely on a Mid-Ohio Valley community study in which PFOA exposure was significantly positively associated with self-reported thyroid disease among children and adolescents aged 1–17 years in a cross-sectional

analysis, but not with self-reported hypothyroidism or subclinical hypo- or hyperthyroidism; in the same study, estimated *in utero* PFOA exposure and cross-sectionally measured PFOS and PFNA exposure also were not significantly associated with any of these outcomes (Lopez-Espinosa et al. 2012b). Self-report is known to be an inaccurate basis for ascertaining thyroid disease (Brix et al. 2001), calling into question the validity of observed associations with self-reported thyroid outcomes.

Current epidemiologic evidence continues to show an inconsistent association between exposure to PFOA or other PFAS and risk of non-malignant thyroid disease, including nine other available studies of PFOA, PFOS, or other PFAS exposure (PFBA, PFBS, PFPA, PFHxA, PFHxS, PFHpA, PFHpS, PFNA, PFDA, PFunDA, PFdoDA, PFtrDA, and combinations of these). Significant positive associations between PFOA exposure and thyroid disease were found in prospective and retrospective analyses of Mid-Ohio Valley community and occupational cohorts combined (where associated outcomes included validated functional thyroid disease, hyperthyroidism, and hypothyroidism) (Winquist and Steenland 2014b), as well as with self-reported thyroid disease in a cross-sectional study of Mid-Ohio Valley class action litigation participants compared with published general population statistics (Anderson-Mahoney et al. 2008); however, no significant association with self-reported thyroid disease was found in a cross-sectional analysis of PFOA exposure based on serum levels in Mid-Ohio Valley residents (Emmett et al. 2006). A cross-sectional study of Wisconsin fishermen found no significant association of exposure to PFOA (or PFOS, PFHxS, PFHpS, PFNA, PFDA, PFunDA, or the sum of these PFAS) with self-reported thyroid disease (Christensen et al. 2016b), and retrospective cohort studies also found no significant association between PFOA exposure and validated self-reported thyroid disease (Steenland et al. 2015) or between PFOS exposure and episodes of medical care for thyroid disease based on claims records (Olsen et al. 2004).

Three general-community studies also yielded inconsistent results. A significant positive association was observed between exposure to PFOA (and PFNA, PFDA, PFunDA, and the sum of PFAS, but not PFBA, PFBS, PFHxA, PFHxS, PFHpA, PFHpS, PFOS, PFdoDA, or PFtrDA) and risk of clinically diagnosed congenital hypothyroidism in a retrospective case-control study of infants in Seoul, South Korea (Kim et al. 2016a). In the general U.S. population, a significant cross-sectional association was found between PFOA exposure and self-reported ever or current thyroid disease among women, but not men, and between PFOS exposure and current thyroid disease among men (Melzer et al. 2010). By contrast, a retrospective case-control study in Pavia, Italy, found no significant association between exposure to PFOA or PFOS and clinically diagnosed toxic/non-toxic multinodular goiter or Hashimoto's thyroiditis or Graves' disease (Pirali et al. 2009). Thus, overall, no consistent association has been established between exposure to PFOA, PFOS, or other PFAS and risk of any or all combined types of non-malignant thyroid disease.

Thirty-three studies evaluated associations of PFOA, PFOS, or other PFAS exposure (PFBA, PFBS, PFPA, PFHxA, PFHxS, PFHpA, PFHpS, PFNA, PFDA, PFunDA, PFdoDA, PFtrDA, PFteDA, and combinations of these) with thyroid-related hormone, protein, or antibody levels, with a multitude of associations that overall were mostly statistically null, with inconsistent significant findings. Although most studies detected at least one statistically significant association (but some detected none (Inoue et al. 2004, Emmett et al. 2006, Bloom et al. 2010, Chan et al. 2011, Espino-Hernandez et al. 2011, Lopez-Espinosa et al. 2012a, Audet-Delage et al. 2013)), most tested numerous hypotheses related to several PFAS and several thyroid outcomes, such that some significant associations would be expected by chance. For example, associations between PFOA exposure and TSH levels were a variety of significant positive (Kim et al. 2011a, Jain 2013, Webster et al. 2014, Webster et al. 2016), significant inverse (Olsen et al. 1998, Lopez-Espinosa et al. 2012b, Lewis et al. 2015), and mostly statistically null associations (Olsen et al. 1998, Emmett et al. 2006, Olsen and Zobel 2007, Bloom et al. 2010, Kim et al. 2011a, Knox et al. 2011a, Ji et al. 2012, Raymer et al. 2012, Lopez-Espinosa et al. 2012b, Wang et al. 2013, Wen et al. 2013, Lin et al. 2013b, Wang et al. 2014, Webster et al. 2014, Berg et al. 2015, Lewis et al. 2015, Shrestha et al. 2015, Kato et al. 2016, Shah-Kulkarni et al. 2016, Webster et al. 2016, Yang et al. 2016, Kim et al. 2016a, Berg et al. 2017, Crawford et al. 2017, Li et al. 2017, Tsai et al. 2017); several studies had different results by subgroup. Associations between PFOA exposure and total T4 level consisted of a few significant positive (Knox et al. 2011a, Lopez-Espinosa et al. 2012b, de Cock et al. 2014) and mostly statistically null findings (Olsen and Zobel 2007, Kim et al. 2011a, Knox et al. 2011a, Ji et al. 2012, Raymer et al. 2012, Lopez-Espinosa et al. 2012b, Jain 2013, Wen et al. 2013, de Cock et al. 2014, Wang et al. 2014, Webster et al. 2014, Berg et al. 2015, Lewis et al. 2015, Shrestha et al. 2015, Shah-Kulkarni et al. 2016, Webster et al. 2016, Yang et al. 2016, Berg et al. 2017, Crawford et al. 2017, Tsai et al. 2017). Similarly, PFOA exposure was significantly positively associated with total T3 level in some studies (Olsen and Zobel 2007, Jain 2013, Wen et al. 2013, Crawford et al. 2017), but not significantly associated in most (Olsen and Zobel 2007, Kim et al. 2011a, Raymer et al. 2012, Wen et al. 2013, Wang et al. 2014, Berg et al. 2015, Lewis et al. 2015, Shrestha et al. 2015, Shah-Kulkarni et al. 2016, Webster et al. 2016, Yang et al. 2016, Kim et al. 2016a, Berg et al. 2017, Tsai et al. 2017). Anti-thyroid peroxidase antibody levels interacted positively with PFOA, PFOS, and PFNA exposure in relation to higher TSH levels in a Canadian cross-sectional study (Webster et al. 2014), but antibody levels were not significantly positively associated with PFOA exposure in another cross-sectional study in southern China (Li et al. 2017). Results were similarly inconsistent for other thyroid-related hormones, proteins, and antibodies, and in association with exposure to PFOS and other PFAS.

A recent systematic literature review of epidemiologic studies of PFOA, PFOS, PFHxS, or PFNA exposure in relation to thyroid hormone levels or thyroid dysfunction in pregnant women and/or children concluded that data are insufficient to conclude that a causal relation has been

demonstrated. As stated by the authors, who included one member of the C8 Science Panel (Ballesteros et al. 2017) (emphasis added):

> In conclusion, heterogeneity was found across studies in terms of study design, study setting, timing of PFAS exposure assessment, timing and type of thyroid-related outcome assessment, adjustment for potential confounders, and statistical approach. As a consequence, there were insufficient numbers of comparable studies in each population group except for two cases: mothers and 11–19-year-old children. Based on the current literature, we found some consistency of a positive association between PFHxS and PFOS in relation to TSH levels measured in maternal blood and PFNA and TSH levels measured in the blood of boys aged ≥11 years. However, *further studies are warranted to confirm these possible relationships*.

Another literature review conducted by several U.S. EPA authors reached a similar conclusion about the lack of an established causal association between PFAS exposure and thyroid outcomes (Rappazzo et al. 2017):

> While some associations are observed between thyroid hormones and PFAS, no clear patterns emerge. There is some evidence for hypothyroidism, a finding that has also been observed in an adult [National Health and Nutrition Examination Survey] population …, but not in other studies of PFAS and thyroid function. Given the limited number of studies and the variability in the responses, no conclusions can be reached with certainty.

In summary, based on the available published literature, the epidemiologic evidence on exposure to PFOA, PFOS, and other PFAS and non-malignant thyroid outcomes, including thyroid disease and thyroid biomarker levels, was inconsistent. Associations did not vary systematically according to anticipated exposure levels across occupational cohorts, contaminated communities, and general communities. The preponderance of evidence consisted of statistically non-significant associations, and the observed statistically significant findings were not consistent for specific thyroid endpoints. In light of the inconsistency of findings, as well as the methodological limitations of the available studies, including the cross-sectional nature of most studies, the generally one-time measurement of exposure and outcome, the incomplete exclusion of individuals taking thyroid medications, and the remaining potential for results to be distorted by confounding or bias, the weight of epidemiologic evidence does not demonstrate a causal relationship between PFOA, PFOS, or other PFAS exposure and thyroid disease or other thyroid outcomes.

8.19 Epidemiology of PFAS and Other Health Outcomes

A causal effect of exposure to PFOA, PFOS, or other PFAS on various other health outcomes in humans, besides those already discussed above, has not been established by the available published epidemiologic studies. I identified 23 published studies that reported the association between specific PFAS and other health outcomes that are not encompassed by the categories previously discussed in this report (Gilliland and Mandel 1993, Olsen et al. 2000, Olsen et al. 2004, Grice et al. 2007, Leonard et al. 2008, Lundin et al. 2009, Wang et al. 2012, Caserta et al. 2013, Consonni et al. 2013, Fletcher et al. 2013, Vagi et al. 2014, Watkins et al. 2014, La Rocca et al. 2015, Pennings et al. 2016, Toft et al. 2016, Kim et al. 2016b, Franken et al. 2017, Kingsley et al. 2017, Kobayashi et al. 2017, Vriens et al. 2017, Goudarzi et al. 2017a, Lind et al. 2017a, Wang et al. 2017b) (Table 19 in Appendix A).

Four studies measured gene expression (Caserta et al. 2013, Fletcher et al. 2013, La Rocca et al. 2015, Pennings et al. 2016), including two related retrospective case-control studies (one in Italian men, one in Italian women) that evaluated expression of the same nuclear receptor genes (Caserta et al. 2013, La Rocca et al. 2015). PFOA exposure was significantly associated with lower expression of the aryl hydrocarbon receptor gene among infertile women, but not among all women combined; and PFOA exposure also was significantly associated with lower expression of the pregnane X receptor gene among fertile women, but not among all women combined (Caserta et al. 2013). Significant inverse associations with expression of these two genes also were detected among men in the same study population (La Rocca et al. 2015). However, positive associations between PFOS exposure and greater expression of the androgen receptor and pregnane X receptor genes among infertile women were not consistent with statistically null results for these associations among men; and significant inverse associations of PFOA exposure with expression of other nuclear receptors among men (estrogen receptor-alpha, estrogen receptor-beta, and androgen receptor) were not replicated among women. The other studies evaluated non-overlapping sets of genes, such that consistency of results across studies could not be assessed.

Three studies evaluated gene methylation, again of non-overlapping sets of genes, precluding an assessment of consistency (Watkins et al. 2014, Kingsley et al. 2017, Kobayashi et al. 2017).

Several studies measured levels of various biomarkers, including markers of DNA damage (Franken et al. 2017), glucocorticoid hormones and their ratios with and androgenic hormones in newborns (Toft et al. 2016, Goudarzi et al. 2017a) (largely non-overlapping, except that these two studies found opposite results for the association between PFOS exposure and cortisol level), malondialdehyde (Kim et al. 2016b), cholecystokinin (Olsen et al. 2000), placental mitochondrial DNA content (Vriens et al. 2017), circulating microRNAs (Wang et al. 2012), metabolome biomarkers (Wang et al. 2017b), and 8-hydroxy-2'-deoxyguanosine (a biomarker of

oxidative stress evaluated in two studies, but in association with different PFAS) (Kim et al. 2016b, Franken et al. 2017), that were not comparable between studies.

A few studies ascertained certain disease endpoints, such as polycystic ovary syndrome (Vagi et al. 2014), acute pancreatitis, and urinary tract infections (Olsen et al. 2004), that were not evaluated elsewhere. One study evaluated associations between maternal PFAS exposure and anogenital distance and penile width as potential indicators of endocrine disruption in 3-month-old infants (Lind et al. 2017a).

No significant excess of mortality from benign neoplasms (Leonard et al. 2008, Lundin et al. 2009, Consonni et al. 2013), episodes of care for benign colonic polyps, benign neoplasms of skin, or other benign/unspecified neoplasms (Olsen et al. 2004), or colon polyps (Grice et al. 2007) was observed in five retrospective occupational cohort studies of PFOA or PFOS workers.

One study found that male PFOA workers at the 3M Cottage Grove plant had a significantly lower risk of mortality from gastrointestinal disease than expected (Gilliland and Mandel 1993), but two other studies found no significant difference in risk of digestive disease mortality (Consonni et al. 2013) or stomach ulcer mortality (Lundin et al. 2009) in PFOA worker cohorts.

Results of two studies of PFOS workers at the 3M Decatur plant yielded inconsistent results for benign colonic polyps and cystitis, with significant positive findings in one study (Olsen et al. 2004) but not the other (Grice et al. 2007), and statistically null results for cystitis, bladder/urinary tract calculi, benign prostatic hyperplasia, and prostatitis in both studies.

Overall, results for these other health outcomes were too sparse to evaluate consistency across studies, and they provided an insufficient scientific basis on which to establish a causal relationship. Thus, the published epidemiologic evidence on health outcomes other than those evaluated earlier in this report has not established a causal relationship between exposure to PFOA, PFOS, or other PFAS on other human health endpoints.

8.20 Summary of Epidemiologic Evidence on PFAS

In summary, this systematic review of the available published epidemiologic evidence on associations of exposure to PFOA, PFOS, and other PFAS with a broad range of human health outcomes—including all of the epidemiologic studies that I identified based on documented search terms used in the publicly available PubMed biomedical literature database—shows that significant associations between exposure to specific PFAS and specific health endpoints are not consistently detected across study populations, and the majority of associations with a given

health outcome are not statistically significant. Additionally, methodological limitations of these studies prevent a reliable conclusion that the statistically significant associations detected in a minority studies are due to causal effects of PFAS exposure.

To my knowledge, no major health or regulatory agency has concluded that a causal effect of PFOA, PFOS, or other PFAS on adverse human health outcomes has been established. On the contrary, based on systematic reviews of the available scientific literature, at least four major health or regulatory agencies have reached conclusions that do not clearly indicate causal human health effects of PFAS (ATSDR 2015, IARC 2016, NTP 2016b, U.S. EPA 2016d, b, c, a). These agencies review and weigh the balance of scientific evidence for the purpose of identifying and ultimately preventing the causes of human disease. Their reports are used by local, national, and international authorities for such purposes as risk assessment, development of disease prevention and control programs, and regulatory decision-making on public health. Thus, their characterizations of human health hazards are generally viewed as precautionary, with the overarching goal of protecting human health.

IARC (2016) concluded that PFOA is "possibly carcinogenic to humans (Group 2B)," based on "limited evidence" in humans, as well as "limited evidence" in experimental animals and "moderate evidence" for mechanisms of PFOA-associated carcinogenesis. IARC did not classify PFOA as "probably carcinogenic to humans (Group 2A)" or "carcinogenic to humans (Group 1)," which are stronger classifications of potential carcinogenicity.

NTP (2016) classified PFOA and PFOS as "presumed to be an immune hazard to humans," based in part on a "moderate" level of evidence from human studies; NTP did not classify PFOA or PFOS as "known to be an immune hazard to humans," which is a stronger classification of potential immunotoxicity.

ATSDR (2015) released a draft toxicological profile for PFAS in which it concluded (emphasis added):

> Although a large number of epidemiology studies have examined the potential of perfluoroalkyl compounds to induce adverse health effects, most of the studies were cross-sectional in design and *did not establish causality*. ATSDR used a weight-of-evidence approach to evaluate whether the available data supported a link between perfluoroalkyl exposure and a particular health effect. This weight-of-evidence approach takes into consideration the consistency of the findings across studies, the quality of the studies, dose-response, and plausibility. It should be noted that although the data may provide strong evidence for an association, it does not imply that the observed is biologically relevant because the magnitude of the change is within the normal limits or not indicative of an adverse health

> outcome. Plausibility depends primarily on experimental toxicology studies that establish a plausible biological mechanism for the observed effects.
>
> Epidemiology studies have found statistically significant associations between serum perfluoroalkyl levels (particularly PFOA and PFOS) and a wide range of health effects. When the subjects were categorized by serum perfluoroalkyl levels, dose-response relationships were found for most of the effects. However, findings were not always consistent across studies. However, consistent findings were found for association of serum PFOA and PFOS with increases in serum lipid levels, decreases in birth weight, increases in uric acid levels, and alterations in biomarkers of liver damage. There was also equivocal evidence of carcinogenicity. Although other effects have been reported, they have not been consistently found in similar types of studies, have only been examined in a single study, or were only found in general population studies.

In its Drinking Water Health Advisories for PFOA and PFOS, U.S. EPA (2016d, b) also acknowledged the uncertainties and limitations in the epidemiologic database for these chemicals, and referred repeatedly to "associations" but not necessarily causal links with various health outcomes:

> The human epidemiology studies provide evidence of an association between PFOA exposure and health effects in humans, and is another line of evidence supporting this assessment. The human data demonstrate an association between PFOA exposure and endpoints, including effects on serum lipids, antibody responses, fetal growth and development, and the liver. They provide support for identification of hazards of PFOA exposure. The associations observed for serum lipids, and reproductive parameters and immunotoxicity are the strongest. For many endpoints, however, the results are inconsistent. Although the human studies collectively support the conclusion that PFOA exposure is a hazard, EPA concluded that, based on several uncertainties associated with the database, the human studies are adequate for use qualitatively in the identification hazard at this time (U.S. EPA 2016d).
>
> The human epidemiology studies provide evidence of an association between PFOS exposure and health effects in humans, and is another line of evidence supporting this assessment. The human data demonstrate an association between PFOS exposure and endpoints including effects on serum lipids, antibody responses, the thyroid, and fetal growth and development. The data provide support for identification of hazards of PFOS exposure. The associations observed for serum lipids and reproductive outcomes are the strongest. For many endpoints,

> the results are inconsistent, however. Although the human studies collectively support the conclusion that PFOS exposure is a hazard, EPA concluded that based on several uncertainties associated with the database, the human studies are adequate for use qualitatively in the identification hazard at this time (U.S. EPA 2016b).

With respect to potential effects of PFOA or PFOS exposure on diminished immune response to vaccination, U.S. EPA (2016d, b) explicitly stated that a conclusion of causality could not be reached based on the available epidemiologic studies (emphasis added): "Overall, although *these results are not sufficient to establish a causal effect* of [PFOA or PFOS] exposure on an impaired serological vaccine response, some of the positive associations are striking in magnitude and require replication in independent studies."

Uncertainties identified by U.S. EPA (2016d, b) in the epidemiologic database for PFOA and PFOS, making it unsuitable for quantitative risk assessment (i.e., adequate only for qualitative use), included the following:

- Lack of precise, individual-level information on etiologically relevant PFOA/PFOS exposure ("Although mean serum values are presented in the human studies, actual estimates of exposure (i.e., doses/duration) are not available. Thus, the serum level at which the effects were first manifest and whether the serum had achieved steady state or was in decline at the point the effect was evaluated cannot be determined.")
- Lack of specificity of serum PFOA levels as an indicator of exposure to PFOA itself, rather than exposure to telomer alcohol derivatives of PFOA that originate from sources other than drinking water ("Some of the human exposure that results in serum PFOA can come from telomer alcohol PFOA derivatives that break down metabolically to PFOA … The derivatives do not originate from PFOA in drinking water; they usually originate from diet and materials used in the home.")
- Inability to distinguish among associations with exposure to various PFAS and other persistent organic pollutants ("[M]ost of the subjects of the epidemiology studies had other perfluorinated carboxylates and sulfonates and/or other biopersistent contaminants in their blood. Although the study designs adjust for other potential toxicants as confounding factors, their presence constitutes a level of uncertainty that is usually absent in the animal studies.")
- Absence of control for inter-individual physiological variation in clearance rates due to factors that can affect serum levels and risk of health outcomes ("Interspecies and gender variation in PFOS clearance half-life can vary by several orders of magnitude … The potential for confounding influences is decreased under the controlled conditions of the animal studies.")

These uncertainties and other methodological problems, as well as the inconsistent results from published studies, prevent the available epidemiologic evidence from establishing conclusive causal relationships between exposure to PFOA, PFOS, or other PFAS and specific human health outcomes.

9.0 Discussion of Points in Dr. Grandjean's Report

9.1 Selective Citation of the Literature

Dr. Grandjean does not describe how he identified and selected the epidemiologic studies that he cites to support his opinions; however, a comparison with the studies cited in Tables 1–19 of this report reveals that he omits numerous relevant studies, many of which show results that do not support his opinions. For example, in his report section on immune outcomes, Dr. Grandjean cites 18 published epidemiologic studies (Fei et al. 2010a, Wang et al. 2011, Grandjean et al. 2012, Okada et al. 2012, Granum et al. 2013, Steenland et al. 2013, Looker et al. 2014, Steenland et al. 2015, Mogensen et al. 2015a, Dalsager et al. 2016, Kielsen et al. 2016, Pennings et al. 2016, Stein et al. 2016b, Grandjean et al. 2017, Oulhote et al. 2017, Qin et al. 2017, Timmermann et al. 2017a, Goudarzi et al. 2017b), but he omits 23 other studies that I identified as reporting on the relationship between PFAS exposure and immune endpoints (Emmett et al. 2006, Anderson-Mahoney et al. 2008, Leonard et al. 2008, Costa et al. 2009, Lundin et al. 2009, Melzer et al. 2010, Lin et al. 2011, White et al. 2011, Shankar et al. 2011a, Dong et al. 2013, Kishi et al. 2013, Jiang et al. 2014, Okada et al. 2014, Osuna et al. 2014, Ashley-Martin et al. 2015, Genser et al. 2015, Smit et al. 2015, Buser and Scinicariello 2016, Conway et al. 2016, Zhu et al. 2016, Goudarzi et al. 2016a, Stein et al. 2016a, Zhou et al. 2017b).[15]

In his report section on thyroid outcomes, Dr. Grandjean cites six published epidemiologic studies (Olsen et al. 2003a, Melzer et al. 2010, Knox et al. 2011a, Lopez-Espinosa et al. 2012b, Wen et al. 2013, Winquist and Steenland 2014b), thereby omitting 36 other relevant studies that I identified (Olsen et al. 1998, Inoue et al. 2004, Olsen et al. 2004, Emmett et al. 2006, Olsen and Zobel 2007, Anderson-Mahoney et al. 2008, Dallaire et al. 2009, Pirali et al. 2009, Bloom et al. 2010, Chan et al. 2011, Espino-Hernandez et al. 2011, Kim et al. 2011a, Ji et al. 2012, Raymer et al. 2012, Lopez-Espinosa et al. 2012a, Audet-Delage et al. 2013, Jain 2013, Wang et al. 2013, Lin et al. 2013b, de Cock et al. 2014, Wang et al. 2014, Webster et al. 2014, Berg et al. 2015, Lewis et al. 2015, Shrestha et al. 2015, Steenland et al. 2015, Kato et al. 2016, Shah-

[15] In some instances, the papers cited by Dr. Grandjean are ambiguous or incorrect because the reference numbers provided in his report do not correspond with the appropriate paper. For example, on p. 41, paragraph b, he refers to a Norwegian study (almost certainly Granum et al. 2013), but he cites reference 35, which is Grandjean et al. 2012. On p. 44, paragraph o, he refers to a study of allergies in children, but he cites reference 132, which is a study of mice. On p. 65, paragraph c, he cites a study of PFOA and fasting serum insulin, but he cites reference 235, which is study of mouse tissue; and he also cites a C9 Science Panel study of diabetes, but he cites reference 219, which is a study of thyroid disease. On p. 71, paragraph c, he cites two cross-sectional studies of liver function biomarkers, but he cites references 191 and 248, which are a C8 Science Panel report on birth defects and a review article on the developmental origins of non-communicable disease, respectively. In counting the number of cited papers, I have included only those that appear to be properly identified.

Kulkarni et al. 2016, Webster et al. 2016, Yang et al. 2016, Kim et al. 2016a, Christensen et al. 2016b, Berg et al. 2017, Crawford et al. 2017, Li et al. 2017, Tsai et al. 2017).

Dr. Grandjean cites 11 published epidemiologic studies of diabetes, insulin resistance, or glucose homeostasis (including some studies of BMI) to support his opinion on insulin and diabetes (Leonard et al. 2008, Lin et al. 2009, Lundin et al. 2009, MacNeil et al. 2009, Halldorsson et al. 2012, Andersen et al. 2013, Timmermann et al. 2014, Høyer et al. 2015b, Zong et al. 2016, Karlsen et al. 2017, Mora et al. 2017), but he ignores 34 other epidemiologic studies on the topic of metabolic outcomes (Gilliland and Mandel 1993, Olsen et al. 2004, Anderson-Mahoney et al. 2008, Costa et al. 2009, Melzer et al. 2010, Nelson et al. 2010, Nolan et al. 2010, Lin et al. 2011, Shankar et al. 2011a, 2012, Steenland and Woskie 2012, Consonni et al. 2013, Fisher et al. 2013, Lin et al. 2013a, Jiang et al. 2014, Karnes et al. 2014, Lind et al. 2014, Raleigh et al. 2014, Kataria et al. 2015, Steenland et al. 2015, Zhang et al. 2015, Conway et al. 2016, Domazet et al. 2016, Shapiro et al. 2016, Su et al. 2016, Christensen et al. 2016a, Christensen et al. 2016b, Kim et al. 2016b, Ashley-Martin et al. 2017, Fleisch et al. 2017, Minatoya et al. 2017, Starling et al. 2017, Valvi et al. 2017, Lind et al. 2017b).

With respect to neurodevelopmental outcomes, Dr. Grandjean cites 9 published epidemiologic studies to support his opinion (Fei et al. 2008a, Hoffman et al. 2010, Fei and Olsen 2011, Gump et al. 2011, Stein and Savitz 2011, Liew et al. 2014, Stein et al. 2014a, Oulhote et al. 2016, Vuong et al. 2016), thereby omitting 14 other relevant epidemiologic studies on neurodevelopmental endpoints (Chen et al. 2013, Stein et al. 2013, Braun et al. 2014, Ode et al. 2014, Strom et al. 2014, Donauer et al. 2015, Forns et al. 2015, Liew et al. 2015, Wang et al. 2015, Høyer et al. 2015a, Lien et al. 2016, Quaak et al. 2016, Goudarzi et al. 2016b, Jeddy et al. 2017).

Taken together, in the sections of his report that summarize the epidemiologic evidence on PFAS exposure with respect to specific human health outcomes (i.e., the subsections on "Epidemiological evidence" within section VII, "Adverse Health Effects at Individual Endpoints"), Dr. Grandjean cites a total of 105 published epidemiologic studies. In comparison, in the corresponding sections of my report (i.e., the subsections within section 8.0, "Epidemiology of PFAS and Specific Human Health Outcomes"), I have cited a total of 311 published epidemiologic studies, including all of those cited by Dr. Grandjean.[16]

[16] All of the original epidemiologic studies of human health outcomes included among the 89 documents in STATE_07513465–07516558 also are included in my literature review. Several of those documents are reviews or reports rather than original studies, and several do not pertain directly to humans (e.g., STATE_07514199, STATE_07514267, STATE_07514862, STATE_07514872, STATE_07514970, STATE_07515924, STATE_07515937, STATE_07516022, STATE_07516029, STATE_07516079, STATE_07516140, STATE_07516156, STATE_07516456, STATE_07516490, STATE_07516543).

Within studies, Dr. Grandjean also selectively cites statistically significant positive findings, but ignores other associations tested but found not to be significant, and even some significant inverse (protective) associations. For example, when he describes the results of a study of PFOA, PFOS, PFHxS, and PFNA exposure in relation to thyroid outcomes in U.S. adults (Wen et al. 2013), Dr. Grandjean states that "higher serum concentrations of PFOA were associated with increased serum concentrations of T3, while PFHxS was linked to increases in both T3 and T4, but to lower T4 in men." However, he omits the consistently statistically null associations between exposure to PFOS (as well as PFNA) and total and free T4, total and free T3, TSH, thyroglobulin, subclinical hypothyroidism, and subclinical hyperthyroidism.

When describing associations between maternal PFAS levels and the antibody response to immunization among Norwegian children, Dr. Grandjean describes statistically significant inverse associations of PFHxS, PFOA, PFOS, and PFNA exposure with anti-rubella antibody levels, but he neglects to acknowledge that no significant associations were observed with antibody levels against the measles, influenza type B, and tetanus vaccines (Granum et al. 2013). Similarly, he points out inverse associations of serum PFOA with anti-influenza A/H3N2 antibody levels in a study of Mid-Ohio Valley adults (Looker et al. 2014), but omits statistically null associations of PFOA exposure with anti-influenza type B and A/H1N1 antibody levels, PFOS exposure with all three anti-influenza antibody levels, as well as both PFOA and PFOS exposure with cold and "flu" infections in the past year.

Dr. Grandjean also does not mention the statistically null results for PFOA in association with total and free T4, free T3, TSH, and thyroglobulin in both men and women, and the significant inverse association between PFOA exposure and subclinical hyperthyroidism in men. In describing a study of PFOA exposure and type 2 diabetes in Mid-Ohio Valley adults (MacNeil et al. 2009), he states that "PFOA exposure in 54,000 adults was not found to be associated with development of [type 2 diabetes], and neither was the fasting serum glucose concentration associated with the exposure." However, MacNeil et al. (2009) found statistically significant *inverse* associations between serum PFOA concentrations and risk of both self-reported and validated type 2 diabetes—findings that are inconsistent with Dr. Grandjean's opinion that exposure to PFAS increases the risk of diabetes.

When summarizing a review article on PFAS exposure in relation to reproductive outcomes (Bach et al. 2016b), Dr. Grandjean paraphrases the authors' statements in the abstract that "[s]ixteen studies investigated the association between PFAS exposure in men and semen parameters, reproductive hormone levels, or TTP [time to pregnancy]. There was a lack of consistent results among the numerous investigated exposure-outcome combinations. However, subtle associations between higher PFOS and lower testosterone or abnormal semen morphology cannot be excluded. Eleven studies assessed the association between PFAS exposure in women and TTP or reproductive hormones levels. Four of eight studies found

prolonged TTP with higher PFOS or PFOA, but only one study found an association when restricting to nulliparous women." However, he omits the authors' ensuing two statements: "In men, there is little evidence of an association between PFAS exposure and semen quality or levels of reproductive hormones. For PFOS and PFOA, the literature indicates an association with female fecundability in parous women, *which is most likely not causal*" (emphasis added).

Overall, Dr. Grandjean's citation of the published epidemiologic literature on PFAS exposure in relation to all of the human health outcomes that he discusses, and his description of specific results within the papers that he cites, is highly incomplete and slanted to support his opinions. Selective citation of the literature to support an opinion is not scientifically valid because it does not take the full weight of the evidence into consideration. A well-informed, scientifically justified opinion on a potentially causal association should not be based on a selective subset of the literature; rather, it should be based on all of the relevant epidemiologic studies identified in a rigorous, transparent, and unbiased manner (NRC 2011, Rhomberg et al. 2013). Thus, Dr. Grandjean's incomplete and one-sided review of the epidemiologic literature on PFAS does not provide a valid basis on which to draw conclusions about potential causal effects of PFAS exposure on specific human health outcomes.

9.2 Scientific Value of Occupational Studies

Dr. Grandjean criticizes the existing occupational cohort studies of PFOA and PFOS workers in part because such studies are subject to the healthy worker effect, that is, the tendency of overall mortality rates to be lower among employed workers than in the general population, due to the exclusion of severely ill and chronically disabled from employment (Porta et al. 2014). As Dr. Grandjean acknowledges, however, the healthy worker effect "does not affect all health outcomes uniformly." In particular, the influence of this phenomenon tends to be stronger for younger age groups; for active employees rather than retired or otherwise departed workers; and for the period shortly after the start of employment, with a reduction of impact as the duration of follow-up increases (Carpenter 1987, Choi 1992). Moreover, bias due to the healthy worker effect is anticipated to be greater for diseases that have readily apparent symptoms that appear shortly before diagnosis or death and/or that affect younger age groups, such as respiratory and cardiovascular diseases, than for diseases that are unlikely to be manifest at the time of employment and/or occur later in life, such as cancer (Carpenter 1987, Choi 1992).

In many instances, the published occupational studies used multiple comparison groups, including both general populations and worker populations with lower exposure levels. Internal comparisons with other, unexposed or less-exposed workers would not have been biased by the healthy worker effect.

In general, occupational epidemiologic studies are of unique scientific value because they enable the evaluation of potential health effects of high-level, often long-term exposures that typically are not encountered in the general population or even in populations with environmental contamination (see Figures 1 and 2). Thus, the various occupational studies of cancer incidence, mortality, and other health outcomes in the 3M, DuPont, Miteni, and combined polytetrafluoroethylene plants remain, as a whole, important sources of information on the potential health effects of chronic, high-level exposure to PFOA or PFOS.

9.3 Bias Toward the Null

Dr. Grandjean states in his report that in epidemiology, "there is a well-known and often misleading bias toward the null" due to causes such as inadequate statistical power in small studies, exposure misclassification, insensitive or imprecise outcome measures, failure to adjust for confounders with effects in the opposite direction, and pressure to avoid false alarm, among other reasons. However, this viewpoint ignores considerable evidence that published results in epidemiology, as well as other scientific fields, are skewed toward over-reporting of positive associations, and even toward exaggeration of the magnitude of such associations, due in part to the greater perceived newsworthiness or potential impact of positive than null results (Ioannidis 2005, Kavvoura et al. 2007, Ioannidis 2008, Turner et al. 2008, Fanelli 2010, Ioannidis et al. 2011, Fanelli 2012, Franco et al. 2014, Kivimaki et al. 2014). Simulation studies have shown that most published scientific research findings are anticipated to be false (Ioannidis 2005); that the proportion of false-positive findings outweighs the proportion of false-negative findings in epidemiology (Ioannidis et al. 2011); and that the magnitude of true associations tends to be overestimated in published studies (Ioannidis 2008). An analysis of published epidemiologic studies found that statistically significant results were substantially more likely than null results to be reported in the main text and highlighted in the abstract, and that results were selectively presented to emphasize the largest possible RRs based on extreme contrasts between groups (Kavvoura et al. 2007). Likewise, an analysis of funded survey-based research studies revealed that 95.6% of strong positive statistical results were written into manuscripts and 61.5% were published, whereas only 35.4% of null statistical results were written up and only 20.8% were published (Franco et al. 2014). Thus, the pressure to publish positive findings in order to secure limited research funding and jobs probably far outweighs any pressure to avoid false alarm (Fanelli 2010, 2012).

Although small studies may have insufficient statistical power to detect a true association, such studies are also more likely than larger studies to yield false-positive, inflated results. That is, statistically significant results from small studies are more likely to be false (due to chance) than significant results from larger studies (Button et al. 2013); and the magnitude of RR estimates, regardless of statistical significance, is more likely to be overestimated in smaller studies

(Greenland et al. 2000). These precautions should be borne in mind when interpreting the results of individual studies, as well as the overall body of epidemiologic literature.

Dr. Grandjean indicates that failure to adjust for confounders with effects in the opposite direction from the exposure can lead to bias toward the null. This is correct if a confounder is independently positively associated with the exposure, but not if the confounder is inversely associated with the exposure, in which case the observed association will be biased away from the null. Moreover, Dr. Grandjean neglects to mention that failure to adjust for confounders with effects in the *same* direction as the exposure can lead to *overestimation* of an association (if the confounders are positively associated with the exposure). There is no reason why confounders with opposing effects would be more likely to be neglected than those with parallel effects. The ability to adjust for confounding depends on the availability of sufficiently detailed and complete information on potential confounders, as well as the size of the study population; it is unrelated to the direction of the association between the confounder and the outcome. Therefore, Dr. Grandjean's selective emphasis on only one potential effect of confounding is scientifically imbalanced.

Another common misconception is that misclassification of the exposure and/or the outcome of interest predictably leads to underestimation of associations, that is, bias toward the null, as suggested by Dr. Grandjean. On the contrary, misclassification must be non-differential (i.e., random)—that is, exposure misclassification must be completely independent of outcome status, and outcome misclassification must be completely independent of exposure status—and additional stringent conditions, such as independence of other classification errors, must hold for even perfectly non-differential misclassification to produce bias toward the null (Thomas 1995, Weinberg et al. 1995, Jurek et al. 2005, Jurek et al. 2008, Ogburn and VanderWeele 2012). Under virtually all realistic scenarios—for example, if misclassification is even approximately non-differential, if the misclassification error depends on errors in other variables in the analysis, if the misclassification error interacts with other sources of error, such as selection bias and confounding, or if the misclassified variable has more than two levels, in which case additional conditions must be met—then the direction of bias is not necessarily toward the null. Even under the unlikely circumstance that all conditions for random misclassification are fulfilled, the tendency of bias toward the null applies only to the expected value of the observed association on average over many repetitions; thus, any given estimate of the association may still by chance be an overestimate.

9.4 Value of Null Results

Dr. Grandjean states in his report: "Studies that do not show a statistical significance are sometimes called 'negative,' although this is misleading. A better word in [*sic*] non-informative." Here he suggests that only studies that show statistically significant positive

results are informative, and that null results do not contribute valuable scientific information. In making this statement, Dr. Grandjean evinces the same bias toward reporting and publication of positive findings and downplaying of null findings that is commonly seen in the epidemiologic and broader scientific literature, and that leads to a distortion of published science.

Contrary to Dr. Grandjean's suggestion, null findings are essential to the scientific method of developing hypotheses and repeatedly testing and refining them through experimentation and observation, making modifications when results do not support the original hypotheses. Without publication of null results, such as studies that do not replicate prior findings (which may be false) or empirical observations that do not support existing theories (which may be inaccurate), science cannot correct itself; instead, the body of scientific literature becomes skewed and non-representative. Additionally, reporting of null results can help scientists to redirect their time and resources toward other research questions.

The scientific value of null research findings is underscored by the growing number of journals that proactively publish null results (Granqvist 2015, van Hilten 2015), the creation of databases to register studies at their inception and provide a venue for transparent reporting of all results (Schooler 2011, Wieseler et al. 2012), and calls for publication of null results from agencies such as the World Health Organization (WHO 2015) and the World Medical Association (WMA 2013), which specifies in the current Declaration of Helsinki—a widely adopted set of ethical principles for medical research involving human subjects—that "[r]esearchers have a duty to make publicly available the results of their research … Negative and inconclusive as well as positive results must be published or otherwise made publicly available."

An illustrative example is that of saccharin, an artificial sweetener discovered in 1879 that was found in 1977 to cause urinary bladder cancer in rats, especially males, leading NTP in 1981 to classify saccharin as "reasonably anticipated to be a human carcinogen" and IARC in 1987 to classify it as "possibly carcinogenic to humans (Group 2B)" (IARC 1987, NTP 2016a). Subsequent experimental animal studies produced null results showing that saccharin was not carcinogenic to animals other than rats (including mice, hamsters, guinea pigs, and monkeys), or in organs other than the rat urinary bladder, where sodium saccharin acted through a carcinogenic mechanism that is not relevant to humans. The preponderance of epidemiologic studies also yielded null results; as summarized by IARC (1999): "In subsequent population-based studies, including a study of several thousand people in the United States, estimates for the entire population of each study did not confirm the existence of an assocation [*sic*]. In some studies, estimates of the strength of the association between consumption of sweeteners and bladder cancer differed between smokers and non-smokers, but the direction of the difference and its distribution between the sexes was inconsistent over the studies." Consequently, IARC downgraded its classification of saccharin and its salts to "not classifiable as to their carcinogenicity to humans (Group 3)" (IARC 1999).

Likewise, in 1997, NTP nominated saccharin to be delisted from its Report on Carcinogens because the scientific data were not deemed sufficient to list the chemical as "reasonably anticipated to be a human carcinogen." Regarding the epidemiologic evidence, NTP stated: "Taken together, the available epidemiology data show no consistent evidence that saccharin is associated with increased urinary-bladder cancer in general; however, a small increased risk in some subgroups, such as heavy users of artificial sweeteners, cannot be unequivocally excluded. With regard to the general population, if sodium saccharin is a risk factor, it is weak, and a causal relationship with cancer cannot be proven or disproven, because of a lack of exposure data and intrinsic limitations of the available epidemiology studies" (NTP 2016a). Thus, null results, including null epidemiologic findings, played an instrumental role in advancing the body of scientific knowledge on saccharin and correcting misconceptions about its potential human carcinogenicity. Far from being "non-informative," null results are essential to the progression of science and public health.

10.0 Ecologic and Semi-Ecologic Studies Cannot Demonstrate Causal Effects

In his expert report, Dr. Sunding describes a number of ecologic and semi-ecologic studies comparing frequencies of low birth weight and premature birth, number of births per 1,000 women of child-bearing age, cancer incidence, and cancer mortality between communities with higher versus lower groundwater PFAS levels in Washington County, Minnesota, or between Washington County and other counties in Minnesota.[17] He interprets differences in frequencies or rates of health outcomes between comparison groups as evidence of an adverse health effect of PFAS.

Studies that compare rates of human health endpoints between residents of certain counties, communities, ZIP codes, or other geographic areas are ecologic studies (or semi-ecologic if individual-level health data are available). In such studies, exposure information is available only at the group level, rather than the level of individual persons. These studies rely on the erroneous assumption that all residents of a given geographic area have the same average level of exposure. This assumption ignores individual variation in exposure based on where people work and travel, the time they spend indoors and outdoors, and the assorted habits of daily life.

Ecologic studies are generally considered by epidemiologists to be among the weakest of epidemiologic study designs because associations observed for groups cannot be assumed to hold for individuals (Greenland and Morgenstern 1989, Morgenstern 1995). That is, average exposure levels or health risks across a group may differ substantially from those for individuals within that group, meaning that individual-level exposures and outcomes can be grossly misclassified in ecologic studies. When both exposures and outcomes are assessed at the group level, there is no assurance that *any* of the individuals who developed a certain health condition actually experienced the exposure of interest. The failure of group-level associations to properly reflect individual-level associations is known as ecologic bias. This bias can occur due to confounding or effect modification by the group-classification variable or other risk factors associated with that variable, even when they are not confounders or effect modifiers at the individual level (Greenland and Morgenstern 1989). Moreover, ecologic bias can be in either direction (toward overestimates or underestimates, including in the opposite direction from the

[17] During his deposition, Dr. Sunding stated that these analyses were "econometric" studies, not epidemiologic studies (368;12–14, 374;8–9, 375;13–14, 380;11–14). However, epidemiologists conventionally describe analyses similar to Dr. Sunding's, in which health outcomes are compared at the group level, as ecologic epidemiologic studies (Greenland and Morgenstern 1989, Morgenstern 1995). For instance, Morgenstern (1995) describes "an ecologic study of cancer incidence by county" and a study in which "we compare the rate of disease among many regions during the same period"—that is, analyses directly comparable to those conducted by Dr. Sunding—as ecologic epidemiologic studies.

individual-level association). In general, standard approaches to controlling for group-level variables that create ecological bias cannot eliminate such bias (Greenland and Morgenstern 1989, Morgenstern 1995).

A well-known historical example of ecologic bias is provided by Robinson (1950), who described a positive correlation between the percentage of the statewide population who were foreign-born (i.e., a group-level exposure) and the percentage of the statewide population who were literate in 1930 (i.e., a group-level outcome). The individual-level interpretation of the ecologic correlation would suggest that foreign-born people were more likely to be literate (in American English) than native-born people. In reality, however, the opposite was true: at the individual level, foreign-born people were less likely to be literate. The biased ecologic association at the group level existed because foreign-born people were more likely to live in states where the native-born were relatively literate. This example shows that due to ecologic bias, an association observed at the group level can be the opposite of the true association at the individual level.

Ecologic studies are especially vulnerable to confounding because information on confounders also is unavailable at the individual level. To control adequately for confounding when estimating the association between an exposure and an outcome, it is necessary for a study to have collected sufficient information on potential confounders. Even if a study has adjusted for a confounder, residual confounding can occur if insufficient information about the confounder has been collected or if the confounder is inappropriately represented in a statistical model. Other methodological limitations of ecologic studies include frequent reliance on crude, incomplete, or inaccurate data; temporal ambiguity regarding whether the exposure preceded the outcome, and if so by how long; strong correlations between many sociodemographic and environmental factors, making it difficult to estimate their separate associations; and potential selection bias due to migration of individuals across groups (Morgenstern 1995). Taken together, the major methodological shortcomings of ecologic studies prevent a causal interpretation of their results.

10.1 Dr. Sunding's Ecologic and Semi-Ecologic Analyses in Washington County, Minnesota, Cannot Demonstrate Causal Effects of Exposure to PFAS in Drinking Water

Dr. Sunding's ecologic and semi-ecologic analyses of health outcomes in Oakdale or Washington County, which are based on Minnesota Department of Health birth data, death records, and aggregated cancer incidence data (STATE_07507028–07507088, STATE_07512741–07513049, STATE_07513050–07513089, and STATE_07513090–07513464), are undermined by the same methodological limitations that preclude all ecologic

analyses from yielding results that can reliably be interpreted as causal. Some of these limitations are described here specifically with respect to Dr. Sunding's analyses. For his analysis of cancer mortality, Dr. Sunding relies on the death certificate data coded by Ms. Schmor; thus, the limitations discussed below with respect to his semi-ecologic analysis of cancer mortality apply also to the data that she provided, insofar as they cannot be used to draw valid conclusions about any causal effect of PFAS exposure on cancer mortality in certain communities.

- No individual-level information (e.g., serum levels) on PFAS exposure is available, nor do the analyses account for individual differences in drinking water sources and consumption patterns, duration of residence in the study area, exposure to PFAS from sources other than drinking water (e.g., food, textiles, occupation), or other influences on PFAS exposure or biological dose received. No evidence is provided to link PFAS levels in the public water supply to serum PFAS levels in the various populations included in the analyses.
- In the semi-ecologic analyses of adverse birth outcomes and the ecologic analysis of birth rate in Oakdale, other "affected communities,"[18] and "unaffected communities" in Washington County, estimated exposure to PFAS is classified based on maternal residential ZIP code at the time of birth. No information is available on residential history, including address during gestation or years before. Given that the estimated half-life of PFOA is over 2 years and that of PFOS is over 4 years, exposure from drinking water during and prior to gestation is more relevant to fetal growth and live births than that at the time of delivery.
    - In light of the relatively long half-life values of these chemicals, the stratification of analyses before and after 2006, when a water filtration facility was constructed in Oakdale, fails to account for any lag in exposure due to persistent PFAS levels in the body.
- In the ecologic analysis of cancer incidence in Washington County and other counties in Minnesota, estimated exposure to PFAS is classified based on residential county at the time of cancer diagnosis. No information is available on residential address years or decades in the past, which is the relevant exposure window for chronic diseases, such as many types of cancer, that have a long latency between causal exposure and disease onset. County-level data are cruder than ZIP-code-level data, combining potentially even more heterogeneous individual-level exposure and confounder profiles into a single, indistinguishable group.
- In the semi-ecologic analysis of cancer deaths in Oakdale, other "affected communities," and other ZIP codes within five miles of the "affected communities," estimated exposure

[18] Dr. Sunding defines the "affected communities" as Cottage Grove, Grey Cloud Island Township, Lake Elmo, Newport, Oakdale, St. Paul Park, West Lakeland Township, and Woodbury; Afton and Denmark Township are excluded because they lack a municipal water supply and have few recent water sampling events.

to PFAS is classified based on residential ZIP code at the time of death. Again, no information is available on residential history, including place of residence years or decades prior, during the course of potential cancer development.

- Dr. Sunding states that misclassification of PFAS exposure due to the use of group-level estimates, and the failure to account for individual differences in drinking water sources and habits, as well as residential history, "will tend to attenuate the magnitude and statistical significance of the patterns identified, so those I report below are conservative in this regard." Similarly, he states: "Measures of exposure are … noisy and, as with the birth analyses, such noisiness attenuates the patterns identified below." These statements are incorrect. On the contrary, ecologic bias can lead to both overestimation and underestimation—including bias so severe that it leads to reversal in the direction—of true individual-level associations (Greenland and Morgenstern 1989, Morgenstern 1995). Therefore, an ecologic analysis cannot, on its own, necessarily be considered to provide any valid information on an individual-level exposure-outcome association.
- Adjustment for potential confounders is minimal; therefore, observed differences in outcomes between communities or counties could be due to unequal distributions of any of these risk factors, rather than differences in PFAS exposure. In the absence of adequate control for individual-level and group-level confounders, associations cannot reliably be attributed to any given exposure.
    - In the analysis of birth outcomes, unadjusted potential confounders include gestational age and maternal BMI, pregnancy weight gain, race/ethnicity, marital status, diet/nutrition, smoking, alcohol intake, drug abuse, kidney function, hypertension (chronic or gestational), diabetes (chronic or gestational), parity, health care access/usage, occupation, and many other factors (Valero De Bernabe et al. 2004).
    - In the analysis of birth rate, unadjusted potential confounders include maternal medical history (e.g., ovulatory disorders, hypertension, thyroid disease, sexually transmitted infection, pelvic inflammatory disease), reproductive history, BMI, race/ethnicity, diet/nutrition, physical activity, smoking, occupation, education, other indicators of socioeconomic status, health care (including assisted reproductive technology) access/usage, and numerous other factors (Kelly-Weeder and O'Connor 2006).
    - In the analysis of cancer incidence, individual-level age—one of the strongest determinants of risk of virtually every type of cancer (Schottenfeld and Fraumeni 2006)—is a key confounder that was not adjusted in the analysis. Other unadjusted potential confounders vary by cancer type, but include individual-level race/ethnicity, socioeconomic status, family history, smoking, alcohol intake, body size, physical activity, diet/nutrition, ionizing radiation exposure, occupation, reproductive history, medical history (e.g., immune disorders, oncogenic infections), and many other factors (Schottenfeld and Fraumeni 2006).

  - In the analysis of cancer deaths, besides the same confounders as for cancer incidence (except for individual-level age, which was available from death certificates), other unadjusted potential confounders include factors that influence cancer prognosis, such as health care access and usage, treatment regimen, and comorbidities.
- The analysis of cancer mortality compares the percentage of death records including cancer across communities; this percentage is influenced not only by the occurrence of cancer, but also the occurrence of other diseases. For example, if the mortality rate from heart disease is lower in Oakdale than in comparison communities, then the percentage of deaths from cancer will correspondingly be greater in Oakdale, but not due to a higher cancer mortality rate. Likewise, if the mortality rate from respiratory disease or accidents is higher in comparison communities than in Oakdale, then the percentage of deaths from cancer will be smaller in comparison communities. Thus, this analysis is susceptible to bias from factors that influence mortality from other major causes of death, not only cancer.

Finally, the results of Dr. Sunding's ecologic and semi-ecologic analyses are not consistent with the preponderance of epidemiologic evidence from studies based on individual-level data, including prospective studies and those based in populations with higher, comparable, or lower serum PFOA and PFOS levels (see Figures 1 and 2). As discussed earlier in this report, most epidemiologic studies found no significant association between exposure to PFOA or PFOS and low birth weight, prematurity, fecundity, infertility, or any cancer type, including non-Hodgkin lymphoma, kidney cancer, bladder cancer, leukemia, prostate cancer, breast cancer, and overall cancer mortality. Thus, Dr. Sunding's analyses do not provide reliable evidence in support of a causal effect of PFOA or PFOS exposure on adverse birth outcomes, birth rate, cancer incidence, or cancer mortality in the communities of Washington County, Minnesota, and other epidemiologic studies do not establish such causal effects in general.

11.0 Regulatory Levels Are Not Thresholds for Health Effects

Government and regulatory agency health-based exposure limits and recommendations cannot be relied upon as a basis for conclusions regarding health injury or causation. Such limits and guidelines are set by governments and agencies charged with protecting public health with an adequate margin of safety based on conservative guidelines, often relying on extrapolation from studies of high exposure levels to low exposure levels, and/or from studies of animals to humans. These extrapolations involve conservative assumptions and the inclusion of safety/uncertainty factors that are intended to guard against harm to human health in general, including sensitive subgroups. For example, in the derivation of reference doses and reference concentrations for toxic substances (i.e., the maximum acceptable chronic doses by the oral and inhalation exposure routes, respectively), U.S. EPA applies specific uncertainty factors intended to account for 1) uncertainty in extrapolating from a lowest observed adverse effect level rather than a no observed adverse effect level; 2) uncertainty in extrapolating animal data to humans; 3) variation in susceptibility among members of the human population; 4) uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure; and 5) uncertainty associated with extrapolation when the scientific database is incomplete (U.S. EPA 2002).

The use of protective exposure limits or recommendations, such as the State of Minnesota's health based values for PFOA, PFOS, and PFBA (Minnesota Department of Health 2017b, a, c) and U.S. EPA's health advisory levels for PFOA and PFOS (U.S. EPA 2016d, b), for determining whether a person has developed an illness or is at significantly increased risk of developing an illness is not scientifically appropriate. According to the Federal Judicial Center and the National Research Council (NRC) of the National Academies, regulatory standards and guidelines are not intended to be used for determining causation in toxic tort litigation (NRC 2011):

> Particularly problematic are generalizations made in personal injury litigation from regulatory positions. Regulatory standards are set for purposes far different than determining the preponderance of evidence in a toxic tort case. For example, if regulatory standards are discussed in toxic tort cases to provide a reference point for assessing exposure levels, it must be recognized that there is a great deal of variability in the extent of evidence required to support different regulations … In addition, regulatory standards traditionally include protective factors to reasonably ensure that susceptible individuals are not put at risk. Furthermore, standards often are based on the risk that results from lifetime exposure. Accordingly, the mere fact that an individual has been exposed to a level above a standard does not necessarily mean that an adverse effect has occurred.

Thus, regulatory exposure levels are often set to demarcate points below which no harm to human health is anticipated, but not thresholds above which adverse health effects will necessarily occur.

To my knowledge, no federal, state, or other regulatory agency has set health-based exposure limits or recommendations for PFOA, PFOS, any other PFAS, or total PFAS based on quantitative risk assessment of human epidemiologic data. Instead, these limits and recommendations have been set based on animal toxicology data. The reliance on results of experimental animal studies to derive protective exposure levels for humans reflects the substantial uncertainty and imprecision in the epidemiologic literature, making the collective epidemiologic database unsuitable for quantitative risk assessment.

12.0 Conclusions

The existing epidemiologic studies of PFOA, PFOS, and other PFAS in relation to human health outcomes are insufficient to establish causation because of their inconsistent findings and multiple methodological limitations. These limitations include, variously, small size, limited control for potential confounding by disease risk factors, lack of adjustment for the confounding or reverse-causal influence of underlying physiological/pharmacokinetic mechanisms on exposure levels and health outcomes, an inability to ascertain all incident disease, cross-sectional or retrospective study design, failure to consider intra-individual variation in PFAS exposure levels and time-varying health outcomes, lack of restriction to fasting and/or unmedicated outcome measures, reliance on self-reported outcomes, and use of an ecologic estimate of PFAS exposure.

To my knowledge, no major health or regulatory agency has concluded that a causal effect has been established between exposure to PFOA, PFOS, or other PFAS and any adverse human health outcome. Given the inconsistent and methodologically limited epidemiologic evidence in the published literature, and the consequent inability to rule out chance, bias, and confounding as alternative explanations of the statistically significant associations reported in the published body of epidemiologic literature, no causal relationship has been established between exposure to PFOA, PFOS, PFBA, PFBS, or other PFAS and any specific human health outcome in general, nor has such a causal relationship been demonstrated by studies of communities in Washington County, Minnesota.

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Appendix A

Summary Tables of Epidemiologic Studies of PFAS

Table 1. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and body size

Group	Reference	Study Design	Study Setting	Age Group	N (Maximum)	Exposure	Significant Associations	Nonsignificant Associations
Body size	Alkhalawi 2016	Prospective cohort	General community (Duisburg, Germany)	Infants	148	PFHxS PFOA PFOS	PFHxS and lower weight in first year PFHxS and greater length in first year	PFHxS and ponderal index in first year PFOA and ponderal index in first year PFOA and weight in first year PFOA and length in first year PFOS and ponderal index in first year PFOS and weight in first year PFOS and length in first year
Body size	Andersen 2010	Prospective cohort	General community (Denmark)	Infants	1,154	PFOA PFOS	PFOA and lower weight z-score at 5 months (boys) PFOA and lower BMI z-score at 5 months (boys) PFOA and lower weight z-score at 12 months (boys) PFOA and lower BMI z-score at 12 months (boys) PFOS and lower weight z-score at 12 months (total, boys) PFOS and lower BMI z-score at 12 months (total, boys)	PFOA and weight z-score at 5 months (total, girls) PFOA and height z-score at 5 months (total, boys, girls) PFOA and BMI z-score at 5 months (total, girls) PFOA and weight z-score at 12 months (total, girls) PFOA and height z-score at 12 months (total, boys, girls) PFOA and BMI z-score at 12 months (total, girls) PFOS and weight z-score at 5 months (total, boys, girls) PFOS and height z-score at 5 months (total, boys, girls) PFOS and BMI z-score at 5 months (total, boys, girls) PFOS and weight z-score at 12 months (girls) PFOS and height z-score at 12 months (total, boys, girls) PFOS and BMI z-score at 12 months (girls)
Body size	Andersen 2013	Prospective cohort	General community (Denmark)	Children	811	PFOA PFOS	None	PFOA and BMI z-score at 7 years (boys, girls) PFOA and waist circumference z-score at 7 years (boys, girls) PFOA and overweight at 7 years (boys, girls) PFOS and BMI z-score at 7 years (boys, girls) PFOS and waist circumference z-score at 7 years (boys, girls) PFOS and overweight at 7 years (boys, girls)
Body size	Ashley-Martin 2016	Prospective and retrospective cohort	General community (Canada)	Pregnant women	1,609	PFHxS PFOA PFOS	PFOA (cord) and greater gestational weight gain (total, boys, girls) PFOS (maternal) and greater gestational weight gain (underweight/normal) PFOS (cord) and greater gestational weight gain (total, girls)	PFHxS (maternal) and gestational weight gain (underweight/normal, overweight, obese) PFHxS (cord) and gestational weight gain (total, boys, girls) PFOA (maternal) and gestational weight gain (underweight/normal, overweight, obese) PFOS (maternal) and gestational weight gain (overweight, obese) PFOS (cord) and gestational weight gain (boys)
Body size	Barry 2014	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Adults	8,764	PFOA	None	PFOA (estimated early-life) and overweight (males, females) PFOA (estimated early-life) and obesity (males, females) PFOA (estimated early-life) and BMI (males, females)

Table 1. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and body size

Group	Reference	Study Design	Study Setting	Age Group	N (Maximum)	Exposure	Significant Associations	Nonsignificant Associations
Body size	Braun 2016	Prospective cohort	General community (Cincinnati, Ohio)	Children	204	PFHxS PFOA PFOS PFNA	PFOA (tertile 2, not 3) and greater BMI z-score at 8 years PFOA (tertile 2, not 3) and greater waist circumference at 8 years PFOA (tertile 2, not 3) and greater body fat percentage at 8 years PFOA (tertile 2, not 3) and greater BMI z-score gain from 2 to 8 years	PFHxS and BMI z-score at 8 years PFHxS and waist circumference at 8 years PFHxS and body fat percentage at 8 years PFHxS and overweight/obesity at 8 years PFHxS and BMI z-score gain from 2 to 8 years PFOA and overweight/obesity at 8 years PFOS and BMI z-score at 8 years PFOS and waist circumference at 8 years PFOS and body fat percentage at 8 years PFOS and overweight/obesity at 8 years PFOS and BMI z-score gain from 2 to 8 years PFNA and BMI z-score at 8 years PFNA and waist circumference at 8 years PFNA and body fat percentage at 8 years PFNA and overweight/obesity at 8 years PFNA and BMI z-score gain from 2 to 8 years
Body size	Cariou 2015	Cross-sectional	General community (Toulouse, France)	Women	100	PFHxS PFOA PFOS PFNA	None	PFHxS and BMI PFOA and BMI PFOS and BMI PFNA and BMI
Body size	Chen 2017	Prospective cohort	General community (Taipei and New Taipei, Taiwan)	Infants, children	429	PFOA PFOS	PFOS and lower weight at 6-12 m and 12-24 m (girls) PFOS and lower weight until 12 m (total) PFOS and greater height at 24-60 m and 60-108 m (boys) PFOS and lower BMI at 6-12 m and 12-24 m (girls) PFOS and greater BMI at 60-108 m (girls)	PFOA and weight at 0-6 m, 6-12 m, 12-24 m, 24-60 m, and 60-108 m (total, boys, girls) PFOA and weight until 6 m, 12 m, 24 m, 60 m, and 108 m (total) PFOA and height at 0-6 m, 6-12 m, 12-24 m, 24-60 m, and 60-108 m (total, boys, girls) PFOA and height until 6 m, 12 m, 24 m, 60 m, and 108 m (total) PFOA and BMI at 0-6 m, 6-12 m, 12-24 m, 24-60 m, and 60-108 m (total, boys, girls) PFOA and BMI until 6 m, 12 m, 24 m, 60 m, and 108 m (total) PFOS and weight at 0-6 m, 6-12 m, 12-24 m, 24-60 m, and 60-108 m (total, boys, girls except 6-12 m and 12-24 m) PFOS and weight until 6 m, 24 m, 60 m, and 108 m (total) PFOS and height at 0-6 m, 6-12 m, 12-24 m, 24-60 m, and 60-108 m (total, boys except 24-60 m and 60-108 m, girls) PFOS and height until 6 m, 12 m, 24 m, 60 m, and 108 m (total) PFOS and BMI at 0-6 m, 6-12 m, 12-24 m, 24-60 m, and 60-108 m (total, boys, girls except 6-12 m, 12-24 m, and 60-108 m) PFOS and BMI until 6 m, 12 m, 24 m, 60 m, and 108 m (total)
Body size	Christensen 2016a	Cross-sectional	Fishing community (Wisconsin anglers)	Men	154	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA	PFOA and lower BMI PFOS and lower BMI PFNA and lower BMI PFDA and lower BMI PFunDA and lower BMI	PFHxS and BMI PFHpS and BMI
Body size	Costa 2009	Cross-sectional	Occupational (Trissino, Italy)	Men	53	PFOA	None	PFOA and body mass index

Table 1. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and body size

Group	Reference	Study Design	Study Setting	Age Group	N (Maximum)	Exposure	Significant Associations	Nonsignificant Associations
Body size	Domazet 2016	Prospective cohort and cross-sectional	General community (Odense, Denmark)	Adolescents, young adults	277	PFOA PFOS	PFOA (21 years) and lower waist circumference at 21 years PFOS (9 years) and greater BMI at 15 years PFOS (9 years) and greater waist circumference at 15 years PFOS (9 years) and greater skinfold thickness at 15 years PFOS (9 years) and greater waist circumference at 21 years PFOS (9 years) and greater skinfold thickness at 21 years	PFOA (9 years) and BMI at 15 years PFOA (9 years) and waist circumference at 15 years PFOA (9 years) and skinfold thickness at 15 years PFOA (9 years) and BMI at 21 years PFOA (9 years) and waist circumference at 21 years PFOA (9 years) and skinfold thickness at 21 years PFOA (15 years) and BMI at 21 years PFOA (15 years) and waist circumference at 21 years PFOA (15 years) and skinfold thickness at 21 years PFOS (9 years) and BMI at 21 years PFOS (15 years) and BMI at 21 years PFOS (15 years) and waist circumference at 21 years PFOS (15 years) and skinfold thickness at 21 years Other cross-sectional associations at 15 and 21 years
Body size	Eriksen 2011	Cross-sectional	General community (Copenhagen area, Denmark)	Men	652	PFOA PFOS	PFOA and lower BMI PFOS and lower BMI	None
Body size	Halldorsson 2012	Prospective cohort	General community (Aarhus, Denmark)	Young adults	665	PFOA PFOS PFOSA PFNA	PFOA and greater BMI at 20 years (females) PFOA and greater waist circumference at 20 years (females) PFOA and greater risk of overweight at 20 years (females) PFOA and greater risk of waist circumference > action level II at 20 years (females)	PFOA and BMI at 20 years (males) PFOA and waist circumference at 20 years (males) PFOA and overweight at 20 years (males) PFOA and waist circumference > action level II at 20 years (males) PFOS and BMI at 20 years (males, females) PFOS and waist circumference at 20 years (males, females) PFOS and overweight at 20 years (males, females) PFOS and waist circumference > action level II at 20 years (males, females) PFOSA and BMI at 20 years (males, females) PFOSA and waist circumference at 20 years (males, females) PFOSA and overweight at 20 years (males, females) PFOSA and waist circumference > action level II at 20 years (males, females) PFNA and BMI at 20 years (males, females) PFNA and waist circumference at 20 years (males, females) PFNA and overweight at 20 years (males, females) PFNA and waist circumference > action level II at 20 years (males, females)

Table 1. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and body size

Group	Reference	Study Design	Study Setting	Age Group	N (Maximum)	Exposure	Significant Associations	Nonsignificant Associations
Body size	Hartman 2017	Prospective cohort	General community (Avon, United Kingdom)	Girls	359	PFHxS PFOA PFOS PFNA	Interaction of PFHxS × maternal education and trunk fat PFOA and lower waist circumference at 9 years Interaction of PFOA × maternal education and total body fat, BMI, and waist circumference PFOS and lower trunk fat at 9 years PFOS and lower BMI at 9 years PFOS and lower waist circumference at 9 years Interaction of PFOS × maternal education and total body fat, trunk fat, and waist circumference	PFHxS and total body fat at 9 years PFHxS and trunk fat at 9 years PFHxS and BMI at 9 years PFHxS and waist circumference at 9 years Interaction of PFHxS × maternal education and total body fat, BMI, and waist circumference PFOA and total body fat at 9 years PFOA and trunk fat at 9 years PFOA and BMI at 9 years Interaction of PFOA × maternal education and trunk fat PFOS and total body fat at 9 years Interaction of PFOS × maternal education and BMI PFNA and total body fat at 9 years PFNA and trunk fat at 9 years PFNA and BMI at 9 years PFNA and waist circumference at 9 years Interaction of PFNA × maternal education and total body fat, trunk fat, BMI, and waist circumference
Body size	Hoyer 2015b	Prospective cohort	Fishing/general community (Greenland; Kharkiv, Ukraine)	Children	1,022	PFOA PFOS	PFOA and greater risk of overweight at 5-9 years (Greenland girls) PFOA (tertile 2, not 3) and greater waist-to-height ratio at 5-9 years (Greenland girls) PFOS and greater risk of waist-to-height ratio > 0.5 at 5-9 years (total, all girls, Greenland girls (tertile 2, not 3))	PFOA and overweight at 5-9 years (total, all boys, all girls, Greenland total, Greenland boys, Ukraine total, Ukraine girls, Ukraine boys) PFOA and waist-to-height ratio > 0.5 at 5-9 years (total, all boys, all girls, Greenland total, Greenland boys, Ukraine total, Ukraine girls, Ukraine boys) PFOS and overweight at 5-9 years (total, all boys, all girls, Greenland total, Greenland girls, Greenland boys, Ukraine total, Ukraine girls, Ukraine boys) PFOS and waist-to-height ratio > 0.5 at 5-9 years (boys, Greenland total, Greenland boys, Ukraine total, Ukraine girls, Ukraine boys)
Body size	Jaacks 2016	Prospective cohort	General community (Michigan; Texas)	Pregnant women	218	PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	PFOS and greater area under the gestational weight gain curve (pre-pregnancy BMI < 25 kg/m^2)	PFOA, PFOS, PFOSA, Et-PFOSA-AcOH, Me-PFOSA-AcOH, PFNA, and PFDeA and gestational weight gain PFOA, PFOS, PFOSA, Et-PFOSA-AcOH, Me-PFOSA-AcOH, PFNA, and PFDeA and inadequate gestational weight gain PFOA, PFOS, PFOSA, Et-PFOSA-AcOH, Me-PFOSA-AcOH, PFNA, and PFDeA and excessive gestational weight gain PFOA, PFOS (pre-pregnancy BMI ≥ 25 kg/m^2), PFOSA, Et-PFOSA-AcOH, Me-PFOSA-AcOH, PFNA, and PFDeA and area under the gestational weight gain curve

Table 1. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and body size

Group	Reference	Study Design	Study Setting	Age Group	N (Maximum)	Exposure	Significant Associations	Nonsignificant Associations
Body size	Karlsen 2017	Prospective cohort and cross-sectional	Fishing community (Faroe Islands)	Children	444	PFHxS PFOA PFOS PFNA PFDA	PFOA (postpartum) and greater risk of overweight at 5 years PFOA (5 years) and lower BMI z-score at 5 years PFOS (postpartum) and greater BMI z-score at 18 months PFOS (postpartum) and greater risk of overweight at 18 months PFNA (5 years) and lower BMI z-score at 5 years PFDA (postpartum) and lower risk of overweight at 5 years (tertile 2, not 3; not continuous) PFDA (5 years) and lower BMI z-score at 5 years PFDA (5 years) and lower risk of overweight at 5 years	PFHxS (postpartum) and BMI z-score and overweight at 18 months PFHxS (postpartum) and BMI z-score and overweight at 5 years PFHxS (5 years) and BMI z-score and overweight at 5 years PFOA (postpartum) and BMI z-score and overweight at 18 months PFOA (postpartum) and BMI z-score at 5 years PFOA (5 years) and overweight at 5 years PFOS (postpartum) and BMI z-score and overweight at 5 years PFOS (5 years) and BMI z-score and overweight at 5 years PFNA (postpartum) and BMI z-score and overweight at 18 months PFNA (postpartum) and BMI z-score and overweight at 5 years PFNA (5 years) and overweight at 5 years PFDA (postpartum) and BMI z-score and overweight at 18 months PFDA (postpartum) and BMI z-score at 5 years
Body size	Kataria 2015	Cross-sectional	General community (United States)	Adolescents	1,960	ΣPFAS (PFHxS+PFOA+PFOS+PFNA)	ΣPFAS and lower risk of obesity	None
Body size	Lin 2009	Cross-sectional	General community (United States)	Adolescents, adults	474 adolescents 969 adults	PFHxS PFOA PFOS PFNA	PFHxS and lower risk of waist circumference component of metabolic syndrome (adolescents) PFOA and lower risk of waist circumference component of metabolic syndrome (adolescents) PFOS and lower risk of waist circumference component of metabolic syndrome (adolescents)	PFHxS and waist circumference component of metabolic syndrome (adults) PFOA and waist circumference component of metabolic syndrome (adults) PFOS and waist circumference component of metabolic syndrome (adults) PFNA and waist circumference component of metabolic syndrome (adolescents, adults)
Body size	Lin 2013a	Cross-sectional	General community (Taiwan)	Adolescents, young adults	664	PFOA PFOS PFNA PFunDA	PFNA and lower BMI	PFOA and BMI PFOS and BMI PFunDA and BMI
Body size	Maisonet 2012	Prospective cohort	General community (Avon, United Kingdom)	Girls	324	PFHxS PFOA PFOS	PFOS and greater weight at 20 months (total, birth weight < 3,200 g (tertile 1))	PFHxS and weight at 20 months PFOA and weight at 20 months PFOS and weight at 20 months (birth weight 3,200-3,580 g or > 3,580 g (tertiles 2 and 3))

Table 1. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and body size

Group	Reference	Study Design	Study Setting	Age Group	N (Maximum)	Exposure	Significant Associations	Nonsignificant Associations
Body size	Mattsson 2015	Prospective case-control	General community (rural Sweden)	Men	231 cases, 231 controls	PFHpA PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA	PFHpA and greater BMI PFOA and greater BMI	PFHxS and BMI PFOS and BMI PFNA and BMI PFDA and BMI PFunDA and BMI PFdoDA and BMI
Body size	Mora 2017	Prospective cohort	General community (Boston area, Massachusetts)	Children	992	PFHxS PFOA PFOS PFNA	PFOS and greater BMI at ~3 years (total) PFOA and greater waist circumference at ~3 years (total, boys) PFHxS and greater subscapular+triceps skinfold thickness at ~3 years (total)	PFHxS, PFOA, PFOS, and PFNA and BMI z-score at ~3 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and waist-to-hip ratio at ~3 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and subscapular-to-triceps skinfold thickness ratio at ~3 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and overweight at ~3 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and obesity at ~3 years (total, boys, girls) PFHxS and BMI and waist circumference at ~3 years (total, boys, girls) PFHxS and subscapular+triceps skinfold thickness at ~3 years (boys, girls) PFOA and BMI and subscapular+triceps skinfold thickness at ~3 years (total, boys, girls) PFOA and waist circumference at ~3 years (girls) PFOS and BMI at ~3 years (boys, girls) PFOS and subscapular+triceps skinfold thickness and waist circumference at ~3 years (total, boys, girls) PFNA and BMI, subscapular+triceps skinfold thickness, and waist circumference at ~3 years (total, boys, girls)

Table 1. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and body size

Group	Reference	Study Design	Study Setting	Age Group	N (Maximum)	Exposure	Significant Associations	Nonsignificant Associations
Body size	Mora 2017 (continued)	Prospective cohort	General community (Boston area, Massachusetts)	Children	873	PFHxS PFOA PFOS PFNA	PFHxS and greater subscapular+triceps skinfold thickness at ~8 years (girls) PFHxS and greater subscapular-to-triceps skinfold thickness ratio at ~8 years (girls) PFOS and greater BMI ~8 years (girls) PFNA and greater BMI at ~8 years (girls) PFNA and greater subscapular+triceps skinfold thickness at ~8 years (total, girls) PFNA and greater subscapular-to-triceps skinfold thickness ratio at ~8 years (total, girls)	PFHxS, PFOA, PFOS, and PFNA and BMI z-score at ~8 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and dual X-ray absorptiometry total fat mass index at ~8 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and dual X-ray absorptiometry total fat-free mass index at ~8 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and waist circumference at ~8 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and waist-to-hip ratio at ~8 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and dual X-ray absorptiometry trunk fat mass index at ~8 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and overweight at ~8 years (total, boys, girls) PFHxS, PFOA, PFOS, and PFNA and obesity at ~8 years (total, boys, girls) PFHxS and BMI at ~8 years (total, boys, girls) PFHxS and subscapular+triceps skinfold thickness and subscapular-to-triceps skinfold thickness ratio at ~8 years (total, boys) PFOA and BMI, subscapular+triceps skinfold thickness, and aubscapular-to-triceps skinfold thickness ratio at ~8 years (total, boys, girls) PFOS and BMI at ~8 years (total, boys) PFOS and subscapular+triceps skinfold thickness and subscapular-to-triceps skinfold thickness ratio at ~8 years (total, boys, girls) PFNA and BMI at ~8 years (total, boys) PFNA and subscapular+triceps skinfold thickness and subscapular-to-triceps skinfold thickness ratio at ~8 years (boys)
Body size	Nelson 2010	Cross-sectional	General community (United States)	Adolescents, adults	1,055	PFHxS PFOA PFOS PFNA	PFHxS and lower BMI (men 20-59 years; women 60-80 years; adults 20-80 years) PFHxS and lower waist circumference (men 20-59 years; adults 20-80 years) PFOS and lower BMI (males 12-19 years) PFOS and greater BMI (men 60-80 years) PFOS and lower waist circumference (males 12-19 years) PFOS and greater waist circumference (men 60-80 years) PFNA and lower waist circumference (males 12-19 years)	PFHxS and BMI (males and females 12-19 years; women 20-59 years; men 60-80 years) PFHxS and waist circumference (males and females 12-19 years; women 20-59 years; men and women 60-80 years) PFOA and BMI (males and females 12-19 years, 20-59 years, and 60-80 years; adults 20-80 years) PFOA and waist circumference (males and females 12-19 years, 20-59 years, and 60-80 years; adults 20-80 years) PFOS and BMI (females 12-19 years; men and women 20-59 years; women 60-80 years; adults 20-80 years) PFOS and waist circumference (females 12-19 years; men and women 20-59 years; women 60-80 years; adults 20-80 years) PFNA and BMI (males and females 12-19 years, 20-59 years, and 60-80 years; adults 20-80 years) PFNA and waist circumference (females 12-19 years; men and women 20-59 years and 60-80 years; adults 20-80 years)
Body size	Olsen 1998	Cross-sectional	Occupational (Cottage Grove, Minnesota)	Men	111	PFOA	None	PFOA and BMI in 1993 and 1995
Body size	Olsen 1999	Cross-sectional	Occupational (Decatur, Alabama; Antwerp, Belgium)	Men	266	PFOS	PFOS and greater BMI in 1995	PFOS and BMI in 1997

Table 1. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and body size

Group	Reference	Study Design	Study Setting	Age Group	N (Maximum)	Exposure	Significant Associations	Nonsignificant Associations
Body size	Olsen 2000	Cross-sectional	Occupational (Cottage Grove, Minnesota)	Men	111 in 1993 80 in 1995 74 in 1997	PFOA	None	PFOA and BMI in 1993, 1995, and 1997
Body size	Olsen 2003a	Prospective cohort and cross-sectional	Occupational (Decatur, Alabama; Antwerp, Belgium)	Adults	263 Decatur 255 Antwerp	PFOS	PFOS and greater BMI (women)	PFOS and BMI (men)
Body size	Olsen 2007	Cross-sectional	Occupational (Antwerp, Belgium; Cottage Grove, Minnesota; Decatur, Alabama)	Men	506	PFOA	PFOA and greater BMI PFOA and greater risk of obesity	None
Body size	Rylander 2009	Cross-sectional	General community (Khanh Hoa province, southern central Vietnam)	Pregnant women	91	PFHxS PFHpS PFOA PFOS PFNA	None	PFHxS and BMI PFHpS and BMI PFOA and BMI PFOS and BMI PFNA and BMI
Body size	Sakr 2007a	Cross-sectional	Occupational (Parkersburg, West Virginia)	Adults	1,019	PFOA	PFOA and greater BMI	None
Body size	Shankar 2011a	Cross-sectional	General community (United States)	Adults	4,587	PFOA PFOS	None	PFOA and BMI PFOS and BMI
Body size	Shankar 2012	Cross-sectional	General community (United States)	Adults	1,216	PFOA	None	PFOA and BMI
Body size	So 2006	Cross-sectional	General community (Zhoushan, China)	Infants, women	19	PFOA PFOS	None	PFOA and infant weight PFOA and maternal weight PFOS and infant weight PFOS and maternal weight
Body size	Timmermann 2014	Cross-sectional	General community (Denmark)	Children	499	PFOA PFOS	None	PFOA and BMI at 8-10 years PFOA and skinfold thickness at 8-10 years PFOA and waist circumference at 8-10 years PFOS and BMI at 8-10 years PFOS and skinfold thickness at 8-10 years PFOS and waist circumference at 8-10 years
Body size	Vested 2013	Prospective cohort	General community (Aarhus, Denmark)	Men	169	PFOA PFOS	None	PFOA and BMI PFOS and BMI

Table 1. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and body size

Group	Reference	Study Design	Study Setting	Age Group	N (Maximum)	Exposure	Significant Associations	Nonsignificant Associations
Body size	Wang 2012	Cross-sectional	Occupational and contaminated community (Changshu City, Jiangsu Province, China)	NR (includes adults)	55 workers 132 residents	PFOA	None	PFOA and BMI
Body size	Wang 2016	Prospective cohort	General community (Taiwan)	Children	162	PFOA PFNA PFDA PFunDA PFdoDA	PFOA and lower weight z-score at 2 years (boys) PFOA and lower height z-score at 2 years (boys) PFNA and lower height z-score at 8 and 11 years (boys) PFDA and lower average weight z-score at 2-11 years (girls) PFDA and lower average height z-score at 2-11 years (girls) PFDA and lower height z-score at 2 and 11 years (girls) PFunDA and lower average weight z-score at 2-11 years (girls) PFunDA and lower average height z-score at 2-11 years (girls) PFunDA and lower height z-score at 11 years (boys) PFdoDA and lower average weight z-score at 2-11 years (girls) PFdoDA and lower average height z-score at 2-11 years (girls) PFdoDA and lower height z-score at 5, 8, and 11 years (boys)	PFOA and average weight z-score and average height z-score at 2-11 years (girls, boys) PFOA and weight z-score at 2 years (girls) and 5, 8, and 11 years (girls, boys) PFOA and height z-score at 2 years (girls) and 5, 8, and 11 years (girls, boys) PFNA and average weight z-score and average height z-score at 2-11 years (girls, boys) PFNA and weight z-score at 2, 5, 8, and 11 years (girls, boys) PFNA and height z-score at 2 and 5 years (girls, boys) and 8 and 11 years (girls) PFDA and average weight z-score and average height z-score at 2-11 years (boys) PFDA and weight z-score at 2, 5, 8, and 11 years (girls, boys) PFDA and height z-score at 5 and 8 years (girls, boys) and 2 and 11 years (boys) PFunDA and average weight z-score and average height z-score at 2-11 years (boys) PFunDA and weight z-score at 2, 5, 8, and 11 years (girls, boys) PFunDA and height z-score at 2, 5, and 8 years (girls, boys) and 11 years (girls) PFdoDA and average weight z-score and average height z-score at 2-11 years (boys) PFdoDA and weight z-score at 2, 5, 8, and 11 years (girls, boys) PFdoDA and height z-score at 2 years (girls, boys) and 5, 8, and 11 years (girls)

Table 2. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and bone and tissue outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Bone or tissue	Galloway 2015	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Women	189	PFOA PFOS	PFOA and lower expression of parathyroid hormone 2 receptor gene (*PTH2R*) (all) PFOA and loewr expression of parathyroid hormone 2 gene (*PTH2*) (postmenopausal) PFOS and lower expression of *PTH2R* (all)	PFOA and expression of parathyroid hormone 2 receptor gene (*PTH2R*) (premenopasual, postmenopausal) PFOA and expression of parathyroid hormone gene (*PTH)* (all, premenopausal, postmenopausal) PFOA and expression of parathyroid hormone-like gene (*PTHLH*) (all, premenopausal, postmenopausal) PFOA and expression of parathyroid hormone 2 gene (*PTH2*) (all, premenopausal) PFOS and expression of *PTH2R* (premenopausal, postmenopausal) PFOS and expression of *PTH* (premenopausal, postmenopausal) PFOS and expression of *PTHLH* (all, premenopausal, postmenopausal) PFOS and expression of *PTH2* (all, premenopausal, postmenopausal)
Bone or tissue	Innes 2011	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	49,432	PFOA PFOS	PFOA and greater risk of osteoarthritis (total, age < 55 years, BMI < 30 kg/m2) PFOS and lower risk of osteoarthritis	PFOA and osteoarthritis (age ≥ 55 years, BMI ≥ 30 kg/m^2)

Table 2. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and bone and tissue outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Bone or tissue	Khalil 2016	Cross-sectional	General community (United States)	Adults	1,914	PFHxS PFOA PFOS PFNA	PFHxS and lower total femur bone mineral density (women, postmenopausal) PFHxS and greater risk of osteoporosis in women PFOA and lower femoral neck bone mineral density (women) PFOA and greater risk of osteoporosis in women PFOS and lower total femur bone mineral density (women, postmenopausal) PFOS and lower femoral neck bone mineral density (men, women, postmenopausal) PFNA and lower total femur bone mineral density (women, premenopausal, postmenopausal) PFNA and lower femoral neck bone mineral density (postmenopausal) PFNA and lower lumbar spine bone mineral density (postmenopausal) PFNA and greater risk of osteoporosis in women	PFHxS and total femur bone mineral density (men, premenopausal) PFHxS and femoral neck bone mineral density (men, women, premenopausal, postmenopausal) PFHxS and lumbar spine bone mineral density (men, women, premenopausal, postmenopausal) PFOA and total femur bone mineral density (men, women, premenopausal, postmenopausal) PFOA and femoral neck bone mineral density (men, premenopausal, postmenopausal) PFOA and lumbar spine bone mineral density (men, women, premenopausal, postmenopausal) PFOS and total femur bone mineral density (men, premenopausal) PFOS and femoral neck bone mineral density (premenopausal) PFOS and lumbar spine bone mineral density (men, women, premenopausal, postmenopausal) PFOS and osteoporosis in women PFNA and total femur bone mineral density (men) PFNA and femoral neck bone mineral density (men, women, premenopausal) PFNA and lumbar spine bone mineral density (men, women, premenopausal)
Bone or tissue	Lin 2014	Cross-sectional	General community (United States)	Adults	2,339	PFOA PFOS	PFOS and lower total lumbar spine bone mineral density (premenopausal)	PFOA and total lumbar spine bone mineral density (men, premenopausal, postmenopausal) PFOA and total hip bone mineral density (men, premenopausal, postmenopausal) PFOA and all fracture, hip/wrist/spine fracture, hip fracture, wrist fracture, or spine fracture (men, premenopausal, postmenopausal) PFOS and total lumbar spine bone mineral density (men, postmenopausal) PFOS and total hip bone mineral density (men, premenopausal, postmenopausal) PFOS and all fracture, hip/wrist/spine fracture, hip fracture, wrist fracture, or spine fracture (men, premenopausal, postmenopausal)
Bone or tissue	Melzer 2010	Cross-sectional	General community (United States)	Adults	3,974	PFOA PFOS	PFOA and greater risk of arthritis ever (quartiles 2 and 3, not 4)	PFOS and arthritis ever

Table 2. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and bone and tissue outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Bone or tissue	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	None	PFOA and osteoarthritis

Table 3. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cancer

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cancer	Alexander 2003	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	2,083	PFOS	PFOS and greater risk of mortality from bladder/other urinary organ cancer (high exposure) PFOS and lower risk of all-cancer mortality (total cohort)	PFOS and mortality from all cancers (high, low, or no exposure) PFOS and mortality from digestive organ/peritoneal cancer PFOS and mortality from esophageal cancer PFOS and mortality from large intestine cancer PFOS and mortality from biliary passage/liver cancer PFOS and mortality from respiratory system cancer PFOS and mortality from bronchus/trachea/lung cancer PFOS and mortality from breast cancer PFOS and mortality from urinary organ cancer PFOS and mortality from bladder/other urinary organ cancer (total cohort, low or no exposure) PFOS and mortality from malignant melanoma PFOS and mortality from lymphatic/hematopoietic cancer
Cancer	Alexander 2007	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,400	PFOS	None	PFOS and bladder cancer
Cancer	Barry 2013	Retrospective cohort	Contaminated community (Mid-Ohio Valley) and occupational (Parkersburg, West Virginia)	Adults	32,254	PFOA	PFOA and lower risk of bladder cancer (occupational) PFOA and lower risk of breast cancer (total 10-year lag only) PFOA and greater risk of kidney cancer (community 10-year lag trend only; community no lag quartiles 3 and 4, no trend; occupational no lag quartile 3, not 4, no trend) PFOA and greater risk of testicular cancer (total, community, community 10-year lag trend only) PFOA and greater risk of thyroid cancer (occupational no lag, occupational 10-year lag)	PFOA and bladder cancer (total, community) PFOA and brain cancer PFOA and breast cancer (total no lag, occupational, community) PFOA and cervical cancer PFOA and colorectal cancer PFOA and esophageal cancer PFOA and kidney cancer (total, occupational) PFOA and leukemia PFOA and liver cancer PFOA and lung cancer PFOA and lymphoma PFOA and melanoma PFOA and oral cancer PFOA and ovarian cancer PFOA and bladder cancer PFOA and pancreatic cancer PFOA and prostate cancer PFOA and soft tissue cancer PFOA and stomach cancer PFOA and testicular cancer (occupational) PFOA and thyroid cancer (total, community) PFOA and uterine cancer

Table 3. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cancer

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cancer	Bonefeld-Jorgensen 2011	Case-control	Fishing community (Greenland Inuit)	Women	31 cases, 115 controls	PFOA PFOS ΣPFSA (PFHxS+PFOS+PFOSA) ΣPFCA (PFHpA+PFOA+PFNA+PFDA+PFunDA+PFdoDA+PFtrDA)	PFOS and greater breast cancer risk ΣPFSA and greater breast cancer risk	PFOA and breast cancer ΣPFCA and breast cancer
Cancer	Bonefeld-Jorgensen 2014	Prospective case-cohort	General community (Denmark)	Women	250 cases, 233 controls	PFHxS PFOA PFOS PFOSA PFNA ΣPFSA (PFBS+PFHxS+PFHpS+PFOS+PFOSA+PFDS) ΣPFCA (PFPA+PFHpA+PFOA+PFNA+PFDA+PFunDA+PFdoDA+PFtrDA+PFteDA)	PFHxS and lower breast cancer risk (total, ≤ 40 years) PFOSA and greater breast cancer risk (total, ≤ 40 years)	PFHxS and breast cancer (> 40 years) PFOA and breast cancer PFOS and breast cancer PFOSA and breast cancer (> 40 years) PFNA and breast cancer ΣPFSA and breast cancer ΣPFCA and breast cancer ΣPFAS and breast cancer

Table 3. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cancer

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cancer	Christensen 2016a	Cross-sectional	Fishing community (Wisconsin anglers)	Men	154	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA	PFOA and greater cancer risk	PFHxS and cancer PFHpS and cancer PFOS and cancer PFNA and cancer PFDA and cancer PFunDA and cancer
Cancer	Consonni 2013	Retrospective cohort	Occupational (Germany, Netherlands, Italy, United Kingdom, New Jersey, West Virginia)	Adults	4,773	PFOA/tetrafluoroethylene (TFE)	PFOA and lower risk of mortality from all cancers PFOA and lower risk of mortality from stomach cancer PFOA and lower risk of mortality from colon cancer PFOA and lower risk of mortality from respiratory cancer PFOA and lower risk of mortality from lung cancer	PFOA and mortality from digestive cancer PFOA and mortality from esophageal cancer PFOA and mortality from stomach cancer PFOA and mortality from rectal cancer PFOA and mortality from liver cancer PFOA and mortality from pancreatic cancer PFOA and mortality from laryngeal cancer PFOA and mortality from genitourinary cancer PFOA and mortality from prostate cancer PFOA and mortality from testicular cancer PFOA and mortality from bladder cancer PFOA and mortality from kidney/other urinary cancer PFOA and mortality from nervous system cancer PFOA and mortality from brain cancer PFOA and mortality from lymphatic/hematopoietic cancer PFOA and mortality from non-Hodgkin lymphoma PFOA and mortality from multiple myeloma PFOA and mortality from leukemia
Cancer	Eriksen 2009	Prospective case-cohort	General community (Denmark)	Adults	713 prostate, 332 bladder, 128 pancreatic, 67 liver; 772 controls (680 men)	PFOA PFOS	None	PFOA and prostate cancer PFOA and bladder cancer PFOA and pancreatic cancer PFOA and liver cancer PFOS and prostate cancer PFOS and bladder cancer PFOS and pancreatic cancer PFOS and liver cancer

Table 3. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cancer

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cancer	Ghisari 2014	Case-control	Fishing community (Greenland Inuit)	Women	31 cases, 115 controls	PFOA PFOS	No significant interactions	PFOA × *CYP1A1* Ile462Val and breast cancer PFOA × *COMT* Val158Met and breast cancer PFOA × *CYP1B1* Leu432Val and breast cancer PFOA × *CYP19* C>T and breast cancer PFOA × *CYP19* (TTTA)n and breast cancer PFOA × *CYP17* -34 T>C and breast cancer PFOS × *CYP1A1* Ile462Val and breast cancer PFOS × *COMT* Val158Met and breast cancer PFOS × *CYP1B1* Leu432Val and breast cancer PFOS × *CYP19* C>T and breast cancer PFOS × *CYP19* (TTTA)n and breast cancer PFOS × *CYP17* -34 T>C and breast cancer
Cancer	Ghisari 2017	Prospective nested case-control	General community (Denmark)	Women	178 cases, 233 controls	PFHxS PFOA PFOS PFOSA	PFHxS and lower breast cancer risk (total) PFOA × *CYP19* C>T and greater breast cancer risk (CC genotype) PFOSA and greater breast cancer risk (total) PFOSA × *COMT* Val158Met and greater breast cancer risk (Met/Met genotype)	PFHxS × *CYP1A1* Ile462Val and breast cancer PFHxS × *CYP1B1* Leu432Val and breast cancer PFHxS × *COMT* Val158Met and breast cancer PFHxS × *CYP17* -34 T>C and breast cancer PFHxS × *CYP19* C>T and breast cancer PFOA and breast cancer risk (total) PFOA × *CYP1A1* Ile462Val and breast cancer PFOA × *CYP1B1* Leu432Val and breast cancer PFOA × *COMT* Val158Met and breast cancer PFOA × *CYP17* -34 T>C and breast cancer PFOS and breast cancer risk (total) PFOS × *CYP1A1* Ile462Val and breast cancer PFOS × *CYP1B1* Leu432Val and breast cancer PFOS × *COMT* Val158Met and breast cancer PFOS × *CYP17* -34 T>C and breast cancer PFOS × *CYP19* C>T and breast cancer PFOSA × *CYP1A1* Ile462Val and breast cancer PFOSA × *CYP1B1* Leu432Val and breast cancer PFOSA × *CYP17* -34 T>C and breast cancer PFOSA × *CYP19* C>T and breast cancer

Table 3. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cancer

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cancer	Gilliland 1993	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,537	PFOA	PFOA and greater mortality from prostate cancer	PFOA and mortality from all cancers PFOA and mortality from gastrointestinal cancer PFOA and mortality from colon cancer PFOA and mortality from pancreatic cancer PFOA and mortality from respiratory cancer PFOA and mortality from lung cancer PFOA and mortality from breast cancer PFOA and mortality from female genital cancer PFOA and mortality from testicular cancer PFOA and mortality from bladder cancer PFOA and mortality from lymphopoietic cancer
Cancer	Grice 2007	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,400	PFOS	None	PFOS and colon cancer PFOS and melanoma PFOS and prostate cancer
Cancer	Hardell 2014	Case-control	General community (Örebro, Sweden)	Men	201 cases, 186 controls	PFHxS PFOA PFOS PFNA PFDA PFunDA	PFHxS and greater risk of hereditary prostate cancer PFHxS x heredity interaction and risk of prostate cancer	PFHxS and prostate cancer (overall, Gleason score 2-6 or 7-10, prostate-specific antigen ≤ 10 or ≥ 11, non-hereditary PFOA and prostate cancer (overall, Gleason score 2-6 or 7-10, prostate-specific antigen ≤ 10 or ≥ 11, hereditary or not) PFOS and prostate cancer (overall, Gleason score 2-6 or 7-10, prostate-specific antigen ≤ 10 or ≥ 11, hereditary or not) PFNA and prostate cancer (overall, Gleason score 2-6 or 7-10, prostate-specific antigen ≤ 10 or ≥ 11, hereditary or not) PFDA and prostate cancer (overall, Gleason score 2-6 or 7-10, prostate-specific antigen ≤ 10 or ≥ 11, hereditary or not) PFunDA and prostate cancer (overall, Gleason score 2-6 or 7-10, prostate-specific antigen ≤ 10 or ≥ 11, hereditary or not)
Cancer	Innes 2014	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	208 cases, 47,151 non-cases	PFOA PFOS	PFOA and lower risk of colorectal cancer (total, men, non-obese, diagnosed in 2000 or later) PFOS and lower risk of colorectal cancer (total, all subgroups)	PFOA and colorectal cancer (women, obese, diagnosed before 2000)

Table 3. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cancer

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cancer	Leonard 2008	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	6,027	PFOA	PFOA and lower risk of mortality from all cancers (vs. US total, West Virginia total, US men, or West Virginia men) PFOA and lower risk of mortality from digestive system cancer (vs. US or West Virginia) PFOA and lower risk of mortality from stomach cancer (vs. US) PFOA and lower risk of mortality from respiratory system cancer (vs. US or West Virginia) PFOA and lower risk of mortality from lung cancer (vs. US or West Virginia) PFOA and lower risk of mortality from prostate cancer (vs. US) PFOA and greater risk of mortality from thyroid/other endocrine gland cancer (vs. DuPont Region 1)	PFOA and mortality from all cancers (vs. DuPont Region 1 total, DuPont Region 1 men, US women, West Virginia women, or DuPont Region 1 women) PFOA and mortality from buccal cavity/pharynx cancer PFOA and mortality from digestive system cancer (vs. DuPont Region 1) PFOA and mortality from esophageal cancer PFOA and mortality from stomach cancer (vs. West Virginia or DuPont Region 1) PFOA and mortality from large intestine cancer PFOA and mortality from rectal cancer PFOA and mortality from liver cancer PFOA and mortality from pancreatic cancer PFOA and mortality from other digestive cancer PFOA and mortality from respiratory system cancer (vs. DuPont Region 1) PFOA and mortality from laryngeal cancer PFOA and mortality from lung cancer (vs. DuPont Region 1) PFOA and mortality from other respiratory cancer PFOA and mortality from breast cancer PFOA and mortality from prostate cancer (vs. West Virginia or Dupont Region 1) PFOA and mortality from testicular/other male genital cancer PFOA and mortality from kidney cancer (vs. US total, West Virginia total, DuPont Region 1 total, US men, West Virginia men, or DuPont Region 1 men) PFOA and mortality from bladder cancer PFOA and mortality from melanoma PFOA and mortality from central nervous system cancer PFOA and mortality from thyroid/other endocrine gland cancer (vs. US or West Virginia)
Cancer	Leonard 2008 (continued)	"	"	"	"	"	"	PFOA and mortality from bone cancer PFOA and mortality from lymphatic/hematopoietic tissue cancer PFOA and mortality from non Hodgkin lymphoma PFOA and mortality from Hodgkin disease PFOA and mortality from leukemia/aleukemia PFOA and mortality from other lymphopoietic cancer PFOA and mortality from all other cancers PFOA and mortality from bladder and other urinary cancers (US men, West Virginia men, DuPont Region 1 men)

Table 3. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cancer

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cancer	Lundin 2009	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,993	PFOA	PFOA and greater mortality from prostate cancer	PFOA and mortality from all cancers PFOA and mortality from buccal cavity/pharynx cancer PFOA and mortality from digestive organ/peritoneal cancer PFOA and mortality from esophageal cancer PFOA and mortality from stomach cancer PFOA and mortality from large intestine cancer PFOA and mortality from rectal cancer PFOA and mortality from biliary passage/liver cancer PFOA and mortality from pancreatic cancer PFOA and mortality from other digestive organ cancer PFOA and mortality from respiratory system cancer PFOA and mortality from laryngeal cancer PFOA and mortality from trachea/bronchus/lung cancer PFOA and mortality from other respiratory system cancer PFOA and mortality from breast cancer PFOA and mortality from female genital organ cancer PFOA and mortality from cervical cancer PFOA and mortality from other female genital organ cancer PFOA and mortality from male genital organ cancer PFOA and mortality from other male genital organ cancer PFOA and mortality from urinary organ cancer PFOA and mortality from kidney cancer PFOA and mortality from bladder/other urinary organ cancer PFOA and mortality from other/unspecified cancer PFOA and mortality from skin cancer PFOA and mortality from central nervous system cancer PFOA and mortality from thyroid cancer
Cancer	Lundin 2009	"	"	"	"	"	"	PFOA and mortality from lymphatic/hematopoietic tissue cancer PFOA and mortality from lymphosarcoma/reticulosarcoma PFOA and mortality from Hodgkin's disease PFOA and mortality from leukemia and aleukemia PFOA and mortality from other lymphosarcoma/reticulosarcoma PFOA and mortality from all other cancers
Cancer	Olsen 2004	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,311	PFOS	PFOS and greater risk of episodes of care for malignant melanoma of skin (chemical workers)	PFOS and episodes of care for colon cancer PFOS and episodes of care for liver cancer PFOS and episodes of care for rectal cancer PFOS and episodes of care for lower respiratory tract cancer PFOS and episodes of care for malignant melanoma of skin (long-term chemical workers) PFOS and episodes of care for bladder cancer PFOS and episodes of care for prostate cancer PFOS and episodes of care for thyroid cancer

Table 3. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cancer

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cancer	Pirali 2009	Case-control	General community (Pavia, Italy)	Adults	4-6 cases 7-10 controls	PFOA PFOS	None	PFOA and differentiated thyroid cancer PFOS and differentiated thyroid cancer
Cancer	Raleigh 2014	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	9,027	PFOA	PFOA and lower risk of all-cause mortality (total; quartile 1, not 2, 3, 4) PFOA and lower risk of mortality from all cancers (total; quartile 1, not 2, 3, 4) PFOA and lower risk of breast cancer incidence (quartiles 1-2, not 3-4)	PFOA and mortality from liver cancer PFOA and mortality from pancreatic cancer PFOA and mortality from prostate cancer PFOA and mortality from kidney cancer PFOA and mortality from breast cancer PFOA and mortality from bladder cancer PFOA and prostate cancer PFOA and kidney cancer PFOA and pancreatic cancer PFOA and bladder cancer
Cancer	Steenland 2012	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	5,791	PFOA	PFOA and lower risk of bladder cancer (no lag Q4 and trend log-cumulative exposure, 10-year lag only for trend across categories)	PFOA and mortality from all cancers PFOA and mortality from liver cancer PFOA and mortality from pancreatic cancer PFOA and mortality from lung cancer PFOA and mortality from breast cancer PFOA and mortality from prostate cancer PFOA and mortality from testicular cancer PFOA and mortality from bladder cancer PFOA and mortality from non-Hodgkin's lymphoma PFOA and mortality from leukemia
Cancer	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	PFOA and lower risk of bladder cancer (no lag, 10-year lag)	PFOA and colorectal cancer PFOA and prostate cancer PFOA and melanoma
Cancer	Ubel 1980	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,688	PFOA	None	PFOA and "specific causes of death due to cancer"
Cancer	Vassiliadou 2010	Cross-sectional	General community (Athens area, Greece)	Adults	40 cases, 142 non-cases	PFOA PFOS	PFOA and unequal risk of unspecified cancer among Athens controls (highest PFOA), Argolida controls (lowest PFOA), and cases (middle PFOA)	PFOS and unspecified cancer

Table 3. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cancer

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cancer	Vieira 2013	Case-control	Contaminated community (Mid-Ohio Valley)	Adults	25,107 cases (≤ 4,926 site-specific cases)	PFOA	PFOA and greater risk of brain cancer (medium serum PFOA) PFOA and greater risk of kidney cancer (Tupper Plains district; high serum PFOA; very high or high serum PFOA with 10-year residency and latency) PFOA and greater risk of lung cancer (total exposed districts; Mason district; high serum PFOA with 10-year residency and latency and multiple imputation) PFOA and greater risk of melanoma of the skin (medium serum PFOA with 10-year residency and latency and multiple imputation) PFOA and lower risk of melanoma of the skin (Mason district) PFOA and greater risk of testicular cancer (Little Hocking district) PFOA and greater risk of uterine cancer (high serum PFOA)	PFOA and bladder cancer PFOA and brain cancer (total exposed districts, all water districts, very high or high serum PFOA) PFOA and female breast cancer PFOA and cervical cancer PFOA and colorectal cancer PFOA and kidney cancer (total exposed districts, all water districts, very high or medium serum PFOA) PFOA and leukemia PFOA and liver cancer PFOA and lung cancer (all other water districts, all other serum PFOA groups) PFOA and melanoma of the skin (all other water districts and all serum PFOA groups) PFOA and multiple myeloma PFOA and non-Hodgkin lymphoma PFOA and ovarian cancer PFOA and pancreatic cancer PFOA and prostate cancer PFOA and testicular cancer (total exposed districts, all water districts except Little Hocking, all serum PFOA groups) PFOA and thyroid cancer PFOA and uterine cancer (total expsed districts, all water districts, very high serum or medium PFOA)
Cancer	Wielsoe 2017	Case-control	Fishing community (Greenland Inuit)	Women	77 cases, 84 controls	PFHpA PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA ΣPFSAs (PFHxS+POS) ΣPFCAs (all others) ΣPFAAs (all)	PFHxS and greater breast cancer risk PFOA and greater breast cancer risk PFOS and greater breast cancer risk PFNA and greater breast cancer risk (tertile 2, not 3) PFDA and greater breast cancer risk ΣPFSAs and greater breast cancer risk ΣPFCAs and greater breast cancer risk (tertile 2, not 3) ΣPFAAs and greater breast cancer risk	PFHpA and breast cancer PFunDA and breast cancer PFdoDA and breast cancer ΣPFCAs and breast cancer

Table 4. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cardiovascular outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cardiovascular	Alexander 2003	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	2,083	PFOS	PFOS and lower risk of mortality from all heart disease (total cohort, high exposure, no exposure)	PFOS and mortality from cerebrovascular disease PFOS and mortality from all heart disease (low exposure)
Cardiovascular	Anderson-Mahoney 2008	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	566	PFOA	Residence in PFOA water district and greater risk of cardiovascular problems (total; males and females, all ages) Residence in PFOA water district and greater risk of high blood pressure (males 18-34, 35-49, 50-64, and 65+ y; females 50-64 y) Residence in PFOA water district and lower risk of high blood pressure (females 65+ y)	Residence in PFOA water district and high blood pressure (total; females 18-34 and 35-49 y)
Cardiovascular	Bao 2017	Cross-sectional	Contaminated community (Shenyang, China)	Adults	1,612	PFBA PFBS PFPA PFHpA PFHxA PFHxS PFOA (total, n-, 6m-) PFOS (total, n-, total Br-, 1m-, 6m-, 3+4+5m-, Σ2m-) PFNA PFDA PFDS PFunDA PFdoDA PFtrDA PFteDA	PFBA and greater risk of hypertension (all groups) 6m-PFOA, total PFOS, total-Br-PFOS, 3+4+5m-PFOS, 6m-PFOS, and PFNA and greater risk of hypertension (total, women) Σ2m-PFOS and greater risk of hypertension (total) PFPA, PFHxA, n-PFOS, and 1m-PFOS and greater risk of hypertension (women) PFBA and PFHpA and greater systolic blood pressure (total, men) Total-Br-PFOS, 6m-PFOS, 3+4+5m-PFOS, and Σ2m-PFOS and greater systolic blood pressure (all groups) Total PFOA, n-PFOA, total PFOS, n-PFOS, 1m-PFOS, and PFNA and greater systolic blood pressure (total, women) PFPA, PFDS, and PFdoDA and greater systolic blood pressure (women) PFHpA and greater diastolic blood pressure (men) 6m-PFOA, Σ2m-PFOS, and PFDS and greater diastolic blood pressure (total) Total PFOA, n-PFOA, and PFDA and greater diastolic blood pressure (total, men) Total PFOS, n-PFOS, total-Br-PFOS, 1m-PFOS, 6m-PFOS, 3+4+5m-PFOS, and PFdoDA and greater diastolic blood pressure (total, women) PFNA and greater diastolic blood pressure (all groups)	PFBS, PFHxS, PFHpA, total PFOA, n-PFOA, PFDA, PFDS, PFunDA, PFdoDA, PFtrDA, and PFteDA and hypertension (all groups) PFPA, PFHxA, n-PFOS, and 1m-PFOS and hypertension (total, men) Σ2m-PFOS and greater risk of hypertension (men, women) 6m-PFOA, total PFOS, total-Br-PFOS, 3+4+5m-PFOS, 6m-PFOS, and PFNA and hypertension (men) PFBS, PFHxA, PFHxS, 6m-PFOA, PFDA, PFunDA, PFtrDA, and PFteDA and systolic blood pressure (all groups) PFBA and PFHpA and systolic blood pressure (women) PFPA, PFDS, and PFdoDA and systolic blood pressure (total, men) Total PFOA, n-PFOA, total PFOS, n-PFOS, 1m-PFOS, and PFNA and systolic blood pressure (men) PFBA, PFBS, PFPA, PFHxA, PFHxS, PFunDA, PFtrDA, and PFteDA and diastolic blood pressure (all groups) PFHpA and diastolic blood pressure (total, women) 6m-PFOA, Σ2m-PFOS, and PFDS and diastolic blood pressure (women, men) Total PFOA, n-PFOA, and PFDA and diastolic blood pressure (women) Total PFOS, n-PFOS, total-Br-PFOS, 1m-PFOS, 6m-PFOS, 3+4+5m-PFOS, and PFdoDA and diastolic blood pressure (men)

Table 4. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cardiovascular outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cardiovascular	Christensen 2016a	Cross-sectional	Fishing community (Wisconsin anglers)	Men	154	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA	PFNA and lower hypertension risk	PFHxS and any cardiovascular PFHxS and coronary heart disease PFHxS and hypertension PFHpS and any cardiovascular PFHpS and coronary heart disease PFHpS and hypertension PFOA and any cardiovascular PFOA and coronary heart disease PFOA and hypertension PFOS and any cardiovascular PFOS and coronary heart disease PFOS and hypertension PFNA and any cardiovascular PFNA and coronary heart disease PFDA and any cardiovascular PFDA and coronary heart disease PFDA and hypertension PFunDA and any cardiovascular PFunDA and coronary heart disease PFunDA and hypertension
Cardiovascular	Consonni 2013	Retrospective cohort	Occupational (Germany, Netherlands, Italy, United Kingdom, New Jersey, West Virginia)	Adults	4,773	PFOA/tetrafluoroethylene (TFE)	None	PFOA and circulatory disease mortality
Cardiovascular	Geiger 2014b	Cross-sectional	General community (United States)	Adolescents	1,655	PFOA PFOS	None	PFOA and systolic blood pressure PFOA and diastolic blood pressure PFOA and hypertension PFOS and systolic blood pressure PFOS and diastolic blood pressure PFOS and hypertension
Cardiovascular	Gilliland 1993	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,537	PFOA	PFOA and lower risk of mortality from cardiovascular disease (men, men in chemical division) PFOA and lower risk of mortality from coronary and atherosclerotic heart disease (men)	PFOA and mortality from cardiovascular disease (women) PFOA and mortality from coronary and atherosclerotic heart disease (men in chemical division) PFOA and mortality from cerebrovascular disease

Table 4. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cardiovascular outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cardiovascular	Kataria 2015	Cross-sectional	General community (United States)	Adolescents	1,960	ΣPFAS (PFHxS+PFOA+PFOS+PFNA)	None	ΣPFAS and blood pressure ≥ 90th percentile
Cardiovascular	Leonard 2008	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	6,027	PFOA	PFOA and lower risk of mortality from cerebrovascular disease (vs. US men or West Virginia men) PFOA and lower risk of mortality from all heart disease (vs. US or West Virginia) PFOA and lower risk of mortality from ischemic heart disease (vs. US total, West Virginia total, US men, or West Virginia men) PFOA and lower risk of mortality from hypertension with heart disease PFOA and lower risk of mortality from all other heart disease (vs. West Virginia)	PFOA and mortality from cerebrovascular disease (vs. DuPont Region 1 men, US women, West Virginia women, or DuPont Region 1 women) PFOA and mortality from all heart disease (vs. DuPont Region 1) PFOA and mortality from rheumatic heart disease PFOA and mortality from ischemic heart disease (vs. DuPont Region 1 total, DuPont Region 1 men, US women, West Virginia women, or DuPont Region 1 women) PFOA and mortality from chronic endocardial disease and other myocardial insufficiencies PFOA and mortality from all other heart disease (vs. US or DuPont Region 1) PFOA and mortality from hypertension without heart disease
Cardiovascular	Lin 2013a	Cross-sectional	General community (Taipei, Taiwan)	Adolescents, young adults	664	PFOA PFOS PFNA PFunDA	PFOS and greater carotid intima-media thickness (total, females, 12-19 years, BMI < 24 kg/m^2, never smoked, *APOE* E2 carrier or E3/E3) PFNA and lower carotid intima-media thickness	PFOA and systolic blood pressure PFOA and carotid intima-media thickness PFOS and systolic blood pressure PFOS and carotid intima-media thickness (males, 20-30 years, ≥ 24 kg/m^2, ever smoked, *APOE* E4 carrier) PFNA and systolic blood pressure PFunDA and systolic blood pressure PFunDA and carotid intima-media thickness

Table 4. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cardiovascular outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cardiovascular	Lin 2016	Cross-sectional	General community (Taipei, Taiwan)	Adolescents, young adults	848	PFOA PFOS PFNA PFunDA	PFOA and lower levels of CD31+/CD42a- endothelial microparticles PFOS and greater levels of CD31+/CD42a- endothelial microparticles PFOS and greater levels of CD31+/CD42a+ platelet microparticles PFOS and greater carotid intima-media thickness (especially where CD31+/CD42a- > 50% and CD31+/CD42a+ > 50%) PFNA and lower levels of CD31+/CD42a- endothelial microparticles PFNA and lower levels of CD31+/CD42a+ platelet microparticles PFNA and lower carotid intima-media thickness (where CD31+/CD42a- > 50%) PFunDA and lower levels of CD62E endothelial microparticles PFunDA and lower levels of CD31+/CD42a- endothelial microparticles PFunDA and lower carotid intima-media thickness	PFOA and CD62E endothelial microparticles PFOA and CD62P platelet microparticles PFOA and CD31+/CD42a+ platelet microparticles PFOA and 8-hydroxy-2' -deoxyguanosine (8-OHDG; oxidative stress) PFOA and carotid intima-media thickness PFOS and CD62E endothelial microparticles PFOS and CD62P platelet microparticles PFOS and 8-OHDG PFNA and CD62E endothelial microparticles PFNA and CD62P platelet microparticles PFNA and 8-OHDG PFNA and carotid intima-media thickness PFunDA and CD62P platelet microparticles PFunDA and CD31+/CD42a+ platelet microparticles PFunDA and 8-OHDG

Table 4. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cardiovascular outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cardiovascular	Lind 2017c	Cross-sectional	General community (Uppsala, Sweden)	Older adults	1,016	PFHpA PFHxS PFOA Linear PFOS PFOSA PFNA PFDA PFunDA	PFOSA and greater carotid intima-media thickness (total, women) PFNA and greater intima-media complex echogenicity (women) PFunDA and greater number of carotid arteries with atherosclerotic plaques (women)	PFHpA and carotid intima-media thickness PFHpA and intima-media complex echogenicity PFHpA and # carotid arteries with atherosclerotic plaques PFHxS and carotid intima-media thickness PFHxS and intima-media complex echogenicity PFHxS and # carotid arteries with atherosclerotic plaques PFOA and carotid intima-media thickness PFOA and intima-media complex echogenicity PFOA and # carotid arteries with atherosclerotic plaques Linear PFOS and carotid intima-media thickness Linear PFOS and intima-media complex echogenicity Linear PFOS and # carotid arteries with plaques PFOSA and carotid intima-media thickness (men) PFOSA and intima-media complex echogenicity PFOSA and # carotid arteries with atherosclerotic plaques PFNA and carotid intima-media thickness PFNA and intima-media complex echogenicity (total, men) PFNA and # carotid arteries with atherosclerotic plaques PFDA and carotid intima-media thickness PFDA and intima-media complex echogenicity PFDA and # carotid arteries with atherosclerotic plaques PFunDA and carotid intima-media thickness PFunDA and intima-media complex echogenicity PFunDA and # carotid arteries with plaques (total, men)
Cardiovascular	Lundin 2009	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,993	PFOA	PFOA and lower risk of mortality from all heart disease (ever probable/never definite exposure) PFOA and lower risk of mortality from ischemic heart disease (ever probable/never definite exposure)	PFOA and mortality from cerebrovascular disease PFOA and mortality from all heart disease (ever definite exposure) PFOA and mortality from rheumatic heart disease PFOA and mortality from ischemic heart disease (ever definite exposure) PFOA and mortality from chronic disease of endocardium PFOA and mortality from hypertension with heart disease PFOA and mortality from other heart disease PFOA and mortality from hypertension without heart disease

Table 4. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cardiovascular outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cardiovascular	Mattsson 2015	Prospective case-control	General community (rural Sweden)	Men	231 cases, 231 controls	PFHpA PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA	PFHpA at baseline and greater risk of coronary heart disease (quartile 3, not 4) PFunDA at baseline and lower systolic blood pressure	PFHpA at baseline and systolic and diastolic blood pressure PFHxS at baseline and coronary heart disease PFHxS change from 1990/91 to 2002/03 and coronary heart disease PFHxS at baseline and systolic and diastolic blood pressure PFOA at baseline and coronary heart disease PFOA change from 1990/91 to 2002/03 and coronary heart disease PFOA at baseline and systolic and diastolic blood pressure PFOS at baseline and coronary heart disease PFOS change from 1990/91 to 2002/03 and coronary heart disease PFOS at baseline and systolic and diastolic blood pressure PFNA at baseline and coronary heart disease PFNA change from 1990/91 to 2002/03 and coronary heart disease PFNA at baseline and systolic and diastolic blood pressure PFDA at baseline and coronary heart disease PFDA change from 1990/91 to 2002/03 and coronary heart disease PFDA at baseline and systolic and diastolic blood pressure PFunDA at baseline and coronary heart disease PFunDA change from 1990/91 to 2002/03 and coronary heart disease PFunDA at baseline and diastolic blood pressure
Cardiovascular	Melzer 2010	Cross-sectional	General community (United States)	Adults	3,974	PFOA PFOS	None	PFOA and heart disease ever PFOS and heart disease ever
Cardiovascular	Min 2012	Cross-sectional	General community (United States)	Adults	2,934	PFOA	PFOA and greater homocysteine level PFOA and greater systolic blood pressure PFOA and greater risk of hypertension	PFOA and diastolic blood pressure

Table 4. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cardiovascular outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cardiovascular	Nolan 2010	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Women	1,548	PFOA	None	PFOA (Little Hocking Water Association only) and maternal cardiac disease PFOA (Little Hocking Water Association only) and maternal chronic hypertension PFOA (partial Little Hocking Water Association) and maternal cardiac disease PFOA (partial Little Hocking Water Association) and maternal chronic hypertension
Cardiovascular	Olsen 2004	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,311	PFOS	None	PFOS and episodes of care for hypertension PFOS and episodes of care for atherosclerotic coronary vascular disease and cardiac arrest
Cardiovascular	Raleigh 2014	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	9,027	PFOA	PFOA and lower risk of ischemic heart disease mortality	PFOA and mortality from cerebrovascular disease
Cardiovascular	Sakr 2009	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	4,747	PFOA	None	PFOA and ischemic heart disease mortality
Cardiovascular	Shankar 2011a	Cross-sectional	General community (United States)	Adults	4,587	PFOA PFOS	PFOA and greater systolic blood pressure PFOA and greater diastolic blood pressure PFOS and greater systolic blood pressure PFOS and greater diastolic blood pressure	None
Cardiovascular	Shankar 2012	Cross-sectional	General community (United States)	Adults	1,216	PFOA	PFOA and greater risk of cardiovascular disease PFOA and greater risk of peripheral arterial disease (i.e., ankle-brachial blood pressure index < 0.9) PFOA and greater risk of cardiovascular disease or peripheral arterial disease	PFOA and hypertension
Cardiovascular	Simpson 2013	Retrospective cohort	Contaminated community (Mid-Ohio Valley) and occupational (Parkersburg, West Virginia)	Adults	32,254	PFOA	PFOA and greater risk of stroke (retrospective analysis quintiles 2, 3, 4, not 5; no trend)	PFOA and risk of stroke (prospective analysis)
Cardiovascular	Steenland 2012	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	5,791	PFOA	None	PFOA and mortality from ischemic heart disease PFOA and mortality from stroke

Table 4. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and cardiovascular outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Cardiovascular	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	None	PFOA and coronary heart disease PFOA and medicated hypertension PFOA and stroke
Cardiovascular	Winquist 2014a	Retrospective cohort	Contaminated community (Mid-Ohio Valley) and occupational (Parkersburg, West Virginia)	Adults	32,254	PFOA	PFOA and lower risk of coronary artery disease, prospective analysis (total cohort) PFOA and lower risk of hypertension treated with medication, prospective analysis (total cohort)	PFOA and coronary artery disease, retrospective analysis (total cohort; all age/sex subgroups) PFOA and hypertension treated with medication, retrospective analysis (total cohort; all age/sex subgroups)

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Alkhalawi 2016	Prospective cohort	General community (Germany)	Newborns	148	PFHxS PFOA PFOS	PFOA and lower ponderal index at birth PFOS and lower ponderal index at birth	PFHxS and ponderal index at birth PFHxS and birth weight PFHxS and birth length PFOA and birth weight PFOA and birth length PFOS and birth weight PFOS and birth length
Fetal growth	Andersen 2010	Prospective cohort	General community (Denmark)	Newborns	1,118	PFOA PFOS	PFOA and lower birth weight (total) PFOS and lower birth weight (girls)	PFOA and birth weight (boys, girls) PFOS and birth weight (total, boys)
Fetal growth	Antignac 2013	Retrospective	General community (France)	Newborns	48	PFHxS+PFOA+PFOS	None	PFHxS+PFOA+PFOS and birthweight
Fetal growth	Apelberg 2007	Cross-sectional	General community (Baltimore, Maryland)	Newborns	293	PFOA PFOS	PFOA and lower birth weight PFOA and lower ponderal index PFOA and smaller head circumference PFOS and lower birth weight PFOS and lower ponderal index PFOS and smaller head circumference	PFOA and gestational age PFOA and birth length PFOS and gestational age PFOS and birth length
Fetal growth	Ashley-Martin 2017	Prospective cohort and cross-sectional	General community (Canada)	Newborns	1,705	PFHxS PFOA PFOS	None	PFHxS and birth weight z-score (overall and within subgroups of gestational weight gain or infant sex) PFOA and birth weight z-score (overall and within subgroups of gestational weight gain or infant sex) PFOS and birth weight z-score (overall and within subgroups of gestational weight gain or infant sex)

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Bach 2016	Prospective cohort	General community (Aarhus, Denmark)	Newborns	1,507	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA	Interaction between PFOS and sex for birth weight (nonsignificantly positive in boys, nonsignificantly negative in girls)	PFHxS and birth weight, birth weight z-score, birth length, head circumference, gestational age, and preterm birth PFHpS and birth weight, birth weight z-score, birth length, head circumference, gestational age, and preterm birth PFOA and birth weight, birth weight z-score, birth length, head circumference, gestational age, and preterm birth PFOS and birth weight, birth weight z-score, birth length, head circumference, gestational age, and preterm birth PFNA and birth weight, birth weight z-score, birth length, head circumference, gestational age, and preterm birth PFDA and birth weight, birth weight z-score, birth length, head circumference, gestational age, and preterm birth PFunDA and birth weight, birth weight z-score, birth length, head circumference, gestational age, and preterm birth
Fetal growth	Berg 2017	Prospective cohort	General community (northern Norway)	Newborns	370	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA ΣPFAS (PFHxS+PFHpS+PFOA+PFOS+PFNA+PFDA+PFunDA)	None	PFHxS and gestational age, birth weight, head circumference, and birth length PFHpS and gestational age, birth weight, head circumference, and birth length PFOA and gestational age, birth weight, head circumference, and birth length PFOS and gestational age, birth weight, head circumference, and birth length PFNA and gestational age, birth weight, head circumference, and birth length PFDA and gestational age, birth weight, head circumference, and birth length PFunDA and gestational age, birth weight, head circumference, and birth length ΣPFAS and gestational age, birth weight, head circumference, and birth length

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Callan 2016	Prospective cohort	General community (western Australia)	Newborns	98	PFHxS PFOA PFOS PFNA PFDA PFunDA	PFHxS and greater risk of being < 95% optimal birth weight PFunDA and greater proportion of optimal birth weight	PFHxS and birth weight, proportion of optimal birth weight, gestational age, birth length, proportion of optimal birth length, head circumference,proportional optimal head circumference, and ponderal index PFOA and birth weight, proportion of optimal birth weight, < 95% optimal birth weight, gestational age, birth length, proportion of optimal birth length, head circumference, proportional optimal head circumference, and ponderal index PFOS and birth weight, proportion of optimal birth weight, < 95% optimal birth weight, gestational age, birth length, proportion of optimal birth length, head circumference, proportional optimal head circumference, and ponderal index PFNA and birth weight, proportion of optimal birth weight, < 95% optimal birth weight, gestational age, birth length, proportion of optimal birth length, head circumference, proportional optimal head circumference, and ponderal index PFDA and birth weight, proportion of optimal birth weight, < 95% optimal birth weight, gestational age, birth length, proportion of optimal birth length, head circumference, proportional optimal head circumference, and ponderal index PFunDA birth weight, < 95% optimal birth weight, gestational age, birth length, proportion of optimal birth length, head circumference, proportional optimal head circumference, and ponderal index
Fetal growth	Cariou 2015	Cross-sectional	General community (Toulouse, France)	Newborns	100	PFHxS PFOA PFOS PFNA	PFHxS and greater gestational age PFOS and greater gestational age	PFHxS and birth weight PFOA and birth weight PFOA and gestational age PFOS and birth weight PFNA and birth weight PFNA and gestational age

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Chen 2012	Prospective cohort	General community (Taipei, Taiwan)	Newborns	429	PFOA PFOS PFNA PFunDA	PFOS and lower gestational age PFOS and lower birth weight PFOS and smaller head circumference PFOS and greater risk of preterm birth PFOS and greater risk of small for gestational age PFNA and greater birth length PFNA and lower ponderal index	PFOA and gestational age, birth weight, birth length, head circumference, ponderal index, preterm birth, low birth weight, and small for gestational age PFOS and birth length, ponderal index, and low birth weight PFNA and gestational age, birth weight, head circumference, pretern birth, low birth weight, and small for gestational age PFunDA and gestational age, birth weight, birth length, head circumference, ponderal index, preterm birth, low birth weight, and small for gestational age
Fetal growth	Chen 2017	Cross-sectional	General community (Taipei and New Taipei, Taiwan)	Newborns	429	PFOA PFOS	PFOA and lower birth weight (boys) PFOA and lower BMI at birth (boys) PFOS and lower birth weight (total) PFOS and shorter birth length (total)	PFOA and birth weight (total, girls) PFOA and birth length (total, boys, girls) PFOA and BMI at birth (total, girls) PFOS and birth weight (boys, girls) PFOS and birth length (boys, girls) PFOS and BMI at birth (total, boys, girls)
Fetal growth	Darrow 2013	Prospective cohort	Contaminated community (Mid-Ohio Valley)	Newborns	1,630	PFOA PFOS	PFOS and lower birth weight (quintile 4, not 5 for all births; first prospective birth)	PFOA and preterm birth PFOA and low birth weight PFOA and birth weight PFOS and preterm birth PFOS and low birth weight
Fetal growth	de Cock 2016	Cross-sectional	General community (Zwolle, Netherlands)	Newborns	89	PFOA PFOS	PFOS and greater birth weight (total, boys)	PFOA and birth weight (total, girls, boys) PFOS and birth weight (girls)
Fetal growth	Fei 2007	Prospective cohort	General community (Denmark)	Newborns	1,387	PFOA PFOS	PFOA and lower birth weight PFOA and greater risk of preterm birth (quartile 2, not 3 or 4) PFOS and greater risk of preterm birth (quartile 3, not 4)	PFOA and gestational age PFOA and low birth weight PFOA and small for gestational age PFOS and birth weight PFOS and gestational age PFOS and low birth weight PFOS and small for gestational age

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Fei 2008b	Prospective cohort	General community (Denmark)	Newborns	1,376	PFOA PFOS	PFOA and shorter birth length PFOA and lower abdominal circumference PFOA and greater ponderal index (nulliparous women, prepregnancy BMI ≥ 30 kg/m^2)	PFOA and placental weight PFOA and head circumference PFOA and ponderal index (multiparous, prepregnancy BMI < 30 kg/m^2) PFOS and placental weight PFOS and birth length PFOS and head circumference PFOS and abdominal circumference
Fetal growth	Fromme 2010	Prospective cohort	General community (Munich, Germany)	Newborns	44	PFOA	None	PFOA and birth weight (per Johnson et al. 2014)
Fetal growth	Govarts 2016	Prospective cohort	General community (Flanders, Belgium)	Newborns	248	PFOA PFOS	None	PFOA and birth weight PFOS and birth weight
Fetal growth	Grice 2007	Retrospective cohort	Occupational (Decatur, Alabama)	Newborns	263	PFOS	None	PFOS and birth weight
Fetal growth	Hamm 2010	Prospective cohort	General community (Edmonton, Alberta, Canada)	Newborns	252	PFHxS PFOA PFOS	None	PFHxS and birth weight PFHxS and birth weight z-score PFHxS and small for gestational age PFHxS and gestational age PFHxS and preterm delivery PFOA and birth weight PFOA and birth weight z-score PFOA and small for gestational age PFOA and gestational age PFOA and preterm delivery PFOS and birth weight PFOS and birth weight z-score PFOS and small for gestational age PFOS and gestational age PFOS and preterm delivery
Fetal growth	Inoue 2004	Prospective cohort and cross-sectional	General community (Hokkaido, Japan)	Newborns	15	PFOS	None	PFOS and birth weight

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Kim 2011a	Prospective cohort and cross-sectional	General community (Seoul, Cheongju, and Gumi, South Korea)	Newborns	43	PFHxS PFOA PFOS PFtrDA ΣPFAS (PFHxS+PFOA+PFOS+PFtrDA)	None	PFHxS and birth weight PFOA and birth weight PFOS and birth weight PFtrDA and birth weight ΣPFAS and birth weight
Fetal growth	Kim 2011b	Prospective cohort and cross-sectional	General community (Seoul, South Korea)	Newborns	20	PFHxS PFOA PFOS PFNA PFDA PFunDA	None	PFHxS and birth weight PFOA and birth weight PFOS and birth weight PFNA and birth weight PFDA and birth weight PFunDA and birth weight
Fetal growth	Kishi 2013	Prospective cohort	General community (Sapporo and Hokkaido, Japan)	Newborns	514 Sapporo 2,095 Hokkaido	PFHxA PFHxS PFHpA PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA PFteDA	PFOS and lower birth weight (Sapporo) PFNA and lower birth weight (Hokkaido: total, males) PFunDA and lower birth weight (Hokkaido: females) PFtrDA and lower birth weight (Hokkaido: females)	PFHxA and birth weight (Hokkaido) PFHxS and birth weight (Hokkaido) PFHpA and birth weight (Hokkaido) PFOA and birth weight (Sapporo, Hokkaido) PFOS and birth weight (Hokkaido) PFNA and birth weight (Hokkaido: females) PFDA and birth weight (Hokkaido) PFunDA and birth weight (Hokkaido: total, males) PFdoDA and birth weight (Hokkaido) PFtrDA and birth weight (Hokkaido: total, males) PFteDA and birth weight (Hokkaido)
Fetal growth	Kishi 2015	Prospective cohort	General community (Hokkaido, Japan)	Newborns	306	PFOA PFOS	PFOS and lower birth weight (females)	PFOA and birth weight PFOA and birth length PFOA and chest circumference PFOA and head circumference PFOS and birth weight (males) PFOS and birth length PFOS and chest circumference PFOS and head circumference

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Kobayashi 2017	Prospective cohort	General community (Hokkaido, Japan)	Newborns	177	PFOA PFOS	PFOS and lower ponderal index	PFOA and birth weight PFOA and birth length PFOA and ponderal index PFOS and birth weight PFOS and birth length
Fetal growth	Kwon 2016	Cross-sectional	General community (Seoul, South Korea)	Newborns	268	PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA	Interaction of *GSTM1* null and PFHxS on birth weight PFOA and lower birth weight PFOS and lower birth weight PFNA and lower birth weight Interaction of *GSTM1* null and PFNA on birth weight PFDA and lower birth weight Interaction of *GSTM1* null and PFDA on birth weight PFunDA and lower birth weight Interaction of *CYP1A1* Ile/Ile and PFunDA on birth weight Interaction of *GSTM1* null and PFdoDA on birth weight Interaction of *CYP1A1* Ile/Ile and PFdoDA on birth weight Interaction of *CYP1A1* Ile/Ile and PFtrDA on birth weight	PFHxS and birth weight Interaction of *CYP1A1* Ile/Ile and PFHxS on birth weight Interaction of *GSTM1* null and PFOA on birth weight Interaction of *CYP1A1* Ile/Ile and PFOA on birth weight Interaction of *GSTM1* null and PFOS on birth weight Interaction of *CYP1A1* Ile/Ile and PFOS on birth weight Interaction of *CYP1A1* Ile/Ile and PFNA on birth weight Interaction of *CYP1A1* Ile/Ile and PFDA on birth weight Interaction of *GSTM1* null and PFuDA on birth weight PFdoDA and birth weight PFtrDA and birth weight Interaction of *GSTM1* null and PFtrDA on birth weight
Fetal growth	Lauritzen 2017	Prospective case-cohort	General community (Trondheim and Bergen, Norway, and Uppsala, Sweden)	Newborns	424	PFOA PFOS	PFOA and lower birth weight (Sweden: total, boys) PFOA and shorter birth length (Sweden: total, boys) PFOA and greater risk of small for gestational age (Sweden: total, boys) PFOS and lower birth weight (Sweden) PFOS and shorter birth length (Sweden)	PFOA and birth weight (total, Norway, Sweden: girls) Interaction between sex and PFOA on birth weight (Sweden) PFOA and birth length (total, Norway, Sweden: girls) PFOA and head circumference (total, Norway, Sweden) PFOA and gestational age (total, Norway, Sweden) PFOA and small for gestational age (total, Norway, Sweden: girls) PFOS and birth weight (total, Norway) PFOS and birth length (total, Norway) PFOS and head circumference (total, Norway, Sweden) PFOS and gestational age (total, Norway, Sweden) PFOS and small for gestational age (total, Norway, Sweden)

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Lee 2013	Prospective cohort and cross-sectional	General community (Gyeongbuk County, South Korea)	Newborns	59	PFHxS PFOA PFOS	Maternal PFOS and lower risk of ponderal index above median	Maternal or cord blood PFHxS and birth weight, birth length, ponderal index, or head circumference Maternal or cord blood PFOA and birth weight, birth length, ponderal index, or head circumference Maternal or cord blood PFOS and birth weight, birth length, or head circumference
Fetal growth	Lien 2013	Cross-sectional	General community (northern Taiwan)	Newborns	439	PFOA PFOS PFNA PFunDA	PFOS and lower birth weight	PFOA and birth weight PFOA and preterm delivery PFOS and preterm delivery PFNA and birth weight PFNA and preterm delivery PFunDA and birth weight PFunDA and preterm delivery
Fetal growth	Lind 2017a	Prospective cohort	General community (Odense, Denmark)	Infants	649	PFHxS PFOA PFOS PFNA PFDA	PFNA×sex interaction and birth weight PFDA×sex interaction and birth weight	PFHxS and birth weight, gestational length, head circumference, or abdominal circumference (boys, girls) PFOA and birth weight, gestational length, head circumference, or abdominal circumference (boys, girls) PFOS and birth weight, gestational length, head circumference, or abdominal circumference (boys, girls) PFNA and birth weight, gestational length, head circumference, or abdominal circumference (boys, girls) PFDA and birth weight, gestational length, head circumference, or abdominal circumference (boys, girls)
Fetal growth	Maisonet 2012	Prospective cohort	General community (Avon, United Kingdom)	Newborn girls	444	PFHxS PFOA PFOS	PFHxS and lower birth weight PFHxS and lower birth length PFOA and lower birth weight PFOS and lower birth weight	PFHxS and gestational age PFHxS and ponderal index PFOA and birth length PFOA and gestational age PFOA and ponderal index PFOS and birth length PFOS and gestational age PFOS and ponderal index

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Minatoya 2017	Prospective cohort	General community (Hokkaido, Japan)	Newborns	168	PFOA PFOS	PFOA and lower birth weight PFOA and lower ponderal index PFOS and lower ponderal index	Interaction of PFOA × sex and birth weight and ponderal index PFOS and birth weight Interaction of PFOS × sex and birth weight and ponderal index
Fetal growth	Monroy 2008	Prospective cohort and cross-sectional	General community (Hamilton, Ontario, Canada)	Newborns	105	PFHxS PFOA PFOS PFNA	None	PFHxS and birth weight PFOA and birth weight PFOS and birth weight PFOS and gestational length PFNA and birth weight
Fetal growth	Morken 2014 (reanalysis of Whitworth 2012)	Prospective cohort	General community (Norway)	Newborns	953	PFOA	None	PFOA and birth weight z-score (attenuated by 66% after adjustment for eGFR)
Fetal growth	Nolan 2009	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Newborns	1,555	PFOA	PFOA water district and lower risk of low birth weight (partial water service, not exclusive water service from Little Hocking)	PFOA water district and birth weight PFOA water district and gestational age PFOA water district and premature birth

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Robledo 2015	Prospective cohort	General community (Michigan and Texas)	Newborns	234	PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	PFOSA and lower birth weight (maternal serum in boys) Et-PFOSA-AcOH and lower ponderal index (maternal serum in girls)	PFOA and birth weight, birth length, head circumference, and ponderal index PFOS and birth weight, birth length, head circumference, and ponderal index PFOSA and birth weight (maternal serum in girls; paternal serum in boys and girls), birth length, head circumference, and ponderal index Et-PFOSA-AcOH and birth weight, birth length, head circumference, and ponderal index (maternal serum in boys; paternal serum in boys and girls) Me-PFOSA-AcOH and birth weight, birth length, head circumference, and ponderal index PFNA and birth weight, birth length, head circumference, and ponderal index PFDA and birth weight, birth length, head circumference, and ponderal index
Fetal growth	Savitz 2012a	Retrospective case-control and cross-sectional	Contaminated community (Mid-Ohio Valley)	Newborns	8,353 (Study I) 4,547 (Study II)	PFOA	Study II: PFOA and greater risk of preterm birth (continuous (some models), not quintiles) PFOA and greater risk of term low birth weight (continuous (some models), quintile 4, not 5 (some models)) PFOA and greater risk of term small for gestational age (continuous (some models), not quintiles) PFOA and lower term birth weight (continuous (some models), not quintiles)	Study I: PFOA and preterm birth PFOA and term low birth weight PFOA and term small for gestational age PFOA and change in term birth weight Study II PFOA and preterm birth (some models) PFOA and term low birth weight (some models) PFOA and term small for gestational age (some models) PFOA and change in term birth weight (some models)
Fetal growth	Savitz 2012b	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Newborns	11,737	PFOA	None	PFOA and preterm birth PFOA and term low birthweight

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Shi 2017	Cross-sectional	General community (Beijing, China)	Newborns	170	PFHxS PFOA PFOS PFNA PFDA PFunDA	PFHxS and greater birth length (boys) PFunDA and shorter birth length (boys)	PFHxS and birth weight (total, boys, girls), birth length (total, girls), and ponderal index (total, boys, girls) PFOA and birth weight, birth length, and ponderal index (all strata) PFOS and birth weight, birth length, and ponderal index (all strata) PFNA and birth weight, birth length, and ponderal index (all strata) PFDA and birth weight, birth length, and ponderal index (all strata) PFunDA and birth weight (total, boys, girls), birth length (total, girls), and ponderal index (total, boys, girls)
Fetal growth	Starling 2017	Prospective cohort	General community (Colorado)	Newborns	628	PFHxS PFOA PFOS PFNA PFDA	PFHxS and lower birth adiposity PFHxS and lower fat mass PFOA and lower birth weight PFOA and lower birth adiposity PFOA and lower fat mass PFNA and lower birth weight PFNA and lower birth adiposity PFNA and lower fat mass	PFHxS and birth weight PFHxS and fat-free mass PFOA and fat-free mass PFOS and birth weight PFOS and birth adiposity PFOS and fat mass PFOS and fat-free mass PFNA and fat-free mass PFDA and birth weight PFDA and birth adiposity PFDA and fat mass PFDA and fat-free mass
Fetal growth	Stein 2009	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Newborns	1,845 PFOA 5,262 PFOS	PFOA PFOS	PFOA and lower risk of low birth weight (75th-90th percentile, not > 90th) PFOS and greater risk of preterm birth PFOS and greater risk of low birth weight	PFOA and preterm birth

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Valvi 2017	Prospective cohort	Fishing community (Faroe Islands)	Newborns	604	PFHxS PFOA PFOS PFNA PFDA ΣPFAS (PFHxS+PFOA+PFOS+PFNA+PFDA)	PFHxS and larger head circumference (boys) Interaction of PFHxS × sex on head circumference PFOS and lower birth weight (boys) PFOS and larger head circumference (girls) Interaction of PFOS × sex on head circumference Interaction of PFOA × sex on birth weight PFDA and larger head circumference (girls) Interaction of PFDA × sex on head circumference	PFHxS and birth weight, birth length, head circumference (total, girls) PFOA and birth weight, birth length, head circumference PFOS and birth weight (total, girls), birth length, head circumference (total, boys) PFNA and birth weight, birth length, head circumference PFDA and birth weight, birth length, head circumference (total, boys) ΣPFAS and birth weight, birth length, head circumference
Fetal growth	Wang 2016	Prospective cohort	General community (Taiwan)	Newborns	223	PFOA PFNA PFDA PFunDA PFdoDA	PFNA and lower birth weight (girls) PFNA and smaller head circumference (girls quartiles 2 and 3, not 4, not continuous) PFDA and lower birth weight (girls) PFDA and greater risk of small for gestational age (girls) PFDA and lower weight z-score at birth (girls) PFDA and lower length z-score at birth (girls) PFunDA and lower birth weight (girls) PFunDA and greater risk of small for gestational age (girls) PFunDA and lower weight z-score at birth (girls) PFunDA and lower length z-score at birth (girls) PFdoDA and lower birth weight (girls) PFdoDA and smaller head circumference (girls) PFdoDA and lower weight z-score at birth (girls) PFdoDA and lower length z-score at birth (girls)	PFOA and birth weight, birth length, head circumference, and small for gestational age (girls, boys) PFOA and weight z-score at birth (girls, boys) PFOA and length z-score at birth (girls, boys) PFNA and birth length and small for gestational age (girls, boys) PFNA and birth weight and head circumference (boys) PFNA and weight z-score at birth (girls, boys) PFNA and length z-score at birth (girls, boys) PFDA and birth weight, birth length, and head circumference (girls, boys) PFDA and small for gestational age (boys) PFDA and weight z-score at birth (boys) PFDA and length z-score at birth (boys) PFunDA and birth weight, birth length, and head circumference (girls, boys) PFunDA and small for gestational age (boys) PFunDA and weight z-score at birth (boys) PFunDA and length z-score at birth (boys) PFdoDA and birth weight, birth length, and small for gestational age (girls, boys) PFdoDA and head circumference (boys) PFdoDA and weight z-score at birth (boys) PFdoDA and length z-score at birth (boys)

Table 5. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and fetal growth

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Fetal growth	Washino 2009	Prospective cohort	General community (Sapporo, Japan)	Newborns	428	PFOA PFOS	PFOS and lower birth weight (total, girls)	PFOA and birth weight PFOA and birth length PFOA and chest circumference PFOA and head circumference PFOS and birth weight (boys) PFOS and birth length PFOS and chest circumference PFOS and head circumference
Fetal growth	Whitworth 2012a	Prospective cohort	General community (Norway)	Newborns	901	PFOA PFOS	PFOA and lower risk of preterm birth PFOS and lower risk of preterm birth	PFOA and birth weight z-score PFOA and small for gestational age PFOA and large for gestational age PFOS and birth weight z-score PFOS and small for gestational age PFOS and large for gestational age
Fetal growth	Wu 2012	Cross-sectional	Contaminated community (Guiyu Town, Shantou City, Guangdong Province, China)	Newborns	167	PFOA	PFOA and greater risk of premature delivery PFOA and greater risk of term low birth weight PFOA and lower gestational age PFOA and lower birth weight PFOA and lower birth length PFOA and lower 5-minute Apgar score	PFOA and ponderal index

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Anderson-Mahoney 2008	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	566	PFOA	Residence in PFOA water district and greater risk of asthma	None
Immune	Ashley-Martin 2015	Prospective cohort	General community (Canada)	Newborns	1,242	PFHxS PFOA PFOS	None	PFHxS and cord blood interleukin 33/thymic stromal lymphopoietin PFHxS and cord blood IgE PFOA and cord blood interleukin 33/thymic stromal lymphopoietin PFOA and cord blood IgE PFOS and cord blood interleukin 33/thymic stromal lymphopoietin PFOS and cord blood IgE
Immune	Buser 2016	Cross-sectional	General community (United States)	Adolescents	637	PFHxS PFOA PFOS PFNA	PFHxS and greater risk of self-reported food allergy PFOA and greater risk of self-reported food allergy PFOA and greater risk of self-reported peanut/tree nut/shellfish allergy PFOS and greater risk of self-reported food allergy PFOS and lower risk of sensitization to shrimp (IgE level ≥ 0.35 kU/L) PFNA and lower risk of food sensitization (≥ 1 food-specific IgE level ≥ 0.35 kU/L) PFNA and lower risk of sensitization to peanuts (quartile 3, not 4) and shrimp (IgE level ≥ 0.35 kU/L)	PFHxS and food sensitization (≥ 1 food-specific IgE level ≥ 0.35 kU/L) PFHxS and sensitization to milk, peanuts, or shrimp (IgE level ≥ 0.35 kU/L) PFHxS and self-reported peanut/tree nut/shellfish allergy PFOA and food sensitization (≥ 1 food-specific IgE level ≥ 0.35 kU/L) PFOA and sensitization to milk, peanuts, or shrimp (IgE level ≥ 0.35 kU/L) PFOS and food sensitization (≥ 1 food-specific IgE level ≥ 0.35 kU/L) PFOS and self-reported peanut/tree nut/shellfish allergy PFOS and sensitization to milk or peanuts (IgE level ≥ 0.35 kU/L) PFNA and self-reported food allergy PFNA and self-reported peanut/tree nut/shellfish allergy PFNA and sensitization to milk (IgE level ≥ 0.35 kU/L)
Immune	Conway 2016	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Children, adolescents, adults	66,899	PFHxS PFOA PFOS PFNA	PFHxS and lower risk of type 1 diabetes (all) PFOA and lower risk of type 1 diabetes (all, children < 20 years, adults) PFOS and lower risk of type 1 diabetes (all) PFNA and lower risk of type 1 diabetes (all)	None
Immune	Costa 2009	Cross-sectional	Occupational (Trissino, Italy)	Men	53	PFOA	PFOA and greater alpha-2 globulin level (current/formerly/never exposed workers)	PFOA and alpha-1 globulin level PFOA and alpha-2 globulin level (exposed vs. non-exposed or other workers) PFOA and beta-globulin level PFOA and gamma-globulin level PFOA and white blood cell count PFOA and red blood cell count PFOA and hemoglobin level PFOA and hematocrit level PFOA and platelet level PFOA and IgG level PFOA and IgM level PFOA and C-reactive protein level

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Dalsager 2016	Prospective cohort	General community (Odense, Denmark)	Infants, young children	346	PFHxS PFOA PFOS PFNA PFDA	PFOA and greater proportion of days with fever at 1-4 years PFOA and greater number of episodes of co-occurrence of fever and nasal discharge at 1-4 years (medium, not high PFOA) PFOS and greater number and proportion of days with fever at 1-4 years PFNA and lower proportion of days with nasal discharge at 1-4 years PFNA and lower number of days with diarrhea at 1-4 years (medium, not high PFNA) PFDA and lower proportion of days with nasal discharge at 1-4 years (medium, not high PFDA)	PFHxS and proportion or number of days with fever, cough, nasal discharge, diarrhea, or vomiting at 1-4 years PFHxS and number of episodes of co-occurrence of fever and coughing or fever and nasal discharge at 1-4 years PFOA and number of days with fever at 1-4 years PFOA and proportion or number of days with cough, nasal discharge, diarrhea, or vomiting at 1-4 years PFOA and number of episodes of co-occurrence of fever and coughing at 1-4 years PFOS and proportion or number of days with cough, nasal discharge, diarrhea, or vomiting at 1-4 years PFOS and number of episodes of co-occurrence of fever and coughing or fever and nasal discharge at 1-4 years PFNA and proportion of days with fever, cough, diarrhea, or vomiting at 1-4 years PFNA and number of days with fever, cough, nasal discharge, or vomiting at 1-4 years PFNA and number of episodes of co-occurrence of fever and coughing or fever and nasal discharge at 1-4 years PFDA and proportion or number of days with fever, cough, diarrhea, or vomiting at 1-4 years PFDA and number of days with nasal discharge at 1-4 years PFDA and number of episodes of co-occurrence of fever and coughing or fever and nasal discharge at 1-4 years

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Dong 2013	Case-control	General community (northern Taiwan)	Children, adolescents	231 cases, 225 controls	PFBS PFHxA PFHxS PFHpA PFOA PFOS PFNA PFDA PFdoDA PFteDA	PFBS and greater risk of asthma, greater levels of absolute eosinophil counts (asthmatic) at 10-15 years PFHxS and greater risk of asthma, greater levels of absolute eosinophil counts (asthmatic), and eosinophilic cationic protein (asthmatic) at 10-15 years PFHpA and greater risk of asthma (unadjusted test) at 10-15 years PFOA and greater risk of asthma, greater levels of IgE (asthmatic), absolute eosinophil counts (asthmatic), and eosinophilic cationic protein (asthmatic) at 10-15 years PFOS and greater risk of asthma and asthma severity, greater levels of IgE (asthmatic), absolute eosinophil counts (asthmatic), and eosinophilic cationic protein (asthmatic) at 10-15 years PFNA and greater risk of asthma, greater levels of IgE (asthmatic), absolute eosinophil counts (asthmatic), and eosinophilic cationic protein (asthmatic) at 10-15 years PFDA and greater risk of asthma and asthma severity, greater levels of IgE (asthmatic), absolute eosinophil counts (asthmatic), and eosinophilic cationic protein (asthmatic, non-asthmatic) at 10-15 years PFdoDA and greater risk of asthma and asthma severity, greater levels of IgE (asthmatic), absolute eosinophil counts (asthmatic), and eosinophilic cationic protein (asthmatic, non-asthmatic) at 10-15 years PFteDA and greater risk of asthma (unadjusted test) and asthma severity, greater levels of IgE (asthmatic) and absolute eosinophil counts (asthmatic) at 10-15-years	PFBS and asthma severity, asthma control test, IgE (asthmatic, non-asthmatic), absolute eosinophil counts (non-asthmatic), and eosinophilic cationic protein level (asthmatic, non-asthmatic) at 10-15 years PFHxA and asthma, asthma severity, asthma control test, IgE (asthmatic, non-asthmatic), absolute eosinophil counts (asthmatic, non-asthmatic), and eosinophilic cationic protein level (asthmatic, non-asthmatic) at 10-15 years PFHxS and asthma severity, asthma control test, IgE (asthmatic, non-asthmatic), absolute eosinophil counts (non-asthmatic), and eosinophilic cationic protein level (non-asthmatic) at 10-15 years PFOA and asthma severity, asthma control test, IgE (non-asthmatic), absolute eosinophil counts (non-asthmatic), and eosinophilic cationic protein level (non-asthmatic) at 10-15 years PFOS and asthma control test, IgE (non-asthmatic), absolute eosinophil counts (non-asthmatic), and eosinophilic cationic protein level (non-asthmatic) at 10-15 years PFNA and asthma severity, asthma control test, IgE (non-asthmatic), absolute eosinophil counts (non-asthmatic), and eosinophilic cationic protein level (non-asthmatic) at 10-15 years PFDA and asthma control test, IgE (non-asthmatic) and absolute eosinophil count (non-asthmatic) at 10-15 years PFdoDA and asthma control test, IgE (non-asthmatic) and absolute eosinophil counts (non-asthmatic) at 10-15 years PFteDA and asthma control test, IgE (non-asthmatic), absolute eosinophil counts (non-asthmatic), and eosinophilic cationic protein level (asthmatic, non-asthmatic) at 10-15 years
Immune	Emmett 2006	Cross-sectional	Contaminated community (Mid-Ohio Valley)	All (infants, children, adolescents, adults)	371	PFOA	PFOA and greater absolute monocyte counts PFOA and lower risk of abnormal percent neutrophils PFOA and lower risk of abnormal percent lymphocytes	PFOA and white blood cell count PFOA and red blood cell count PFOA and hemoglobin PFOA and hematocrit PFOA and mean corpuscular volume PFOA and mean corpuscular hemoglobin PFOA and mean corpuscular hemoglobin volume PFOA and red cell distribution width PFOA and platelets PFOA and neutrophils, %, absolute, and abnormal count PFOA and lymphocytes, %, absolute, and abnormal count PFOA and monocytes, % and abnormal %/count PFOA and eosinophils, %, absolute, and abnormal %/count PFOA and basophils, %, absolute, and abnormal %/count
Immune	Fei 2010a	Prospective cohort	General community (Denmark)	Infants, young children	1,400	PFOA PFOS	PFOA and lower risk of hospitalization for infectious diseases (0–10 y (quartile 2 only), 0–<1 y (quartile 2 only), 1–<2 y (quartile 2 only), and 2–<4 y (quartile 3 only); boys; multiparous mothers) PFOA and greater risk of hospitalization for infectious diseases (girls) PFOS and lower risk of hospitalization for infectious diseases (0-<1 y; boys (quartile 3 only)) PFOS and greater risk of hospitalization for infectious diseases (≥4 y (quartile 2 only); girls)	PFOA and hospitalization for infectious diseases (≥4 y; primiparous mothers) PFOS and hospitalization for infectious diseases (0–10 y, 1–<2 y, 2–<4 y; primiparous and multiparous mothers)

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Genser 2015	Cross-sectional	Contaminated community (Mid-Ohio Valley)	All (infants, children, adolescents, adults)	25,817	PFOA	PFOA and lower C-reactive protein level	PFOA and lymphocyte count
Immune	Goudarzi 2016a	Prospective cohort	General community (Hokkaido, Japan)	Children	1,558	PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA	PFHxS and lower risk of wheezing (total, boys in quartile 3, not 4) at 4 years PFOA and lower risk of eczema (boys) at age 4 years PFOS and lower risk of total allergic diseases (total in quartile 2, not 3 or 4), eczema (total and boys in quartiles 2 and 3, not 4), rhinoconjunctivitis (total in quartile 2, not 3 or 4) at 4 years PFNA and lower risk of rhinoconjunctivitis (total, girls) at 4 years PFunDA and lower risk of eczema (girls in quartile 2, not 3 or 4), rhinoconjunctivitis (total, boys) at 4 years PFdoDA and lower risk of total allergic diseases (total, boys, girls in quartile 2, not 3 or 4), eczema (total, boys), rhinoconjunctivitis (boys) at 4 years PFtrDA and lower risk of total allergic diseases (total, boys), eczema (total, girls quartile 2, not 3 or 4) at 4 years PFtrDA and greater risk of rhinoconjunctivitis (total and girls in quartile 2, not 3 or 4) at 4 years	PFHxS and total allergic diseases, wheezing (girls), eczema, rhinoconjunctivitis at 4 years PFOA and total allergic diseases, wheezing, eczema (total, girls), rhinoconjunctivitis at 4 years PFOS and wheezing, eczema (girls), rhinoconjunctivitis (boys, girls) at 4 years PFNA and total allergic diseases, wheezing, eczema, rhinoconjunctivitis (boys) at 4 years PFDA and total allergic diseases, wheezing, eczema, rhinoconjunctivitis at 4 years PFunDA and total allergic diseases, wheezing, eczema (total, boys), rhinoconjunctivitis (girls) at 4 years PFdoDA and wheezing, eczema (girls), rhinoconjunctivitis (total, girls) at 4 years PFtrDA and total allergic diseases, wheezing, eczema (boys), rhinoconjunctivitis (boys) at 4 years
Immune	Goudarzi 2017b	Prospective cohort	General community (Hokkaido, Japan)	Children	1,558	PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA	PFHxS and greater risk of total infectious diseases at 4 years (girls) PFOS and greater risk of total infectious diseases at 4 years (total, boys quartile 4 no trend, girls)	PFHxS and total infectious diseases at 4 years (total, boys) PFOA and total infectious diseases at 4 years PFNA and total infectious diseases at 4 years PFDA and total infectious diseases at 4 years PFunDA and total infectious diseases at 4 years PFdoDA and total infectious diseases at 4 years PFtrDA and total infectious diseases at 4 years

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Grandjean 2012	Prospective cohort and cross-sectional	Fishing community (Faroe Islands)	Children	587	PFHxS PFOA PFOS PFNA PFDA ΣPFAS (PFHxS+PFOA+PFOS+PFNA+PFDA)	Maternal PFHxS and greater anti-tetanus antibody level at 7 y (adj. for 5 y) PFHxS at 5 y and lower anti-tetanus antibody level at 5 and 7 y (adj./not adj. for 5 y) PFHxS at 5 y and lower anti-diphtheria antibody level at 7 y (adj. for 5 y) Maternal PFOA and lower anti-diphtheria antibody level at 7 y (not adj. for 5 y) PFOA at 5 y and lower anti-tetanus antibody level at 7 y (adj./not adj. for 5 y) PFOA at 5 y and lower anti-diphtheria antibody level at 7 y (adj./not adj. for 5 y) Maternal PFOS and greater anti-tetanus antibody level at 7 y (adj. for 5 y) Maternal PFOS and lower anti-diphtheria antibody level at 5 y PFOS at 5 y and lower anti-tetanus antibody level at 5 y PFOS at 5 y and lower anti-diphtheria antibody level at 7 y (not adj. for 5 y) PFNA at 5 y and lower anti-diphtheria antibody level at 5 y Maternal PFDA and lower anti-diphtheria antibody level at 5 y PFDA at 5 y and lower anti-tetanus antibody level at 5 and 7 y (not adj. for 5 y) Maternal ΣPFAS and lower anti-diphtheria antibody level at 5 and 7 y ΣPFAS at 5 y and lower anti-tetanus antibody level at 7 y ΣPFAS at 5 y and lower anti-diphtheria antibody level at 7 y ΣPFAS at 5 y and lower anti-tetanus/anti-diphtheria antibody level at 7 y	Maternal PFHxS and anti-tetanus antibody level at 5 y Maternal PFHxS and anti-diphtheria antibody level at 5 or 7 y PFHxS at 5 y and anti-diphtheria antibody level at 5 y Maternal PFOA and anti-tetanus antibody level at 5 or 7 y Maternal PFOA and anti-diphtheria antibody level at 5 y PFOA at 5 y and anti-tetanus antibody level at 5 y PFOA at 5 y and anti-diphtheria antibody level at 5 y Maternal PFOS and anti-tetanus antibody level at 5 y Maternal PFOS and anti-diphtheria antibody level at 7 y PFOS at 5 y and anti-tetanus antibody level at 7 y PFOS at 5 y and anti-diphtheria antibody level at 5 y Maternal PFNA and anti-tetanus antibody level at 5 or 7 y Maternal PFNA and anti-diphtheria antibody level at 5 or 7 y PFNA at 5 y and anti-tetanus antibody level at 5 or 7 y PFNA at 5 y and anti-diphtheria antibody level at 7 y Maternal PFDA and anti-tetanus antibody level at 5 or 7 y Maternal PFDA and anti-diphtheria antibody level at 7 y PFDA at 5 y and anti-diphtheria antibody level at 5 or 7 y Maternal ΣPFAS and anti-tetanus antibody level at 5 or 7 y Maternal ΣPFAS and anti-tetanus/anti-diphtheria level at 5 y ΣPFAS at 5 y and anti-tetanus antibody level at 7 y ΣPFAS at 5 y and anti-diphtheria antibody level at 5 y ΣPFAS at 5 y and anti-tetanus/anti-diphtheria level at 5 y

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Grandjean 2017	Prospective cohort and cross-sectional	Fishing community (Faroe Islands)	Children, adolescents	505	PFHxS PFOA PFOS PFNA PFDA	PFHxS at 7 y and lower anti-diphtheria antibody level at 7 and 13 y (no ER visit/booster, total and indirect effects) PFHxS at 7 y and greater anti-tetanus antibody level at 7 and 13 y (total, indirect effect (via 7-year antibody)) PFOA at 13 y and lower anti-diphtheria antibody level at 13 y (no ER visit/booster) PFOA at 7 y and lower anti-diphtheria antibody level at 7 and 13 y (total, no ER visit/booster, indirect effect (via 7-year antibody)) PFOS at 7 y and greater anti-tetanus antibody level at 13 y (no ER visit/booster) PFOS at 7 y and lower anti-diphtheria antibody level at 7 and 13 y (total, no ER visit/booster, total and indirect effects) PFNA at 7 y and lower anti-diphtheria antibody level at 7 and 13 y (indirect effect (via 7-year antibody)) PFDA at 7 y and lower anti-diphtheria antibody level at 13 y PFDA at 7 y and greater anti-tetanus antibody level at 13 y (total) PFDA at 7 y and lower anti-diphtheria antibody level at 7 and 13 y (total and indirect effects)	PFHxS at 7 or 13 y and anti-diphtheria antibody level at 13 y PFHxS at 7 or 13 y and anti-tetanus antibody level at 13 y PFHxS at 7 y and anti-diphtheria antibody level at 7 and 13 y (total, no ER visit/booster and no antibody increase) PFHxS at 7 y and anti-tetanus antibody level at 7 and 13 y (no ER visit/booster +/- no antibody increase) PFOA at 7 y and anti-diphtheria antibody level at 13 y PFOA at 13 y and anti-diphtheria antibody level at 13 y (total, no ER visit/booster and no antibody increase) PFOA at 7 or 13 y and anti-tetanus antibody level at 13 y PFOA at 7 y and anti-diphtheria antibody level at 7 and 13 y (no ER visit/booster and no antibody increase) PFOA at 7 y and anti-tetanus antibody level at 7 and 13 y PFOS at 7 or 13 y and anti-diphtheria antibody level at 13 y PFOS at 7 y and anti-tetanus antibody level at 13 y (total, no ER visit/booster and no antibody increase) PFOS at 13 y and anti-tetanus antibody level at 13 y PFOS at 7 y and anti-diphtheria antibody level at 7 and 13 y (no ER visit/booster and no antibody increase) PFOS at 7 y and anti-tetanus antibody level at 7 and 13 y PFNA at 7 or 13 y and anti-diphtheria antibody level at 13 y PFNA at 7 or 13 y and anti-tetanus antibody level at 13 y PFNA at 7 y and anti-tetanus antibody level at 7 and 13 y PFDA at 13 y and anti-diphtheria antibody level at 13 y PFDA at 7 y and anti-tetanus antibody level at 13 y (no ER visit/booster -/+ no antibody increase) PFDA at 13 y and anti-tetanus antibody level at 13 y PFDA at 7 y and anti-tetanus antibody level at 7 and 13 y
Immune	Granum 2013	Prospective cohort	General community (Norway)	Infants, young children	93	PFHxS PFOA PFOS PFNA	PFHxS and lower anti-rubella antibody levels at 0-3 years PFHxS and greater number of episodes of gastroenteritis at 0-3 years PFOA and lower anti-rubella antibody levels at 0-3 years PFOA and greater number of episodes of common cold at 0-3 and 3 years PFOA and greater number of episodes of gastroenteritis at 0-3 years PFOS and lower anti-rubella antibody levels at 0-3 years PFNA and lower anti-rubella antibody levels at 0-3 years PFNA and greater number of episodes of common cold at 0-3 and 3 years	PFHxS and anti-measles antibody levels, anti-*H. influenzae* type B antibody levels, anti-tetanus antibody levels, number of episodes of common cold, ever common cold, ever gastroenteritis at 0-3 years (and 3 years exc. antibodies) PFOA and anti-measles antibody levels, anti-*H. influenzae* type B antibody levels, anti-tetanus antibody levels, ever common cold, ever gastroenteritis at 0-3 years (and 3 years exc. antibodies) PFOS and anti-measles antibody levels, anti-*H. influenzae* type B antibody levels, anti-tetanus antibody levels, number of episodes of common cold, ever common cold, number of episodes of gastroenteritis, ever gastroenteritis at 0-3 years (and 3 years exc. antibodies) PFNA and anti-measles antibody levels, anti-*H. influenzae* type B antibody levels, anti-tetanus antibody levels, ever common cold, number of episodes of gastroenteritis, ever gastroenteritis at 0-3 years (and 3 years exc. antibodies)

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Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Humblet 2014	Cross-sectional	General community (United States)	Adolescents	1,877	PFHxS PFOA PFOS PFNA	PFOA and greater risk of ever asthma at 12-19 years	PFHxS and ever asthma, wheeze, or current asthma at 12-19 years PFOA and wheeze or current asthma at 12-19 years PFOS and ever asthma, wheeze, or current asthma at 12-19 years PFNA and ever asthma, wheeze, or current asthma at 12-19 years
Immune	Jiang 2014	Cross-sectional	General community (Tianjin, China)	Women	141	PFHxA PFHxS PFHpA ΣPFOA (n-, iso-, 5m-, %n-) ΣPFOS (n-, iso-, 1m-4m- 3+5m-Σm2- %n-) PFNA PFDA PFunDA PFdoDA ΣPFAS (PFHxA+PFHxS+PFHpA+PFOA+PFOS+PFNA+PFDA+PFunDA+PFdoDA)	PFHxS, 1m-PFOS, 4m-PFOS, 3+5m-PFOS and greater white blood cell count %n-PFOS and lower white blood cell count n-PFOS, iso-PFOS, 3+5m-PFOS, ΣPFOS, PFNA and greater red blood cell count iso-PFOA and lower red blood cell count PFHxS, n-PFOA, ΣPFOA, n-PFOS, iso-PFOS, 1m-PFOS, 4m-PFOS, 3+5m-PFOS, Σm2-PFOS, ΣPFOS, PFNA, PFunDA, ΣPFAS and greater hemoglobin level iso-PFOS and greater platelet level PFHpA and lower platelet level	PFHxA, PFHpA, n-PFOA, iso-PFOA, 5m-PFOA, %n-PFOA, ΣPFOA, n-PFOS, iso-PFOS, Σm2-PFOS, ΣPFOS, PFNA, PFDA, PFunDA, PFdoDA, ΣPFAS and white blood cell count PFHxA, PFHxS, PFHpA, n-PFOA, 5m-PFOA, %n-PFOA, ΣPFOA, 1m-PFOS, 4m-PFOS, Σm2-PFOS, %n-PFOS, PFDA, PFunDA, PFdoDA, ΣPFAS and red blood cell count PFHxA, PFHpA, iso-PFOA, 5m-PFOA, %n-PFOA, %n-PFOS, PFDA, PFdoDA and hemoglobin level PFHxA, PFHxS, n-PFOA, iso-PFOA, 5m-PFOA, %n-PFOA, ΣPFOA, n-PFOS, 1m-PFOS, 4m-PFOS, 3+5m-PFOS, Σm2-PFOS, %n-PFOS, ΣPFOS, PFNA, PFDA, PFunDA, PFdoDA, ΣPFAS and platelet level
Immune	Kielsen 2016	Cross-sectional	General community (Copenhagen, Denmark)	Adults	12	PFHxS PFHpA PFOA PFOS PFNA PFDA PFunDA PFdoDA	PFOS and lower post-vaccination anti-diphtheria antibody increase PFNA and lower post-vaccination anti-diphtheria antibody increase PFDA and lower post-vaccination anti-diphtheria antibody increase PFunDA and lower post-vaccination anti-diphtheria antibody increase PFunDA and lower post-vaccination anti-tetanus antibody increase PFdoDA and lower post-vaccination anti-diphtheria antibody increase PFdoDA and lower post-vaccination anti-tetanus antibody increase	PFHxS and post-vaccination anti-diphtheria antibody level PFHxS and post-vaccination anti-tetanus antibody level PFHpA and post-vaccination anti-diphtheria antibody level PFHpA and post-vaccination anti-tetanus antibody level PFOA and post-vaccination anti-diphtheria antibody level PFOA and post-vaccination anti-tetanus antibody level PFOS and post-vaccination anti-tetanus antibody level PFNA and post-vaccination anti-tetanus antibody level PFDA and post-vaccination anti-tetanus antibody level

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Kishi 2013 (overlap with Okada 2012 and 2014)	Prospective cohort	General community (Sapporo and Hokkaido, Japan)	Newborns, infants	514 Sapporo 2,095 Hokkaido	PFHxA PFHxS PFHpA PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA PFteDA	PFOA and lower cord blood IgE at birth (Sapporo: girls)	PFHxA and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFHxS and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFHpA and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFOA and food allergy, eczema, wheezing, and otitis media at 18 months (Sapporo) PFOA and cord blood IgE at birth (Sapporo: boys) PFOA and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFOS and food allergy, eczema, wheezing, and otitis media at 18 months (Sapporo) PFOS and cord blood IgE at birth (Sapporo) PFOS and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFNA and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFDA and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFunDA and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFdoDA and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFtrDA and food allergy, eczema, and wheezing at 12 months (Hokkaido) PFteDA and food allergy, eczema, and wheezing at 12 months (Hokkaido)
Immune	Leonard 2008	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	6,027	PFOA	PFOA and lower risk of mortality from infectious and parasitic diseases (vs. US)	PFOA and mortality from infectious and parasitic diseases (vs. West Virginia or DuPont Region 1)
Immune	Lin 2011	Cross-sectional	General community (Taiwan)	Adolescents, young adults	287	PFOA PFOS PFNA PFunDA ΣPFAS	None	PFOA and C-reactive protein PFOS and C-reactive protein PFNA and C-reactive protein PFunDA and C-reactive protein ΣPFAS and C-reactive protein
Immune	Looker 2014	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	411	PFOA PFOS	PFOA and lower post-vaccination anti-influenza A/H3N2 antibody increase PFOA and lower odds post-vaccination anti-influenza A/H3N2 seroprotection (titer ≥ 1:40)	PFOA and post-vaccination anti-influenza type B antibody level, seroconversion (4-fold titer increase), or seroprotection (titer ≥ 1:40) PFOA and post-vaccination anti-influenza A/H1N1 antibody level, seroconversion, or seroprotection PFOA and post-vaccination anti-influenza A/H3N2 seroconversion PFOA and self-reported "flu" infection in last 12 months PFOA and self-reported cold in last 12 months PFOA and self-reported cold or "flu" in last 12 months PFOA and number of colds reported in last 12 months PFOS and post-vaccination anti-influenza type B antibody level, seroconversion, or seroprotection PFOS and post-vaccination anti-influenza A/H1N1 antibody level, seroconversion, or seroprotection PFOS and post-vaccination anti-influenza A/H3N2 antibody level, seroconversion, or seroprotection PFOS and self-reported "flu" infection in last 12 months PFOS and self-reported cold in last 12 months PFOS and self-reported cold or "flu" in last 12 months PFOS and number of colds reported in last 12 months

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Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Lundin 2009	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,993	PFOA	None	PFOA and mortality from asthma
Immune	Melzer 2010	Cross-sectional	General community (United States)	Adults	3,974	PFOA PFOS	PFOA and greater risk of asthma ever (quartile 3, not 4)	PFOS and asthma ever
Immune	Mogensen 2015 (reanalysis of Grandjean 2012)	Prospective cohort	Fishing community (Faroe Islands)	Children	459	PFHxS PFOA PFOS ΣPFAS (PFHxS+PFOA+PFOS)	PFHxS at 7 years or 5 and 7 years and lower anti-tetanus antibody levels at 7 years PFOA at 7 years or 5 and 7 years and lower anti-diphtheria antibody levels at 7 years PFOA at 5 and 7 years and lower anti-tetanus antibody levels at 7 years PFOS at 7 years or 5 and 7 years and lower anti-diphtheria antibody levels at 7 years Joint PFAS at 5 years, 7 years, or 5 and 7 years and lower anti-diphtheria antibody levels at 7 years Joint PFAS at 5 years, 7 years, or 5 and 7 years and lower anti-tetanus antibody levels at 7 years Joint PFAS at 7 years or 5 and 7 years and lower joint anti-diphtheria and anti-tetanus antibody levels at 7 years	PFHxS at 7 years or 5 and 7 years and anti-diphtheria antibody levels PFOA at 7 years and anti-tetanus antibody levels PFOS at 7 years or 5 and 7 years and anti-tetanus antibody levels Joint PFAS at 5 years and joint anti-diphtheria and anti-tetanus antibody levels at 7 years
Immune	Okada 2012	Prospective cohort	General community (Sapporo, Japan)	Infants, young children	343	PFOA PFOS	PFOA and lower cord blood IgE at birth (total quadratic, not cubic; girls quadratic and cubic)	PFOA and cord blood IgE at birth (boys) PFOA and food allergy at 18 months PFOA and eczema at 18 months PFOA and wheezing at 18 months PFOA and otitis media at 18 months PFOS and cord blood IgE at birth PFOS and food allergy at 18 months PFOS and eczema at 18 months PFOS and wheezing at 18 months PFOS and otitis media at 18 months

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Okada 2014	Prospective cohort	General community (Hokkaido, Japan)	Infants, young children	2,062	PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA	PFHxS and lower risk of total allergic diseases (boys in quartile 3, not 4, no trend) at 24 months PFHxS and lower risk of eczema (total and boys in quartile 3, not 4, no trend) at 24 months PFOA and lower risk of total allergic diseases at 24 months (total, girls) PFOA and lower risk of eczema at 24 months (total) PFOS and lower risk of eczema (girls in quartile 3, not 4, no trend) at 24 months PFNA and lower risk of total allergic diseases at 24 months (total, girls) PFNA and lower risk of eczema at 24 months (girls in quartile 2, not 3 or 4, no trend) PFDA and lower risk of total allergic diseases at 24 months (girls) PFDA and lower risk of eczema at 24 months (girls in quartile 2, not 3 or 4, no trend) PFunDA and lower risk of total allergic diseases at 24 months (girls) PFunDA and lower risk of eczema at 24 months (girls) PFdoDA and lower risk of total allergic diseases at 24 months (total, girls) PFtrDA and lower risk of total allergic diseases at 24 months (total, girls) PFtrDA and lower risk of eczema at 24 months (total, girls)	PFHxS and total allergic diseases (total, girls), eczema (girls), wheezing at 24 months PFHxS and eczema, wheezing at 12 months PFOA and total allergic diseases (boys), eczema (girls, boys), wheezing at 24 months PFOA and eczema, wheezing at 12 months PFOS and total allergic diseases, eczema (total, boys), wheezing at 24 months PFOS and eczema, wheezing at 12 months PFNA and total allergic diseases (boys), eczema (total, boys), wheezing at 24 months PFNA and eczema, wheezing at 12 months PFDA and total allergic diseases (total, boys), eczema (total, boys), wheezing at 24 months PFDA and eczema, wheezing at 12 months PFunDA and total allergic diseases (total, boys), eczema (total, boys), wheezing at 24 months PFunDA and eczema, wheezing at 12 months PFdoDA and total allergic diseases (boys), eczema, wheezing at 24 months PFdoDA and eczema, wheezing at 12 months PFtrDA and total allergic diseases (boys), eczema (boys), wheezing at 24 months PFtrDA and eczema, wheezing at 12 months
Immune	Osuna 2014	Cross-sectional	Fishing community (Faroe Islands)	Children	38	PFOA PFOS	PFOS and lower levels of IgG autoantibodies against actin at 7 years	PFOA and IgM and IgG autoantibodies against neural (neurofilaments, cholineacetyltransferase, astrocyte glial fibrillary acidic protein, and myelin basic protein) and non-neural (actin, desmin, and keratin) antigens at 7 years PFOS and IgM and IgG autoantibodies against neural (neurofilaments, cholineacetyltransferase, astrocyte glial fibrillary acidic protein, and myelin basic protein) and non-neural (actin (IgM only), desmin, and keratin) antigens at 7 years
Immune	Oulhote 2017	Prospective cohort and cross-sectional	Fishing community (Faroe Islands)	Children	55	PFAS factor (PFHxS, PFOA, PFOS, PFNA, PFDA)	PFAS at 18 months and greater basophils level at 5 years PFAS at 5 years and greater basophils level at 5 years	PFAS at gestation, 18 months, or 5 years and neutrophils level at 5 years PFAS at gestation and basophils level at 5 years PFAS at gestation, 18 months, or 5 years and eosinophils level at 5 years PFAS at gestation, 18 months, or 5 years and lymphocytes level at 5 years PFAS at gestation, 18 months, or 5 years and monocytes level at 5 years PFAS at gestation, 18 months, or 5 years and white blood cells level at 5 years

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Qin 2017	Case-control	General community (northern Taiwan)	Children, adolescents	132 cases, 168 controls	PFBS PFHxA PFHxS PFOA PFOS PFNA PFDA PFteDA	PFHxS and greater risk of asthma at 10-15 years PFHxS and lower FVC, FEV1, and FEF25-75 at 10-15 years (asthmatic) PFOA and greater risk of asthma at 10-15 years PFOA and lower FEV1 and FEF25-75 at 10-15 years (asthmatic) PFOS and greater risk of asthma at 10-15 years PFOS and lower FVC and FEV1 at 10-15 years (asthmatic) PFNA and greater risk of asthma at 10-15 years PFNA and lower FVC and FEV1 at 10-15 years (asthmatic) PFNA and greater PEF at 10-15 years (non-asthmatic) PFteDA and greater risk of asthma at 10-15 years	PFBS and asthma at 10-15 years PFBS and forced vital capacity at 10-15 years (FVC), forced expiratory volume in 1 second at 10-15 years (FEV1), peak expiratory flow rate at 10-15 years (PEF), and forced expiratory flow 25-75% at 10-15 years (FEF25-75) at 10-15 years (asthmatic, non-asthmatic) PFHxA and asthma at 10-15 years PFHxA and FVC, FEV1, PEF, and FEF25-75 at 10-15 years (asthmatic, non-asthmatic) PFHxS and PEF at 10-15 years (asthmatic) PFHxS and FVC, FEV1, PEF, and FEF25-75 at 10-15 years (non-asthmatic) PFOA and FVC and PEF at 10-15 years (asthmatic) PFOA and FVC, FEV1, PEF, and FEF25-75 at 10-15 years (non-asthmatic) PFOS and PEF and FEF25-75 at 10-15 years (asthmatic) PFOS and FVC, FEV1, PEF, and FEF25-75 at 10-15 years (non-asthmatic) PFNA and PEF and FEF25-75 at 10-15 years (asthmatic) PFNA and FVC, FEV1, and FEF25-75 at 10-15 years (non-asthmatic) PFDA and asthma at 10-15 years PFDA and FVC, FEV1, PEF, and FEF25-75 at 10-15 years (asthmatic, non-asthmatic) PFteDA and FVC, FEV1, PEF, and FEF25-75 at 10-15 years (asthmatic, non-asthmatic)
Immune	Shankar 2011a	Cross-sectional	General community (United States)	Adults	4,587	PFOA PFOS	PFOS and lower C-reactive protein	PFOA and C-reactive protein

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Smit 2015	Prospective cohort	Fishing/general community (Greenland and Ukraine)	Children	1,024	PFHxS PFHpA PFOA PFOS PFNA PFDA PFunDA PFdoDA PFAS factor 1 (PFHxS, PFOS, PFNA, PFDA, PFunDA, PFdoDA) PFAS factor 2 (PFHpA, PFOA, PFOS)	PFHpA and lower risk of current wheeze (Ukraine) PFOS and lower risk of current wheeze (Ukraine) PFAS factor 2 and lower risk of current eczema (Ukraine)	PFHxS and ever asthma, ever eczema, ever wheeze, current wheeze, and current eczema PFHpA and ever asthma, ever eczema, ever wheeze, current wheeze (combined, Greenland), and current eczema PFOA and ever asthma, ever eczema, ever wheeze, current wheeze, and current eczema PFOS and ever asthma, ever eczema, ever wheeze, current wheeze (combined, Greenland), and current eczema PFNA and ever asthma, ever eczema, ever wheeze, current wheeze, and current eczema PFDA and ever asthma, ever eczema, ever wheeze, current wheeze, and current eczema PFunDA and ever asthma, ever eczema, ever wheeze, current wheeze, and current eczema PFdoDA and ever asthma, ever eczema, ever wheeze, current wheeze, and current eczema PFAS factor 1 and ever asthma, ever eczema, ever wheeze, current wheeze, and current eczema PFAS factor 2 and ever asthma, ever eczema, ever wheeze, current wheeze, and current eczema (combined, Greenland)
Immune	Steenland 2013	Retrospective cohort	Contaminated community (Mid-Ohio Valley) and occupational (Parkersburg, West Virginia)	Adults	32,254	PFOA	PFOA and greater risk of ulcerative colitis PFOA and greater risk of rheumatoid arthritis (10-year lag quartile 3, not 4, no trend) PFOA and lower risk of type 1 diabetes, broad definition (10 year lag quartile 3, not 4, no trend)	PFOA and Crohn's disease PFOA and rheumatoid arthritis (no lag) PFOA and type 1 diabetes, broad definition (no lag) PFOA and type 1 diabetes, narrow definition PFOA and lupus PFOA and multiple sclerosis
Immune	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	PFOA and greater risk of ulcerative colitis (10-year lag) PFOA and greater risk of rheumatoid arthritis (no lag)	PFOA and ulcerative colitis (no lag) PFOA and rheumatoid arthritis (10-year lag) PFOA and medicated asthma

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Stein 2016a	Cross-sectional	General community (New York, New York)	Adults	78	PFHxS PFOA PFOS PFNA	PFHxS and lower post-vaccination change in serum interferon-gamma (tertile 2, not 3) and tumor necrosis factor-alpha levels PFHxS and greater post-vaccination change in nasal secretion hemagglutinin-specific mucosal immunoglobulin A (tertile 2, not 3) PFOS and greater odds of seroconversion to FluMist vaccine (anti-influenza A/H1N1) measured by hemagglutinin inhibition (low baseline antibodies; tertile 2, not 3) PFOS tertile and lower baseline serum macrophage-inflammatory protein-1a (by class, not trend) PFNA and greater odds of seroconversion to FluMist vaccine (anti-influenza A/H1N1) measured by hemagglutinin inhibition (low baseline antibodies; tertile 2, not 3) PFNA tertile and lower baseline serum interferon-gamma-inducible protein-10 (by class, not trend) and macrophage-inflammatory protein-1a (tertile 2, not 3)	PFHxS and seroconversion to FluMist vaccine (anti-influenza A/H1N1) measured by hemagglutinin inhibition or by immunohistochemisty PFHxS and post-vaccination change in serum interferon-alpha 2, interferon-gamma-inducible protein-10, monocyte chemoattractant protein-1, macrophage-inflammatory protein-1a, or granulocyte colony-stimulating factor level PFHxS and post-vaccination change in nasal secretion interferon-gamma-inducible protein-10, or monocyte chemoattractant protein-1 PFHxS and baseline serum and nasal secretion immune markers (listed above) PFOA and seroconversion to FluMist vaccine (anti-influenza A/H1N1) measured by hemagglutinin inhibition or by immunohistochemisty PFOA and post-vaccination change in interferon-alpha 2, interferon-gamma, tumor necrosis factor-alpha, interferon-gamma-inducible protein-10, monocyte chemoattractant protein-1, macrophage-inflammatory protein-1a, or granulocyte colony-stimulating factor level PFOA and post-vaccination change in nasal secretion interferon-gamma-inducible protein-10, monocyte chemoattractant protein-1, or hemagglutinin-specific mucosal immunoglobulin A PFOA and baseline serum and nasal secretion immune markers (listed above) PFOS and seroconversion to FluMist vaccine (anti-influenza A/H1N1) measured by hemagglutinin inhibition (total population) or by immunohistochemisty PFOS and post-vaccination change in serum interferon-alpha 2, interferon-gamma, tumor necrosis factor-alpha, interferon-gamma-inducible protein-10, monocyte chemoattractant protein-1, macrophage-inflammatory protein-1a, or granulocyte colony-stimulating factor level PFOS and post-vaccination change in nasal secretion interferon-gamma-inducible protein-10, monocyte chemoattractant protein-1, or hemagglutinin-specific mucosal immunoglobulin A PFOS and baseline serum and nasal secretion immune markers (listed above), except as noted

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Stein 2016b	Cross-sectional	General community (United States)	Children, adolescents	1,191	PFHxS PFOA PFOS PFNA	PFHxS and lower anti-rubella antibody level (seropositive) PFHxS and lower risk of allergic sensitization (sIgE ≥ 0.35 kU/L) to cockroach/shrimp PFOA and lower anti-mumps antibody level (seropositive) PFOA and lower anti-rubella antibody level (seropositive) PFOA and greater risk of rhinitis PFOA and greater serum total IgE level PFOS and lower anti-mumps antibody level (all, seropositive) PFOS and lower anti-rubella antibody level (seropositive) PFOS and lower risk of allergic sensitization (sIgE ≥ 0.35 kU/L) to any antigen, plants, or cockroach/shrimp PFOS and greater risk of allergic sensitization (sIgE ≥ 0.35 kU/L) to mold PFNA and greater serum total IgE level	PFHxS and anti-measles antibody level (all, seropositive) PFHxS and anti-mumps antibody level (all, seropositive) PFHxS and anti-rubella antibody level (all) PFHxS and asthma, wheeze, allergy, or rhinitis PFHxS and allergic sensitization (sIgE ≥ 0.35 kU/L) to any antigen, plants, dust mites, pets, rodents, mold, or food PFHxS and serum total IgE PFOA and anti-measles antibody level (all, seropositive) PFOA and anti-mumps antibody level (all) PFOA and anti-rubella antibody level (all) PFOA and asthma, wheeze, or allergy PFOA and allergic sensitization (sIgE ≥ 0.35 kU/L) to any antigen, plants, dust mites, pets, cockroach/shrimp, rodents, mold, or food PFOS and anti-measles antibody level (all, seropositive) PFOS and anti-rubella antibody (all) PFOS and asthma, wheeze, allergy, or rhinitis PFOS and allergic sensitization (sIgE ≥ 0.35 kU/L) to dust mites, pets, rodents, or food PFOS and serum total IgE PFNA and anti-measles antibody level (all, seropositive) PFNA and anti-mumps antibody level (all, seropositive) PFNA and anti-rubella antibody level (all, seropositive) PFNA and asthma, wheeze, allergy, or rhinitis PFNA and allergic sensitization (sIgE ≥ 0.35 kU/L) to any antigen, plants, dust mites, pets, cockroach/shrimp, rodents, mold, or food
Immune	Timmermann 2017a	Prospective cohort and cross-sectional	Fishing community (Faroe Islands)	Children and adolescents	559	PFHxS PFOA PFOS PFNA PFDA	PFHxS at 5 y × MMR vaccination interaction and asthma at 5 y PFHxS at 5 y and greater risk of atopic asthma at 5 y (no MMR vaccination, with interaction) PFOA at 5 y and greater risk of asthma at 5 y (no MMR vaccination, with interaction) PFOA at 5 y and greater risk of atopic asthma at 5 y (no MMR vaccination, with interaction) PFOS at 5 y and greater risk of atopic asthma at 5 y (no MMR vaccination, with interaction) PFNA at 5 y and greater risk of asthma at 5 y (no MMR vaccination, with interaction) PFNA at 5 y and greater risk of atopic asthma at 5 y (no MMR vaccination, with interaction) PFDA at 5 y and greater risk of asthma at 5 y (no MMR vaccination, with interaction) PFDA at 5 y and greater risk of atopic asthma at 5 y (no MMR vaccination, with interaction)	PFHxS at 5 y and asthma at 5 y (no MMR; with MMR); atopic asthma at 5 y (with MMR); allergy at 5 y (no MMR; with MMR); IgE at 7 y Maternal PFHxS and cord blood IgE; asthma at 5 y; allergy at 5 y; IgE at 7 y PFOA at 5 y and asthma at 5 y (with MMR); atopic asthma at 5 y (with MMR); allergy at 5 y (no MMR; with MMR); IgE at 7 y Maternal PFOA and cord blood IgE; asthma at 5 y; allergy at 5 y; IgE at 7 y PFOS at 5 y and asthma at 5 y (no MMR; with MMR); atopic asthma at 5 y (with MMR); allergy at 5 y (no MMR; with MMR); IgE at 7 y Maternal PFOS and cord blood IgE; asthma at 5 y; allergy at 5 y; IgE at 7 y PFNA at 5 y and asthma at 5 y (with MMR); atopic asthma at 5 y (with MMR); allergy at 5 y (no MMR; with MMR); IgE at 7 y Maternal PFNA and cord blood IgE; asthma at 5 y; allergy at 5 y; IgE at 7 y PFDA at 5 y and asthma at 5 y (with MMR); atopic asthma at 5 y (with MMR); allergy at 5 y (no MMR; with MMR); IgE at 7 y Maternal PFDA and cord blood IgE; asthma at 5 y; allergy at 5 y; IgE at 7 y

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Timmermann 2017a (continued)	Prospective cohort and cross-sectional	Fishing community (Faroe Islands)	Children and adolescents	559	PFHxS PFOA PFOS PFNA PFDA	Maternal PFHxS and greater risk of atopic eczema at 13 y PFHxS at 13 y and lower risk of asthma at 13 y PFOA at 5 y and greater risk of asthma at 13 y (no MMR vaccination, with interaction) PFOA at 5 y × MMR vaccination interaction and atopic asthma at 13 y PFNA at 5 y and greater risk of asthma at 13 y (no MMR vaccination, with interaction) PFNA at 5 y and greater risk of atopic asthma at 13 y (no MMR vaccination, with interaction) PFNA at 13 y and lower risk of allergic rhinoconjunctivitis at 13 y PFDA at 5 y and greater risk of asthma at 5 y (no MMR vaccination, with interaction) PFDA at 5 y × MMR vaccination interaction and asthma at 13 y	PFHxS at 5 y and asthma (no MMR; with MMR); nonatopic asthma (no MMR; with MMR); atopic asthma (no MMR; with MMR); positive skin prick test; allergic rhinoconjunctivitis; atopic eczema at 13 y Maternal PFHxS and asthma; positive skin prick test; allergic rhinoconjunctivitis at 13 y PFHxS at 13 y and atopic eczema (no MMR, with MMR); positive skin prick test; allergic rhinoconjunctivitis at 13 y PFOA at 5 y and asthma (with MMR); nonatopic asthma (no MMR; with MMR); atopic asthma (no MMR; with MMR); positive skin prick test; allergic rhinoconjunctivitis; atopic eczema at 13 y Maternal PFOA and asthma; positive skin prick test; allergic rhinoconjunctivitis; atopic eczema at 13 y PFOA at 13 y and atopic eczema (no MMR, with MMR); asthma; positive skin prick test; allergic rhinoconjunctivitis at 13 y PFOS at 5 y and asthma (no MMR, with MMR); nonatopic asthma (no MMR; with MMR); atopic asthma (no MMR; with MMR); positive skin prick test; allergic rhinoconjunctivitis; atopic eczema at 13 y Maternal PFOS and asthma; positive skin prick test; allergic rhinoconjunctivitis; atopic eczema at 13 y PFOS at 13 y and atopic eczema (no MMR, with MMR); asthma; positive skin prick test; allergic rhinoconjunctivitis at 13 y PFNA at 5 y and asthma (with MMR); nonatopic asthma (no MMR; with MMR); atopic asthma (with MMR); positive skin prick test; allergic rhinoconjunctivitis; atopic eczema at 13 y Maternal PFNA and asthma; positive skin prick test; allergic rhinoconjunctivitis; atopic eczema at 13 y PFNA at 13 y and atopic eczema (no MMR, with MMR); asthma; positive skin prick test at 13 y
Immune	Wang 2011	Prospective cohort	General community (Taiwan)	Infants, young children	244	PFOA PFOS PFNA	PFOA and greater cord blood IgE level at 2 years (total, boys) PFOS and greater cord blood IgE level at 2 years (total, boys)	PFOA and serum IgE level at 2 years PFOA and cord blood IgE level at 2 years (girls) PFOA and atopic dermatitis risk at 2 years PFOS and serum IgE level at 2 years PFOS and cord blood IgE level at 2 years (girls) PFOS and atopic dermatitis risk at 2 years PFNA and serum IgE level at 2 years PFNA and cord blood IgE level at 2 years PFNA and atopic dermatitis risk at 2 years

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	White 2011	Cross-sectional	General community (North Carolina)	Women	34	PFHxS PFOS PFOSA PFNA ΣPFAS	PFHxS and lower serum IgE PFHxS and lower serum secretory IgA PFOS and lower serum IgE PFOSA and lower serum IgM PFNA and lower serum secretory IgA Total PFAS and lower serum IgE	PFHxS and serum IgM PFOS and serum secretory IgA PFOS and serum IgM PFOSA and serum IgE PFOSA and serum secretory IgA PFNA and serum IgE PFNA and serum IgM Total PFAS and serum secretory IgA Total PFAS and serum IgM
Immune	Zhou 2017b	Case-control	General community (northern Taiwan)	Children, adolescents	231 cases, 225 controls	PFHxS PFOA PFOS PFNA PFDA	PFOA and lower testosterone level at 10-15 y (asthma) PFOA and greater estradiol level at 10-15 y (asthma) PFOS and lower testosterone level at 10-15 y (asthma) PFOS and greater estradiol level at 10-15 y (asthma) PFOS×testosterone interaction and asthma at 10-15 y (boys, girls) PFOS×estradiol interaction and asthma at 10-15 y (girls) PFNA and greater estradiol level at 10-15 y (no asthma, asthma) PFNA×estradiol interaction and asthma at 10-15 y (girls) PFDA and greater estradiol level at 10-15 y (asthma) PFDA×estradiol interaction and asthma at 10-15 y (boys)	PFHxS and testosterone level at 10-15 y (no asthma, asthma) PFHxS and estradiol level at 10-15 y (no asthma, asthma) PFHxS×testosterone interaction and asthma at 10-15 y (boys, girls) PFHxS×estradiol interaction and asthma at 10-15 y (boys, girls) PFOA and testosterone level at 10-15 y (no asthma) PFOA and estradiol level at 10-15 y (no asthma) PFOA×testosterone interaction and asthma at 10-15 y (boys, girls) PFOA×estradiol interaction and asthma at 10-15 y (boys, girls) PFOS and testosterone level at 10-15 y (no asthma) PFOS and estradiol level at 10-15 y (no asthma) PFOS×estradiol interaction and asthma at 10-15 y (boys) PFNA and testosterone level at 10-15 y (no asthma, asthma) PFNA×testosterone interaction and asthma at 10-15 y (boys, girls) PFNA×estradiol interaction and asthma at 10-15 y (boys) PFDA and testosterone level at 10-15 y (no asthma, asthma) PFDA and estradiol level at 10-15 y (no asthma) PFDA×testosterone interaction and asthma at 10-15 y (boys, girls) PFDA×estradiol interaction and asthma at 10-15 y (girls)

Table 6. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and immune outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Immune	Zhu 2016	Case-control	General community (northern Taiwan)	Children, adolescents	231 cases, 225 controls	PFBS PFHxS PFOA PFOS PFNA PFDA	PFBS and greater risk of asthma at 10-15 y (boys) PFBS and greater interleukin-5 level in asthmatics at 10-15 y (boys) PFHxS and greater risk of asthma at 10-15 y (boys, girls) PFHxS and lower interleukin-2 level in non-asthmatics at 10-15 y (boys, girls) PFOA and greater risk of asthma at 10-15 y (boys, girls) PFOA and greater IgE level at 10-15 y (girls), interleukin-4 at 10-15 y (boys), and interleukin-5 at 10-15 y (boys) levels in asthmatics PFOS and greater risk of asthma at 10-15 y (boys) PFOS and greater IgE level in asthmatics at 10-15 y (girls) PFOS and greater interleukin-4 level in asthmatics at 10-15 y (girls) PFNA and greater risk of asthma at 10-15 y (boys) PFNA and greater IgE, interleukin-4, and interleukin-5 levels in asthmatics at 10-15 y (boys) PFDA and greater risk of asthma at 10-15 y (boys, girls) PFDA and greater IgE level in asthmatics at 10-15 y (boys) PFDA and lower interferon-gamma at 10-15 y (girls quartile 2, not 3 or 4) and interleukin-2 at 10-15 y (boys quartile 2, not 3 or 4) in asthmatics	PFBS and asthma at 10-15 y (girls) PFBS and IgE, interferon-gamma, interleukin-2, interleukin-4, and interleukin 5 (girls) in asthmatics at 10-15 y PFBS and interferon-gamma, interleukin-2, interleukin-4, and interleukin 5 in non-asthmatics at 10-15 y PFHxS and IgE, interferon-gamma, interleukin-2, interleukin-4, and interleukin 5 in asthmatics at 10-15 y PFHxS and interferon-gamma, interleukin-4, and interleukin 5 in non-asthmatics at 10-15 y PFOA and IgE (boys), interferon-gamma, interleukin-2, interleukin-4 (girls), and interleukin 5 (girls) in asthmatics at 10-15 y PFOA and interferon-gamma, interleukin-2, interleukin-4, and interleukin 5 in non-asthmatics at 10-15 y PFOS and asthma at 10-15 y (girls) PFOS and IgE (boys), interferon-gamma, interleukin-2, interleukin-4, and interleukin 5 in asthmatics at 10-15 y PFOS and interferon-gamma, interleukin-2, interleukin-4 (boys), and interleukin 5 in non-asthmatics at 10-15 y PFNA and asthma at 10-15 y (girls) PFNA and IgE (girls), interferon-gamma, interleukin-2, interleukin-4 (girls), and interleukin 5 (girls) in asthmatics at 10-15 y PFNA and interferon-gamma, interleukin-2, interleukin-4, and interleukin 5 in non-asthmatics at 10-15 y PFDA and IgE (girls), interferon-gamma (boys), interleukin-2 (girls), interleukin-4, and interleukin 5 in asthmatics at 10-15 y PFDA and interferon-gamma, interleukin-2, interleukin-4, and interleukin 5 in non-asthmatics at 10-15 y

Table 7. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and kidney outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Kidney	Anderson-Mahoney 2008	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	566	PFOA	Residence in PFOA water district and greater risk of kidney disease (total, males 50-64 y, females 65+ y)	Residence in PFOA water district and risk of kidney disease (males 18-34, 35-49, and 65+ y; females 35-49 and 50-64 y)
Kidney	Consonni 2013	Retrospective cohort	Occupational (Germany, Netherlands, Italy, United Kingdom, New Jersey, West Virginia)	Adults	4,773	PFOA/tetrafluoroethylene (TFE)	None	PFOA and nephritis/nephrosis mortality
Kidney	Costa 2009	Cross-sectional	Occupational (Trissino, Italy)	Men	53	PFOA	PFOA and greater uric acid level	PFOA and urea nitrogen level PFOA and creatinine level PFOA and total protein level PFOA and albumin level
Kidney	Dhingra 2016b	Retrospective and prospective cohort	Contaminated community (Mid-Ohio Valley)	Adults	32,254	PFOA	None	Modeled cumulative or year-specific PFOA and chronic kidney disease (full cohort, prospective cohort, with or without diabetics, and full cohort with 5-, 10-, or 20-year lag)
Kidney	Dhingra 2017	Retrospective cohort and cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	29,499	PFOA	Measured serum PFOA and lower eGFR	Modeled serum or cumulative PFOA and eGFR
Kidney	Emmett 2006	Cross-sectional	Contaminated community (Mid-Ohio Valley)	All (children, adolescents, adults)	371	PFOA	None	PFOA and blood urea nitrogen PFOA and creatinine PFOA and total protein PFOA and albumin
Kidney	Geiger 2013	Cross-sectional	General community (United States)	Adolescents	1,772	PFOA PFOS	PFOA and greater uric acid level PFOA and greater risk of hyperuricemia PFOS and greater uric acid level PFOS and greater risk of hyperuricemia	None
Kidney	Gleason 2015	Cross-sectional	General community (United States)	Adolescents, adults	4,333	PFHxS PFOA PFOS PFNA	PFHxS and greater uric acid level PFOA and greater uric acid level PFOS and greater uric acid level PFNA and greater uric acid level	None
Kidney	Grice 2007	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,400	PFOS	None	PFOS and nephrolithiasis

Table 7. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and kidney outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Kidney	Jiang 2014	Cross-sectional	General community (Tianjin, China)	Women	141	PFHxA PFHxS PFHpA ΣPFOA (n-, iso-, 5m-, %n-) ΣPFOS (n-, iso-, 1m-4m- 3+5m-Σm2- %n-) PFNA PFDA PFunDA PFdoDA ΣPFAS (PFHxA+PFHxS+PFHpA+PFOA+PFOS+PFNA+PFDA+PFunDA+PFdoDA)	PFHpA, iso-PFOA, 5m-PFOA and greater total protein level PFHxA, PFHpA, n-PFOA, iso-PFOA, ΣPFOA, PFNA and greater albumin level	PFHxA, PFHxS, n-PFOA, %n-PFOA, ΣPFOA, n-PFOS, iso-PFOS, 1m-PFOS, 4m-PFOS, 3+5m-PFOS, Σm2-PFOS, %n-PFOS, ΣPFOS, PFNA, PFDA, PFunDA, PFdoDA, ΣPFAS and total protein level PFHxS, 5m-PFOA, %n-PFOA, n-PFOS, iso-PFOS, 1m-PFOS, 4m-PFOS, 3+5m-PFOS, Σm2-PFOS, %n-PFOS, ΣPFOS, PFDA, PFunDA, PFdoDA, ΣPFAS and albumin level
Kidney	Kataria 2015	Cross-sectional	General community (United States)	Adolescents	1,960	PFHxS PFOA PFOS PFNA	PFOA and lower eGFR PFOA and greater uric acid level PFOS and lower eGFR PFOS and greater uric acid level	PFHxS and eGFR PFHxS and uric acid level PFNA and eGFR PFNA and uric acid level
Kidney	Leonard 2008	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	6,027	PFOA	None	PFOA and mortality from nephritis/nephrosis (vs. U.S., West Virginia, or DuPont Region 1)
Kidney	Lin 2013a	Cross-sectional	General community (Taiwan)	Adolescents, young adults	664	PFOA PFOS PFNA PFunDA	None	PFOA and uric acid PFOS and uric acid PFNA and uric acid PFunDA and uric acid
Kidney	Lundin 2009	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,993	PFOA	None	PFOA and mortality from nephritis/nephrosis

Table 7. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and kidney outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Kidney	Qin 2016	Cross-sectional	General community (Taipei, Taiwan)	Adolescents	225	PFBS PFHxA PFHxS PFOA PFOS PFNA PFDA PFdoDA PFteDA	PFHxS and greater uric acid level (total) PFOA and greater uric acid level (total, boys) PFOA and greater risk of hyperuricemia (i.e., uric acid ≥ 6 mg/dL; total, boys)	PFBS and uric acid level or hyperuricemia (i.e., uric acid ≥ 6 mg/dL) PFHxA and uric acid level or hyperuricemia PFHxS and uric acid level (boys, girls) or hyperuricemia (total, boys, girls) PFOA and uric acid level (girls) or hyperuricemia (girls) PFOS and uric acid level or hyperuricemia PFNA and uric acid level or hyperuricemia PFDA and uric acid level or hyperuricemia PFdoDA and uric acid level or hyperuricemia PFteDA and uric acid level or hyperuricemia
Kidney	Raleigh 2014	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	9,027	PFOA	None	PFOA and mortality from chronic renal disease
Kidney	Shankar 2011a	Cross-sectional	General community (United States)	Adults	4,587	PFOA PFOS	PFOA and lower eGFR PFOA and greater risk of chronic kidney disease PFOA and greater uric acid PFOS and lower eGFR PFOS and greater risk of chronic kidney disease PFOS and greater uric acid	None
Kidney	Shankar 2011b	Cross-sectional	General community (United States)	Adults	3,883	PFOA PFOS	PFOA and greater uric acid level (total, men, women, obese, non-obese) PFOA and greater risk of hyperuricemia PFOS and greater uric acid level (total, men, non-obese) PFOS and greater risk of hyperuricemia	PFOS and uric acid level (women, obese)
Kidney	Steenland 2010b	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	54,951	PFOA PFOS	PFOA and greater uric acid level PFOA and greater risk of hyperuricemia PFOS and greater uric acid level PFOS and greater risk of hyperuricemia	None
Kidney	Steenland 2012	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	5,791	PFOA	PFOA and greater risk of mortality from chronic renal disease	None

Table 7. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and kidney outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Kidney	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	None	PFOA and chronic kidney disease
Kidney	Watkins 2013	Retrospective cohort and cross-sectional	Contaminated community (Mid-Ohio Valley)	Children, adolescents	9,660	PFHxS PFOA PFOS PFNA	Measured serum PFHxS and lower eGFR Measured serum PFOA and lower eGFR Measured serum PFOS and lower eGFR Measured serum PFNA and lower eGFR	Modeled PFOA at birth, early life (first 10 years), recent (3 years before enrollment), or enrollment and eGFR

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Chateau-Degat 2010	Cross-sectional	Fishing community (Nunavik, northern Quebec, Canada)	Adults	723	PFOS	PFOS and greater total cholesterol level (categorical, not continuous) PFOS and greater HDL cholesterol level PFOS and lower total cholesterol:HDL cholesterol ratio PFOS and lower triglycerides (women)	PFOS and triglycerides (total, men) PFOS and LDL cholesterol PFOS and non-HDL cholesterol
Lipids	Christensen 2016a	Cross-sectional	Fishing community (Wisconsin anglers)	Men	154	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA	PFHpS and greater risk of high cholesterol	PFHxS and high cholesterol PFOA and high cholesterol PFOS and high cholesterol PFNA and high cholesterol PFDA and high cholesterol PFunDA and high cholesterol
Lipids	Costa 2009	Cross-sectional	Occupational (Trissino, Italy)	Men	53	PFOA	PFOA and greater total cholesterol level	PFOA and HDL cholesterol PFOA and triglycerides PFOA and apolipoprotein A PFOA and apolipoprotein B
Lipids	Domazet 2016	Prospective cohort	General community (Odense, Denmark)	Adolescents, young adults	277	PFOA PFOS	None	PFOA (9 years) and triglycerides at 15 years PFOA (9 years) and triglycerides at 21 years PFOA (15 years) and triglycerides at 21 years PFOS (9 years) and triglycerides at 15 years PFOS (9 years) and triglycerides at 21 years PFOS (15 years) and triglycerides at 21 years Cross-sectional associations at 15 and 21 years
Lipids	Emmett 2006	Cross-sectional	Contaminated community (Mid-Ohio Valley)	All (children, adolescents, adults)	371	PFOA	None	PFOA and total cholesterol
Lipids	Eriksen 2013	Cross-sectional	General community (Denmark)	Adults	753	PFOA PFOS	PFOA and greater total cholesterol level (total, no diabetes, not overweight/obese) PFOS and greater total cholesterol level (total, women, not overweight/obese)	PFOA and total cholesterol (diabetes, overweight/obese) PFOS and total cholesterol (men, overweight/obese)

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Fisher 2013	Cross-sectional	General community (Canada)	Adults	2,700	PFHxS PFOA PFOS	PFHxS and greater total cholesterol:HDL cholesterol ratio PFHxS and greater LDL cholesterol level PFHxS and greater non-HDL cholesterol level PFHxS and greater total cholesterol level PFHxS and risk of high cholesterol	PFHxS and HDL cholesterol PFHxS and triglycerides PFOA and HDL cholesterol PFOA and total cholesterol:HDL cholesterol ratio PFOA and LDL cholesterol PFOA and non-HDL cholesterol PFOA and total cholesterol PFOA and triglycerides PFOA and risk of high cholesterol PFOS and HDL cholesterol PFOS and total cholesterol:HDL cholesterol ratio PFOS and LDL cholesterol PFOS and non-HDL cholesterol PFOS and total cholesterol PFOS and triglycerides PFOS and risk of high cholesterol
Lipids	Fitz-Simon 2013	Prospective cohort	Contaminated community (Mid-Ohio Valley)	Adults	560	PFOA PFOS	PFOA and greater LDL cholesterol level PFOS and greater LDL cholesterol level PFOS and greater total cholesterol level	PFOA and total cholesterol PFOA and HDL cholesterol PFOA and triglycerides PFOS and HDL cholesterol PFOS and triglycerides
Lipids	Frisbee 2010	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Children, adolescents	12,476	PFOA PFOS	PFOA and greater total cholesterol level at 1-<18 years PFOA and greater LDL cholesterol level at 1-<18 years PFOA and greater triglycerides level (girls < 12 years) PFOA and greater risk of high total cholesterol at 1-<18 years PFOA and greater risk of high LDL cholesterol at 1-<18 years PFOS and greater total cholesterol level at 1-<18 years PFOS and greater LDL cholesterol level at 1-<18 years PFOS and greater HDL cholesterol level at 1-<18 years (total, boys) PFOS and lower triglycerides level (girls ≥ 12 years) PFOS and greater risk of high total cholesterol at 1-<18 years PFOS and greater risk of high LDL cholesterol at 1-<18 years	PFOA and HDL cholesterol at 1-<18 years PFOA and triglycerides at 1-<18 years (total, boys, girls ≥ 12 years) PFOA and high HDL cholesterol at 1-<18 years PFOA and high triglycerides at 1-<18 years PFOS and HDL cholesterol at 1-<18 years (girls) PFOS and triglycerides at 1-<18 years (total, boys, girls < 12 years) PFOS and high HDL cholesterol at 1-<18 years PFOS and high triglycerides at 1-<18 years

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Fu 2014	Cross-sectional	General community (Henan, China)	Adults	133	PFOA PFOS PFNA PFDA PFunDA	PFOA and greater total cholesterol level PFOA and greater LDL cholesterol level PFNA and greater total cholesterol level PFNA and greater LDL cholesterol level PFDA and greater total cholesterol level PFDA and greater HDL cholesterol level	PFOA and triglycerides PFOA and HDL cholesterol PFOA and high total cholesterol, triglycerides, HDL cholesterol, or LDL cholesterol PFOS and total cholesterol PFOS and triglycerides PFOS and HDL cholesterol PFOS and LDL cholesterol PFOS and high total cholesterol, triglycerides, HDL cholesterol, or LDL cholesterol PFNA and triglycerides PFNA and HDL cholesterol PFNA and high total cholesterol, triglycerides, HDL cholesterol, or LDL cholesterol PFDA and triglycerides PFDA and LDL cholesterol PFDA and high total cholesterol, triglycerides, HDL cholesterol, or LDL cholesterol PFunDA and total cholesterol PFunDA and triglycerides PFunDA and HDL cholesterol PFunDA and LDL cholesterol PFunDA and high total cholesterol, triglycerides, HDL cholesterol, or LDL cholesterol
Lipids	Geiger 2014a	Cross-sectional	General community (United States)	Adolescents	815	PFOA PFOS	PFOA and greater total cholesterol level PFOA and greater LDL cholesterol level PFOA and lower HDL cholesterol level PFOS and greater total cholesterol level PFOS and greater LDL cholesterol level	PFOA and triglycerides PFOS and HDL cholesterol PFOS and triglycerides
Lipids	Gilliland 1996	Cross-sectional	Occupational (Cottage Grove, Minnesota)	Men	115	Total fluorine (PFOA)	Total fluorine and lower HDL cholesterol level (moderate alcohol drinkers)	Total fluorine and total cholesterol (total) Total fluorine and LDL cholesterol (total) Total fluorine and HDL cholesterol (total)

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Gump 2011	Cross-sectional	General community (Oswego County, New York)	Children	79	PFHxS PFOA PFOS PFOSA PFNA PFDA	PFDA and greater total cholesterol level at 9-11 years PFDA and greater LDL cholesterol level at 9-11 years	PFHxS and total cholesterol at 9-11 years PFHxS and LDL cholesterol at 9-11 years PFHxS and HDL cholesterol at 9-11 year PFHxS and triglycerides at 9-11 yearss PFOA and total cholesterol at 9-11 years PFOA and LDL cholesterol at 9-11 years PFOA and HDL cholesterol at 9-11 years PFOA and triglycerides at 9-11 years PFOS and total cholesterol at 9-11 years PFOS and LDL cholesterol at 9-11 years PFOS and HDL cholesterol at 9-11 years PFOS and triglycerides at 9-11 years PFOSA and total cholesterol at 9-11 years PFOSA and LDL cholesterol at 9-11 years PFOSA and HDL cholesterol at 9-11 years PFOSA and triglycerides at 9-11 years PFNA and total cholesterol at 9-11 years PFNA and LDL cholesterol at 9-11 years PFNA and HDL cholesterol at 9-11 years PFNA and triglycerides at 9-11 years PFDA and HDL cholesterol at 9-11 years PFDA and triglycerides at 9-11 years
Lipids	Kataria 2015	Cross-sectional	General community (United States)	Adolescents	1,960	ΣPFAS (PFHxS+PFOA+PFOS+PFNA)	None	ΣPFAS and total cholesterol ≥ 170 mg/dL

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Kishi 2015	Cross-sectional	General community (Hokkaido, Japan)	Pregnant women	306	PFOA PFOS	PFOA and greater palmitic acid PFOS and lower triglycerides PFOS and lower palmitic acid PFOS and lower palmitoleic acid PFOS and lower oleic acid PFOS and lower linoleic acid PFOS and lower alpha-linolenic acid PFOS and lower arachidonic acid PFOS and lower essential fatty acids PFOS and lower omega-6 fatty acids	PFOA and triglycerides PFOA and palmitic acid PFOA and palmitoleic acid PFOA and stearic acid PFOA and oleic acid PFOA and linoleic acid PFOA and alpha-linolenic acid PFOA and arachidonic acid PFOA and eicosapentaenoic acid PFOA and docosahexaenoic acid PFOA and essential fatty acids PFOA and omega-6 fatty acids PFOA and omega-3 fatty acids PFOS and stearic acid PFOS and eicosapentaenoic acid PFOS and docosahexaenoic acid PFOS and omega-3 fatty acids
Lipids	Lin 2009	Cross-sectional	General community (United States)	Adolescents, adults	474 adolescents 969 adults	PFHxS PFOA PFOS PFNA	PFOS and greater risk of HDL cholesterol component of metabolic syndrome (adults) PFNA and lower risk of HDL cholesterol component of metabolic syndrome (adolescents)	PFHxS and HDL cholesterol component of metabolic syndrome (adolescents, adults) PFHxS and triglycerides component of metabolic syndrome (adolescents, adults) PFOA and HDL cholesterol component of metabolic syndrome (adolescents, adults) PFOA and triglycerides component of metabolic syndrome (adolescents, adults) PFOS and HDL cholesterol component of metabolic syndrome (adolescents) PFOS and triglycerides component of metabolic syndrome (adolescents, adults) PFNA and HDL cholesterol component of metabolic syndrome (adults) PFNA and triglycerides component of metabolic syndrome (adolescents, adults)

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Lin 2011	Cross-sectional	General community (Taiwan)	Adolescents, young adults	287	PFOA PFOS PFNA PFunDA ΣPFAS	None	PFOA and HDL cholesterol at 12-30 years PFOA and triglycerides at 12-30 years PFOS and HDL cholesterol at 12-30 years PFOS and triglycerides at 12-30 years PFNA and HDL cholesterol at 12-30 years PFNA and triglycerides at 12-30 years PFunDA and HDL cholesterol at 12-30 years PFunDA and triglycerides at 12-30 years ΣPFAS and HDL cholesterol at 12-30 years ΣPFAS and triglycerides at 12-30 years
Lipids	Lin 2013a	Cross-sectional	General community (Taiwan)	Adolescents, young adults	664	PFOA PFOS PFNA PFunDA	PFOA and lower triglycerides at 12-30 years	PFOA and LDL cholesterol at 12-30 years PFOS and LDL cholesterol at 12-30 years PFOS and triglycerides at 12-30 years PFNA and LDL cholesterol at 12-30 years PFNA and triglycerides at 12-30 years PFunDA and LDL cholesterol at 12-30 years PFunDA and triglycerides at 12-30 years
Lipids	MacPherson 2011	Phase I clinical trial	Clinical trial (patients with advanced refractory solid tumors, United Kingdom)	Adults	41	PFOA	PFOA and lower LDL cholesterol level	None

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Maisonet 2015b	Prospective cohort	General community (Avon, United Kingdom)	Children, adolescents (girls)	230	PFOA PFOS	PFOA and greater total cholesterol level in lowest prenatal tertile at 7 y PFOA and greater LDL cholesterol level in lowest prenatal tertile at 7 y PFOA and greater LDL cholesterol level in lowest prenatal tertile at 15 y PFOA and lower LDL cholesterol level in highest prenatal tertile at 15 y PFOS and greater total cholesterol level in second prenatal tertile at 15 y PFOS and lower total cholesterol level in highest prenatal tertile at 15 y PFOS and lower LDL cholesterol level in highest prenatal tertile at 15 y	PFOA and total cholesterol in prenatal tertiles 2 and 3 at 7 y PFOA and total cholesterol in prenatal tertiles 1, 2, 3 at 15 y PFOA and LDL cholesterol in prenatal tertiles 2 and 3 at 7 y PFOA and LDL cholesterol in prenatal tertile 2 at 15 y PFOA and HDL cholesterol in prenatal tertiles 1, 2, 3 at 7 and 15 y PFOA and triglycerides in prenatal tertiles 1, 2, 3 at 7 and 15 y PFOS and total cholesterol in prenatal tertiles 1, 2, 3 at 7 y PFOS and total cholesterol in prenatal tertile 1 at 15 y PFOS and LDL cholesterol in prenatal tertiles 1, 2, 3 at 7 y PFOS and LDL cholesterol in prenatal tertiles 1 and 2 at 15 y PFOS and HDL cholesterol in prenatal tertiles 1, 2, 3 at 7 and 15 y PFOS and triglycerides in prenatal tertiles 1, 2, 3 at 7 and 15 y
Lipids	Mattsson 2015	Prospective case-control	General community (rural Sweden)	Men	231 cases, 231 controls	PFHpA PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA	PFHxS and greater total cholesterol PFHxS and greater LDL cholesterol PFOA and greater LDL cholesterol PFOS and greater total cholesterol PFNA and greater HDL cholesterol PFDA and greater total cholesterol PFDA and greater HDL cholesterol PFunDA and greater total cholesterol PFunDA and greater LDL cholesterol PFunDA and greater HDL cholesterol PFdoDA and greater total cholesterol PFdoDA and greater LDL cholesterol PFdoDA and greater HDL cholesterol	PFHpA and total cholesterol PFHpA and LDL cholesterol PFHpA and HDL cholesterol PFHxS and HDL cholesterol PFOA and total cholesterol PFOA and HDL cholesterol PFOS and LDL cholesterol PFOS and HDL cholesterol PFNA and total cholesterol PFNA and LDL cholesterol PFDA and LDL cholesterol

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Nelson 2010	Cross-sectional	General community (United States)	Adolescents, adults	1,055	PFHxS PFOA PFOS PFNA	PFHxS and lower total cholesterol (F 12-19 y; F 20-59 y; all 20-80 y) PFHxS and greater HDL cholesterol (all 20-80 y no trend) PFHxS and lower HDL cholesterol (M 60-80 y quartile 3, not 4, no trend) PFHxS and lower non-HDL cholesterol (F 12-19 y; F 20-59 y; all 20-80 y) PFHxS and lower LDL cholesterol (F 12-19 y; all 20-80 y) PFOA and greater total cholesterol (all 20-80 y) PFOA and greater HDL cholesterol (F 12-19 y) PFOA and lower HDL cholesterol (M 60-80 y) PFOA and greater non-HDL cholesterol (F 60-80 y; all 20-80 y) PFOA and lower LDL cholesterol (F 60-80 y (quartile 2, not 3 or 4, no trend) PFOS and greater total cholesterol (F 60-80 y; all 20-80 y) PFOS and greater non-HDL cholesterol (F 60-80 y; all 20-80 y) PFNA and greater total cholesterol (F 20-59 quartiles 2 and 3, not 4, no trend; all 20-80 y) PFNA and greater non-HDL cholesterol (F 20-59 y quartile 3, not 4, no trend; all 20-80 y) PFNA and greater LDL cholesterol (M 20-59 y; all 20-80 y quartile 2, not 3 or 4, no trend) PFNA and lower LDL cholesterol (males 12-19 y quartile 2, not 3 or 4, no trend)	PFHxS and total cholesterol (M 12-19 y; M 20-59 y; M & F 60-80 y) PFHxS and HDL cholesterol (M & F 12-19 y; M & F 20-59 y; F 60-80 y) PFHxS and non-HDL cholesterol (M 12-19 y; M 20-59 y; M & F 60-80 y) PFHxS and LDL cholesterol (M 12-19 y; M & F 20-59 and 60-80 y) PFOA and total cholesterol (M & F 12-19, 20-59, and 60-80 y) PFOA and HDL cholesterol (M 12-19 y; M & F 20-59 y; F 60-80 y; adults 20-80 y) PFOA and non-HDL cholesterol (M & F 12-19 and 20-59 y; M 60-80 y) PFOA and LDL cholesterol (M & F 12-19 and 20-59 y; M 60-80 y; adults 20-80 y) PFOS and total cholesterol (M & F 12-19 and 20-59 y; M 60-80 y) PFOS and HDL cholesterol (M & F 12-19, 20-59, and 60-80 y; adults 20-80 y) PFOS and non-HDL cholesterol (M & F 12-19 and 20-59 y; M 60-80 y) PFOS and LDL cholesterol (M & F 12-19, 20-59, and 60-80 y; adults 20-80 y) PFNA and total cholesterol (M & F 12-19, M 20-59 y, and M & F 60-80 y) PFNA and HDL cholesterol (M & F 12-19, 20-59, and 60-80 y; adults 20-80 y) PFNA and non-HDL cholesterol (M & F 12-19 y; M 20-59 y; M & F 60-80 y) PFNA and LDL cholesterol (feM 12-19 and 20-59 y; M & F 60-80 y)
Lipids	Olsen 1999	Cross-sectional	Occupational (Decatur, Alabama; Antwerp, Belgium)	Men	178 in 1995 149 in 1997	PFOS	PFOS and greater total cholesterol level in 1997 PFOS and greater LDL cholesterol level in 1997 PFOS and lower HDL cholesterol level in 1995	PFOS and total cholesterol in 1995 PFOS and LDL cholesterol in 1995 PFOS and HDL cholesterol in 1997 PFOS and triglycerides in 1995 and 1997
Lipids	Olsen 2000	Cross-sectional	Occupational (Cottage Grove, Minnesota)	Men	111 in 1993 80 in 1995 74 in 1997	PFOA	Total fluorine and lower HDL cholesterol level, especially in moderate drinkers, in 1990 (Gilliland 1996)	PFOA and total cholesterol in 1993, 1995, and 1997 PFOA and HDL cholesterol in 1993, 1995, and 1997 PFOA and LDL cholesterol in 1993, 1995, and 1997 PFOA and triglycerides in 1993, 1995, and 1997

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Olsen 2003a	Prospective cohort and cross-sectional	Occupational (Decatur, Alabama; Antwerp, Belgium)	Adults	518 174 longitudinal	PFOA PFOS Total organic fluorine	PFOA and greater total cholesterol level (longitudinal) PFOA and greater triglycerides level (longitudinal) PFOS and greater triglycerides level (men) Total organic fluorine and greater total cholesterol level (longitudinal) Total organic fluorine and greater triglycerides level (longitudinal)	PFOA and HDL cholesterol (longitudinal) PFOS and total cholesterol (men, women) PFOS and HDL cholesterol (men, women) PFOS and triglycerides (women) PFOS and total cholesterol, triglycerides, or HDL cholesterol (longitudinal) Total organic fluorine and HDL cholesterol (longitudinal)
Lipids	Olsen 2004	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,311	PFOS	None	PFOS and episodes of care for disorders of lipid metabolism (hyperlipidemia)
Lipids	Olsen 2007	Cross-sectional	Occupational (Antwerp, Belgium; Cottage Grove, Minnesota; Decatur, Alabama)	Men	506	PFOA	PFOA and lower HDL cholesterol level (all) PFOA and greater triglycerides level (all, Antwerp)	PFOA and total cholesterol PFOA and LDL cholesterol PFOA and HDL cholesterol (Antwerp, Cottage Grove, Decatur) PFOA and triglycerides (Cottage Grove, Decatur) PFOA and high total cholesterol PFOA and high LDL cholesterol PFOA and low HDL cholesterol PFOA and high triglycerides
Lipids	Olsen 2012a	Prospective cohort	Occupational (Cottage Grove, Minnesota; Decatur, Alabama)	Adults	179	PFOA PFOS	Increase in PFOA and greater HDL cholesterol level Increase in PFOA and lower total cholesterol:HDL cholesterol ratio PFOA and lower total cholesterol:HDL cholesterol ratio PFOS and greater HDL cholesterol level	Increase or decrease in PFOA and total cholesterol Increase or decrease in PFOA and non-HDL cholesterol Decrease in PFOA and HDL cholesterol Decrease in PFOA and total cholesterol:HDL cholesterol ratio PFOA and total cholesterol PFOA and non-HDL cholesterol PFOA and HDL cholesterol PFOS and total cholesterol PFOS and non-HDL cholesterol PFOS and total cholesterol:HDL cholesterol ratio Increase in PFOA and PFOS (from baseline PFOA < 15 ng/mL, PFOS < 50 ng/mL) and total cholesterol, non-HDL cholesterol, HDL cholesterol, and total cholesterol:HDL cholesterol ratio
Lipids	Sakr 2007a	Cross-sectional	Occupational (Parkersburg, West Virginia)	Adults	1,019	PFOA	PFOA and greater total cholesterol level PFOA and greater LDL cholesterol level PFOA and greater VLDL cholesterol level	PFOA and HDL cholesterol PFOA and triglycerides
Lipids	Sakr 2007b	Prospective cohort	Occupational (Parkersburg, West Virginia)	Adults	454	PFOA	PFOA and greater total cholesterol level	PFOA and triglycerides PFOA and HDL cholesterol PFOA and LDL cholesterol

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Shankar 2011a	Cross-sectional	General community (United States)	Adults	4,587	PFOA PFOS	PFOA and greater total cholesterol PFOS and greater total cholesterol	None
Lipids	Shankar 2012	Cross-sectional	General community (United States)	Adults	1,216	PFOA	None	PFOA and total cholesterol
Lipids	Skuladottir 2015	Cross-sectional	General community (Aarhus, Denmark)	Women	854	PFHxS PFHpA PFHpS PFOA PFOS PFOSA PFNA PFDA PFdoDA	PFOA and greater total cholesterol level PFOS and greater total cholesterol level PFNA and greater total cholesterol level (quintiles 3 and 4, not 5; no trend)	PFHxS and total cholesterol PFHpA and total cholesterol PFHpS and total cholesterol PFOSA and total cholesterol PFDA and total cholesterol PFdoDA and total cholesterol
Lipids	Starling 2017	Cross-sectional	General community (Colorado)	Women	598	PFHxS PFOA PFOS PFNA PFDA	PFHxS and greater HDL cholesterol PFHxS and lower triglycerides PFOA and greater HDL cholesterol	PFOA and triglycerides PFOS and HDL PFOS and triglycerides PFNA and HDL PFNA and triglycerides PFDA and HDL PFDA and triglycerides
Lipids	Steenland 2009	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	46,294	PFHxS PFOA PFOS PFNA	PFHxS and greater total cholesterol level PFOA and greater total cholesterol level PFOA and greater LDL cholesterol level PFOA and greater triglycerides level PFOA and greater total cholesterol:HDL cholesterol ratio PFOA and greater non-HDL cholesterol level PFOA and greater risk of high cholesterol PFOS and greater total cholesterol level PFOS and greater LDL cholesterol level PFOS and greater triglycerides level PFOS and greater total cholesterol:HDL cholesterol ratio PFOS and greater non-HDL cholesterol level PFOS and greater risk of high cholesterol PFNA and greater total cholesterol level	PFOA and HDL cholesterol PFOS and HDL cholesterol

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	None	PFOA and medicated high cholesterol
Lipids	Timmermann 2014	Cross-sectional	General community (Denmark)	Children	495	PFOA PFOS	PFOA and greater triglycerides at 8-10 years (overweight) PFOS and greater triglycerides at 8-10 years (overweight)	PFOA and triglycerides at 8-10 years (normal weight) PFOS and triglycerides at 8-10 years (normal weight)
Lipids	Wang 2012	Cross-sectional	Occupational and contaminated community (Changshu City, Jiangsu Province, China)	NR (includes adults)	55 workers 132 residents	PFOA PFOS	PFOA and lower HDL cholesterol level (workers) PFOA and lower HDL cholesterol:LDL cholesterol ratio (workers)	PFOA and total cholesterol (workers, residents) PFOA and HDL cholesterol (residents) PFOA and LDL cholesterol (workers, residents) PFOA and HDL cholesterol:LDL cholesterol ratio (residents) PFOA and triglycerides (workers, residents) PFOS and total cholesterol PFOS and HDL cholesterol PFOS and LDL cholesterol PFOS and HDL cholesterol:LDL cholesterol ratio PFOS and triglycerides
Lipids	White 2011	Cross-sectional	General community (North Carolina)	Women	34	PFOA Other PFAS (unspecified)	None	PFOA and total cholesterol PFOA and total lipids Other PFAS and total cholesterol Other PFAS and total lipids
Lipids	Winquist 2014a	Retrospective cohort	Contaminated community (Mid-Ohio Valley) and occupational (Parkersburg, West Virginia)	Adults	32,254	PFOA	PFOA and greater risk of hypercholesterolemia treated with medication, retrospective analysis (men and women 20-107 years; men and women 40-59 years; men 20-107 years; men 40-59 years) PFOA and lower risk of hypercholesterolemia treated with medication, prospective analysis (total cohort)	PFOA and hypercholesterolemia treated with medication (men and women 20-39 or 60-79 years; women 20-107, 20-39, 40-59, or 60-79 years; men 20-39 or 60-79 years)

Table 8. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and lipid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Lipids	Zeng 2015	Cross-sectional	General community (Taipei, Taiwan)	Adolescents	225	PFBS PFHxA PFHxS PFOA PFOS PFNA PFDA PFdoDA PFteDA	PFBS and greater total cholesterol level at 12-15 years PFOA and greater total cholesterol, LDL cholesterol, and triglycerides levels at 12-15 years PFOS and greater total cholesterol, LDL cholesterol, and triglycerides levels at 12-15 years PFNA and greater total cholesterol, LDL cholesterol, and triglycerides levels at 12-15 years	PFBS and HDL cholesterol, LDL cholesterol, and triglycerides at 12-15 years PFHxA and total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides at 12-15 years PFHxS and total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides at 12-15 years PFOA and HDL cholesterol at 12-15 years PFOS and HDL cholesterol at 12-15 years PFNA and HDL cholesterol at 12-15 years PFDA and total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides at 12-15 years PFdoA and total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides at 12-15 years PFteA and total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides at 12-15 years

Table 9. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and liver outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Liver	Alexander 2003	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	2,083	PFOS	None	PFOS and liver cirrhosis mortality
Liver	Anderson-Mahoney 2008	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	566	PFOA	Residence in PFOA water district and greater risk of liver problems (females 18-34 y)	Residence in PFOA water district and liver problems (total, males 18-34 y, males and females 35-49 y, males and females 50-64 y, males 65+ y)
Liver	Consonni 2013	Retrospective cohort	Occupational (Germany, Netherlands, Italy, United Kingdom, New Jersey, West Virginia)	Adults	4,773	PFOA/tetrafluoroethylene (TFE)	None	PFOA and liver cirrhosis mortality
Liver	Costa 2009	Cross-sectional	Occupational (Trissino, Italy)	Men	53	PFOA	PFOA and lower total bilirubin level (current/former/never exposed workers) PFOA and greater alanine aminotransferase level (current/former/never exposed workers) PFOA and greater gamma-glutamyl transferase level (current/former/never exposed workers) PFOA and greater alkaline phosphatase level (current/former/never exposed workers)	PFOA and total bilirubin (exposed vs. non-exposed or other workers) PFOA and conjugated bilirublin PFOA and aspartate aminotransferase PFOA and alanine aminotransferase (exposed vs. non-exposed or other workers) PFOA and gamma-glutamyl transferase (exposed vs. non-exposed or other workers) PFOA and alkaline phosphatase (exposed vs. non-exposed or other workers)
Liver	Darrow 2016	Retrospective cohort and cross-sectional	Contaminated community (Mid-Ohio Valley) and occupational (Parkersburg, West Virginia)	Adults	32,254	PFOA	PFOA and greater alanine aminotransferase level (cumulative and current) PFOA and lower direct bilirubin level (cumulative and current)	PFOA and gamma glutamyl transferase (cumulative and current) PFOA and any liver disease PFOA and enlarged liver, fatty liver, or cirrhosis
Liver	Emmett 2006	Cross-sectional	Contaminated community (Mid-Ohio Valley)	All (children, adolescents, adults)	371	PFOA	PFOA and lower risk of abnormal aspartate aminotransferase	PFOA and bilirubin PFOA and alkaline phosphatase PFOA and aspartate aminotransferase (concentration) PFOA and alanine aminotransferase PFOA and gamma glutamyl transpeptidase PFOA and self-reported liver disease
Liver	Fan 2014	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	54,446	PFPA PFHxA PFHxS PFHpA PFOA PFOS PFNA PFDA PFunDA PFdoDA	PFHxA and greater risk of Gilbert syndrome phenotype (total bilirubin 1.1-4.9 mg/dL, direct bilirubin < 0.4 mg/dL, normal liver enzymes, no anemia) (total, men, women) PFNA and greater risk of Gilbert syndrome phenotype (total) PFDA and greater risk of Gilbert syndrome phenotype (total, men)	PFPA and Gilbert syndrome phenotype PFHxS and Gilbert syndrome phenotype PFHpA and Gilbert syndrome phenotype PFOA and Gilbert syndrome phenotype PFOS and Gilbert syndrome phenotype PFNA and Gilbert syndrome phenotype (men, women) PFDA and Gilbert syndrome phenotype (women) PFunDA and Gilbert syndrome phenotype PFdoDA and Gilbert syndrome phenotype

Table 9. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and liver outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Liver	Fisher 2013	Cross-sectional	General community (Canada)	Adults	2,700	PFHxS PFOA PFOS	None	PFHxS and self-reported liver disease PFOA and self-reported liver disease PFOS and self-reported liver disease
Liver	Gallo 2012	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	46,452	PFOA PFOS	PFOA and greater alanine aminotransferase level PFOA and greater gamma glutamyl transferase level PFOA and greater risk of high alanine aminotransferase level PFOA and greater risk of high gamma glutamyl transferase level (decile 7, not, 8, 9, 10, or trend) PFOS and greater alanine aminotransferase level PFOS and greater direct bilirubin level PFOS and greater risk of high alanine aminotransferase level	PFOA and direct bilirubin PFOA and high direct bilirubin PFOS and gamma glutamyl transferase PFOS and high gamma glutamyl transferase PFOS and high direct bilirubin
Liver	Gilliland 1996	Cross-sectional	Occupational (Cottage Grove, Minnesota)	Men	115	Total fluorine (PFOA)	Interaction between total fluorine × obesity and aspartate aminotransferase level Total fluorine and lower aspartate aminotransferase level (non-obese) Total fluorine and greater aspartate aminotransferase level (obese) Interaction between total fluorine × obesity and alanine aminotransferase level Total fluorine and lower alanine aminotransferase level (non-obese) Total fluorine and greater aspartate aminotransferase level (obese)	Total fluorine and aspartate aminotransferase (total) Total fluorine and alanine aminotransferase (total) Total fluorine and gamma glutamyl transferase (total) Total fluorine and self-reported hepatic disease diagnoses, signs, or symptoms Total fluorine and clinical liver dysfunction
Liver	Gleason 2015	Cross-sectional	General community (United States)	Children, adolescents, adults	4,333	PFHxS PFOA PFOS PFNA	PFHxS and greater alanine aminotransferase level PFHxS and greater aspartate aminotransferase level PFHxS and greater alkaline phosphatase level PFHxS and greater total bilirubin level PFOA and greater alanine aminotransferase level PFOA and greater gamma glutamyltransferase level PFOA and greater aspartate aminotransferase level PFOA and greater total bilirubin level PFOS and greater gamma glutamyltransferase level PFOS and greater total bilirubin level PFNA and greater alanine aminotransferase level PFNA and greater gamma glutamyltransferase level	PFHxS and gamma glutamyltransferase PFOA and alkaline phosphatase PFOS and alanine aminotransferase PFOS and aspartate aminotransferase PFOS and alkaline phosphatase PFNA and aspartate aminotransferase PFNA and alkaline phosphatase PFNA and total bilirubin

Table 9. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and liver outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Liver	Grice 2007	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,400	PFOS	None	PFOS and cholelithiasis PFOS and cholecystitis PFOS and liver disease, including cirrhosis and hepatitis
Liver	Jiang 2014	Cross-sectional	General community (Tianjin, China)	Women	141	PFHxA PFHxS PFHpA ΣPFOA (n-, iso-, 5m-, %n-) ΣPFOS (n-, iso-, 1m- 4m- 3+5m- Σm2- %n-) PFNA PFDA PFunDA PFdoDA ΣPFAS (PFHxA+PFHxS+PFHpA+PFOA+PFOS+PFNA+PFDA+PFunDA+PFdoDA)	PFHxA, PFHpA, Σm2-PFOS, PFDA, PFdoDA, and greater total bilirubin level	PFHxS, n-PFOA, iso-PFOA, 5m-PFOA, %n-PFOA, ΣPFOA, n-PFOS, iso-PFOS, 1m-PFOS, 4m-PFOS, 3+5m-PFOS,%n-PFOS, ΣPFOS, PFNA, PFunDA, ΣPFAS and total bilirubin PFHxA, PFHxS, PFHpA, n-PFOA, iso-PFOA, 5m-PFOA, %n-PFOA, ΣPFOA, n-PFOS, iso-PFOS, 1m-PFOS, 4m-PFOS, 3+5m-PFOS, Σm2-PFOS, %n-PFOS, ΣPFOS, PFNA, PFDA, PFunDA, PFdoDA, ΣPFAS and aspartate aminotransferase PFHxA, PFHxS, PFHpA, n-PFOA, iso-PFOA, 5m-PFOA, %n-PFOA, ΣPFOA, n-PFOS, iso-PFOS, 1m-PFOS, 4m-PFOS, 3+5m-PFOS, Σm2-PFOS, %n-PFOS, ΣPFOS, PFNA, PFDA, PFunDA, PFdoDA, ΣPFAS and alanine aminotransferase
Liver	Leonard 2008	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	6,027	PFOA	PFOA and lower risk of mortality from cirrhosis of the liver (vs. US or West Virginia)	PFOA and mortality from cirrhosis of the liver (vs. DuPont Region 1 workers)

Table 9. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and liver outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Liver	Lin 2010	Cross-sectional	General community (United States)	Adults	2,216	PFHxS PFOA PFOS PFNA	PFHxS and greater total bilirubin level PFOA and greater alanine aminotransferase level (total, non-Hispanic white, ≤ high school education, BMI ≥ 30, never smoked, < 60 alcoholic drinks/year, metabolic syndrome, HOMA-IR > 2.91) PFOA and greater gamma glutamyl transferase level (total, non-Hispanic white, BMI ≥ 30, < 60 alcoholic drinks/year, HOMA-IR 1.61-2.91 or > 2.91) PFOS and lower gamma glutamyl transferase level PFOS and lower total bilirubin level PFNA and greater total bilirubin level	PFHxS and alanine aminotransferase PFHxS and gamma glutamyl transferase PFOA and alanine aminotransferase (non-white, > high school education, BMI < 30, ever smoked, ≥ 60 alcoholic drinks/year, no metabolic syndrome, HOMA-IR ≤ 2.91; strata of iron saturation, age, sex) PFOA and gamma glutamyl transferase (non-white, BMI < 30, ≥ 60 alcoholic drinks/year, HOMA-IR ≤ 1.61; strata of iron saturation, metabolic syndrome, smoking status, education, age, sex) PFOA and total bilirubin PFOS and alanine aminotransferase PFNA and alanine aminotransferase PFNA and gamma glutamyl transferase
Liver	Lundin 2009	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,993	PFOA	None	PFOA and mortality from cirrhosis of the liver
Liver	Melzer 2010	Cross-sectional	General community (United States)	Adults	3,974	PFOA PFOS	None	PFOA and liver disease current PFOS and liver disease current
Liver	Olsen 1999	Cross-sectional	Occupational (Decatur, Alabama; Antwerp, Belgium)	Men	266	PFOS	PFOS and lower total bilirubin level in 1995 and 1997 PFOS and lower direct bilirubin level in 1997	PFOS and alkaline phosphatase in 1995 and 1997 PFOS and gamma glutamyl transferase in 1995 and 1997 PFOS and aspartate aminotransferase in 1995 and 1997 PFOS and alanine aminotransferase in 1995 and 1997 PFOS and direct bilirubin in 1995

Table 9. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and liver outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Liver	Olsen 2000	Cross-sectional	Occupational (Cottage Grove, Minnesota)	Men	111 in 1993 80 in 1995 74 in 1997	PFOA	Total fluorine and lower alanine aminotransferase level in 1990 (Gilliland 1996) PFOA and greater alanine aminotransferase level in 1997 Interaction of PFOA × BMI on alanine aminotransferase level in 1990 (positive) and 1997 (negative)	PFOA and alkaline phosphatase in 1993, 1995, and 1997 PFOA and gamma glutamyl transferase in 1993, 1995, and 1997 PFOA and aspartate aminotransferase in 1993, 1995, and 1997 PFOA and alanine aminotransferase in 1993 and 1995 PFOA and total bilirubin in 1993, 1995, and 1997 PFOA and direct bilirubin in 1993, 1995, and 1997
Liver	Olsen 2003a	Prospective cohort and cross-sectional	Occupational (Decatur, Alabama; Antwerp, Belgium)	Adults	263 Decatur 255 Antwerp 174 longitudinal	PFOA PFOS	PFOS and greater alkaline phosphatase level (men, women) PFOS and greater alanine aminotransferase level (men) PFOS and greater gamma glutamyl transferase level (women) PFOS and lower total bilirubin level (men, women)	PFOS and gamma glutamyl transferase (men) PFOS and aspartate aminotransferase (men, women) PFOS and alanine aminotransferase (women) PFOS and abnormally high alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase, gamma glutamyl transferase, or total liver panels (men, women) PFOA and liver function tests: alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase, and gamma glutamyl transferase (longitudinal) PFOS and liver function tests: alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase, and gamma glutamyl transferase (longitudinal) Total organic fluorine and liver function tests: alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase, and gamma glutamyl transferase (longitudinal)

Table 9. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and liver outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Liver	Olsen 2004	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,311	PFOS	PFOS and greater risk of episodes of care for disorders of the biliary tract (long-term chemical workers) PFOS and greater risk of episodes of care for cholelithiasis, acute cholecystitis (chemical workers, long-term chemical workers)	PFOS and episodes of care for liver disorders (chemical workers, long-term chemical workers) PFOS and episodes of care for liver cirrhosis without mention of alcohol (chemical workers, long-term chemical workers) PFOS and episodes of care for disorders of the biliary tract (chemical workers) PFOS and episodes of care for cholelithiasis, chronic cholecystitis (chemical workers, long-term chemical workers) PFOS and episodes of care for cholelithiasis without cholecystitis (chemical workers, long-term chemical workers)
Liver	Olsen 2007	Cross-sectional	Occupational (Antwerp, Belgium; Cottage Grove, Minnesota; Decatur, Alabama)	Men	506	PFOA	PFOA and greater alkaline phosphatase level (Decatur) PFOA and greater alanine aminotransferase level (Decatur) PFOA and greater gamma glutamyl transferase level (all, Decatur) PFOA and lower total bilirubin level (all, Decatur)	PFOA and alkaline phosphatase (all, Antwerp, Cottage Grove) PFOA and aspartate aminotransferase PFOA and alanine aminotransferase (all, Antwerp, Cottage Grove) PFOA and gamma glutamyl transferase (Antwerp, Cottage Grove) PFOA and total bilirubin (Antwerp, Cottage Grove) PFOA and direct bilirubin PFOA and high alanine aminotransferase PFOA and high gamma glutamyl transferase
Liver	Olsen 2012a	Prospective cohort	Occupational (Cottage Grove, Minnesota; Decatur, Alabama)	Adults	179	PFOA PFOS	Decrease in PFOA and greater aspartate aminotransferase level Increase in PFOA and PFOS (from baseline PFOA < 15 ng/mL, PFOS < 50 ng/mL) and lower total bilirubin level	Increase or decrease in PFOA and total bilirubin Increase or decrease in PFOA and alkaline phosphatase Increase in PFOA and aspartate aminotransferase Increase or decrease in PFOA and alanine aminotransferase PFOA and total bilirubin PFOA and alkaline phosphatase PFOA and aspartate aminotransferase PFOA and alanine aminotransferase PFOS and total bilirubin PFOS and alkaline phosphatase PFOS and aspartate aminotransferase PFOS and alanine aminotransferase Increase in PFOA and PFOS (from baseline PFOA < 15 ng/mL, PFOS < 50 ng/mL) and alkaline phosphatase, aspartate aminotransferase, and alanine aminotransferase

Table 9. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and liver outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Liver	Sakr 2007a	Cross-sectional	Occupational (Parkersburg, West Virginia)	Adults	1,019	PFOA	PFOA and greater gamma glutamyl transferase level	PFOA and aspartate aminotransferase PFOA and alanine aminotransferase PFOA and bilirubin
Liver	Sakr 2007b	Prospective cohort	Occupational (Parkersburg, West Virginia)	Adults	454	PFOA	PFOA and lower total bilirubin level PFOA and greater aspartate aminotransferase level	PFOA and gamma glutamyl transferase PFOA and alkaline phosphatase PFOA and alanine aminotransferase
Liver	Steenland 2012	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	5,791	PFOA	None	PFOA and mortality from chronic liver disease
Liver	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	None	PFOA and non-hepatitis liver disease
Liver	Wang 2012	Cross-sectional	Occupational and contaminated community (Changshu City, Jiangsu Province, China)	NR (includes adults)	55 workers 132 residents	PFOA	PFOA and lower aspartate aminotransferase level (workers)	PFOA and alanine aminotransferase (workers, residents) PFOA and aspartate aminotransferase (residents)
Liver	Yamaguchi 2013	Cross-sectional	General community (Japan)	Adolescents, adults	608	PFOA PFOS	PFOA and greater alanine aminotransferase level PFOA and greater aspartate aminotransferase level PFOS and greater alanine aminotransferase level PFOS and greater aspartate aminotransferase level	PFOA and gamma glutamyl transferase PFOS and gamma glutamyl transferase

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Anderson-Mahoney 2008	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	566	PFOA	Residence in PFOA water district and greater risk of diabetes	None
Metabolic	Ashley-Martin 2017	Prospective cohort	General community (Canada)	Newborns	1,705	PFHxS PFOA PFOS	None	PFHxS and adiponectin PFHxS and leptin PFOA and adiponectin PFOA and leptin PFOS and adiponectin PFOS and leptin
Metabolic	Christensen 2016a	Cross-sectional	Fishing community (Wisconsin anglers)	Men	154	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA	PFNA and greater diabetes/pre-diabetes risk PFNA and greater pre-diabetes risk PFDA and greater diabetes/pre-diabetes risk PFDA and greater pre-diabetes risk PFunDA and greater diabetes/pre-diabetes risk PFunDA and greater pre-diabetes risk	PFHxS and diabetes/pre-diabetes PFHxS and pre-diabetes PFHpS and diabetes/pre-diabetes PFHpS and pre-diabetes PFOA and diabetes/pre-diabetes PFOA and pre-diabetes PFOS and diabetes/pre-diabetes PFOS and pre-diabetes
Metabolic	Christensen 2016b	Cross-sectional	Fishing community (Wisconsin anglers)	Men	154	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA ΣPFAS (PFHxS+PFHpS+PFOA+PFOS+PFNA+PFDA+PFunDA)	PFDA and greater pre-diabetes risk PFunDA and greater pre-diabetes risk	PFHxS and diabetes PFHxS and pre-diabetes PFHpS and diabetes PFHpS and pre-diabetes PFOA and diabetes PFOA and pre-diabetes PFOS and diabetes PFOS and pre-diabetes PFNA and diabetes PFNA and pre-diabetes PFDA and diabetes PFunDA and diabetes ΣPFAS and diabetes ΣPFAS and pre-diabetes

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Consonni 2013	Retrospective cohort	Occupational (Germany, Netherlands, Italy, United Kingdom, New Jersey, West Virginia)	Adults	4,773	PFOA/tetrafluoroethylene (TFE)	None	PFOA and diabetes mortality
Metabolic	Conway 2016	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Children, adolescents, adults	66,899	PFHxS PFOA PFOS PFNA	PFHxS and lower risk of type 2 diabetes PFHxS and lower risk of uncategorized diabetes PFOA and lower risk of type 2 diabetes (adults) PFOA and lower risk of uncategorized diabetes (adults) PFOS and lower risk of type 2 diabetes	PFOA and type 2 diabetes (children < 20 years) PFOA and uncategorized diabetes (children < 20 years) PFOS and uncategorized diabetes PFNA and type 2 diabetes PFNA and uncategorized diabetes
Metabolic	Costa 2009	Cross-sectional	Occupational (Trissino, Italy)	Men	53	PFOA	None	PFOA and glucose level
Metabolic	Domazet 2016	Prospective cohort	General community (Odense, Denmark)	Adolescents, young adults	277	PFOA PFOS	PFOA (9 years) and lower HOMA-Beta at 15 years	PFOA (9 years) and glucose at 15 years PFOA (9 years) and insulin at 15 years PFOA (9 years) and triglycerides at 15 years PFOA (9 years) and HOMA-IR at 15 years PFOA (9 years) and glucose at 21 years PFOA (9 years) and insulin at 21 years PFOA (9 years) and triglycerides at 21 years PFOA (9 years) and HOMA-Beta at 21 years PFOA (9 years) and HOMA-IR at 21 years PFOA (15 years) and glucose at 21 years PFOA (15 years) and insulin at 21 years PFOA (15 years) and triglycerides at 21 years PFOA (15 years) and HOMA-Beta at 21 years PFOA (15 years) and HOMA-IR at 21 years PFOS (9 years) and glucose at 15 years PFOS (9 years) and insulin at 15 years PFOS (9 years) and triglycerides at 15 years PFOS (9 years) and HOMA-Beta at 15 years PFOS (9 years) and HOMA-IR at 15 years PFOS (9 years) and glucose at 21 years PFOS (9 years) and insulin at 21 years PFOS (9 years) and triglycerides at 21 years PFOS (9 years) and HOMA-Beta at 21 years PFOS (9 years) and HOMA-IR at 21 years PFOS (15 years) and glucose at 21 years PFOS (15 years) and insulin at 21 years PFOS (15 years) and triglycerides at 21 years PFOS (15 years) and HOMA-Beta at 21 years PFOS (15 years) and HOMA-IR at 21 years Cross-sectional associations at 15 and 21 years

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Fisher 2013	Cross-sectional	General community (Canada)	Adults	2,700	PFHxS PFOA PFOS	None	PFHxS and insulin level PFHxS and glucose level PFHxS and HOMA-IR PFHxS and risk of metabolic syndrome PFOA and insulin level PFOA and glucose level PFOA and HOMA-IR PFOA and risk of metabolic syndrome PFOS and insulin level PFOS and glucose level PFOS and HOMA-IR PFOS and risk of metabolic syndrome
Metabolic	Fleisch 2017	Prospective cohort and cross-sectional	General community (Boston, Massachusetts)	Children	584	PFHxS PFOA PFOS PFNA PFDA	PFHxS and lower leptin level at ~7 years (cross-sectional) PFOA and lower adiponectin level at ~7 years (prospective; quartile 2, not 3 or 4, no trend) PFOA and lower leptin level at ~7 years (cross-sectional) PFOA and lower adiponectin level at ~7 years (cross-sectional; quartiles 2 and 3, not 4, no trend) PFOA and lower HOMA-IR at ~7 years (cross-sectional) PFOS and lower HOMA-IR at ~7 years (cross-sectional; girls) PFNA and lower HOMA-IR at ~7 years (cross-sectional; no trend) PFDA and lower adiponectin level at ~7 years (cross-sectional; no trend) PFDA and lower HOMA-IR at ~7 years (cross-sectional; including after adjustment for other PFAS)	PFHxS and leptin at ~7 years (prospective) PFHxS and adiponectin at ~7 years (prospective) PFHxS and HOMA-IR at ~7 years (prospective) PFHxS and adiponectin at ~7 years (cross-sectional) PFHxS and HOMA-IR at ~7 years (cross-sectional) PFOA and leptin at ~7 years (prospective) PFOA and HOMA-IR at ~7 years (prospective) PFOS and leptin at ~7 years (prospective) PFOS and adiponectin at ~7 years (prospective) PFOS and HOMA-IR at ~7 years (prospective) PFOS and leptin at ~7 years (cross-sectional) PFOS and adiponectin at ~7 years (cross-sectional) PFOS and HOMA-IR at ~7 years (cross-sectional; total, boys) PFNA and leptin at ~7 years (prospective) PFNA and adiponectin at ~7 years (prospective) PFNA and HOMA-IR at ~7 years (prospective) PFNA and leptin at ~7 years (cross-sectional) PFNA and adiponectin at ~7 years (cross-sectional) PFDA and leptin at ~7 years (cross-sectional)
Metabolic	Gilliland 1993	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,537	PFOA	None	PFOA and diabetes mortality (men, men in chemical division)

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Halldorsson 2012	Prospective cohort	General community (Aarhus, Denmark)	Young adults	665	PFOA PFOS PFOSA PFNA	PFOA and greater insulin at 20 years (females) PFOA and greater leptin at 20 years (females) PFOA and lower adiponectin at 20 years (females) PFOA and greater leptin/adiponectin ratio at 20 years (females)	PFOA and greater insulin at 20 years (males) PFOA and greater leptin at 20 years (males) PFOA and lower adiponectin at 20 years (males) PFOA and greater leptin/adiponectin ratio at 20 years (males) PFOS and greater insulin at 20 years (males, females) PFOS and greater leptin at 20 years (males, females) PFOS and lower adiponectin at 20 years (males, females) PFOS and greater leptin/adiponectin ratio at 20 years (males, females) PFOSA and greater insulin at 20 years (males, females) PFOSA and greater leptin at 20 years (males, females) PFOSA and lower adiponectin at 20 years (males, females) PFOSA and greater leptin/adiponectin ratio at 20 years (males, females) PFNA and greater insulin at 20 years (males, females) PFNA and greater leptin at 20 years (males, females) PFNA and lower adiponectin at 20 years (males, females) PFNA and greater leptin/adiponectin ratio at 20 years (males, females)

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Jiang 2014	Cross-sectional	General community (Tianjin, China)	Women	141	PFHxA PFHxS PFHpA ΣPFOA (n-, iso-, 5m-, %n-) ΣPFOS (n-, iso-, 1m-4m- 3+5m-Σm2- %n-) PFNA PFDA PFunDA PFdoDA ΣPFAS (PFHxA+PFHxS+PFHpA+PFOA+PFOS+PFNA+PFDA+PFunDA+PFdoDA)	PFunDA and greater glucose level	PFHxA, PFHxS, PFHpA, n-PFOA, iso-PFOA, 5m-PFOA, %n-PFOA, ΣPFOA, n-PFOS, iso-PFOS, 1m-PFOS, 4m-PFOS, 3+5m-PFOS, Σm2-PFOS, %n-PFOS, ΣPFOS, PFNA, PFDA, PFdoDA, ΣPFAS and glucose level
Metabolic	Karnes 2014	Prospective and retrospective cohort	Contaminated community (Mid-Ohio Valley) and occupational (Parkersburg, West Virginia)	Adults	32,254	PFOA	None	PFOA and type II diabetes (retrospective and prospective) PFOA and fasting glucose in non-diabetics (retrospective)
Metabolic	Kataria 2015	Cross-sectional	General community (United States)	Adolescents	1,960	PFHxS PFOA PFOS PFNA	PFHxS and greater risk of HOMA-IR ≥ 4.39 PFOA and greater risk of HOMA-IR ≥ 4.39 PFOS and greater risk of HOMA-IR ≥ 4.39 PFNA and greater risk of HOMA-IR ≥ 4.39	None

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Kim 2016b	Randomized controlled trial	General community (Seoul, South Korea)	Older adults	126	PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA	PFOS and greater HOMA-IR (baseline) PFOS and greater insulin level (baseline; baseline and placebo) PFOS and greater risk of insulin resistance (HOMA-IR ≥ 2.5) (baseline and placebo) PFdoDA and greater HOMA-IR (baseline; baseline and placebo) PFdoDA and greater insulin level (baseline; baseline and placebo) PFdoDA and greater risk of insulin resistance (HOMA-IR ≥ 2.5) (baseline and placebo)	PFHxS and HOMA-IR, glucose, and insulin (baseline; baseline and placebo; vitamin C supplementation) PFOA and HOMA-IR, glucose, and insulin (baseline; baseline and placebo; vitamin C supplementation) PFOS and HOMA-IR (baseline and placebo, vitamin C supplementation) PFOS and glucose (baseline; baseline and placebo; vitamin C supplementation) PFOS and insulin (vitamin C supplementation) PFOS and insulin resistance (HOMA-IR ≥ 2.5) (vitamin C supplementation) PFNA and HOMA-IR, glucose, and insulin (baseline; baseline and placebo; vitamin C supplementation) PFDA and HOMA-IR, glucose, and insulin (baseline; baseline and placebo; vitamin C supplementation) PFunDA and HOMA-IR, glucose, and insulin (baseline; baseline and placebo; vitamin C supplementation) PFdoDA and HOMA-IR, glucose, and insulin (vitamin C supplementation) PFdoDA and insulin resistance (HOMA-IR ≥ 2.5) (vitamin C supplementation) PFtrDA and HOMA-IR, glucose, and insulin (baseline; baseline and placebo; vitamin C supplementation)
Metabolic	Leonard 2008	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	6,027	PFOA	PFOA and greater risk of diabetes mortality (vs. DuPont Region 1; men)	PFOA and diabetes mortality (vs. US or West Virginia; women)

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Lin 2009	Cross-sectional	General community (United States)	Adolescents, adults	474 adolescents 969 adults	PFHxS PFOA PFOS PFNA	PFOA and greater beta-cell function (adults) PFOS and greater insulin level (adults) PFOS and greater HOMA-IR (adults) PFOS and greater beta-cell function (adults) PFNA and lower insulin level (adolescents) PFNA and lower beta-cell function (adolescents) PFNA and lower risk of metabolic syndrome (adolescents) PFNA and greater risk of glucose component of metabolic syndrome (adolescents)	PFHxS and glucose level (adolescents, adults) PFHxS and insulin level (adolescents, adults) PFHxS and HOMA-IR (adolescents, adults) PFHxS and beta-cell function (adolescents, adults) PFHxS and metabolic syndrome (adolescents, adults) PFHxS and glucose component of metabolic syndrome (adolescents, adults) PFOA and glucose level (adolescents, adults) PFOA and insulin level (adolescents, adults) PFOA and HOMA-IR (adolescents, adults) PFOA and beta-cell function (adolescents) PFOA and metabolic syndrome (adolescents, adults) PFOA and glucose component of metabolic syndrome (adolescents, adults) PFOS and glucose level (adolescents, adults) PFOS and insulin level (adolescents) PFOS and HOMA-IR (adolescents) PFOS and beta-cell function (adolescents) PFOS and metabolic syndrome (adolescents, adults) PFOS and glucose component of metabolic syndrome (adolescents, adults) PFNA and glucose level (adolescents, adults) PFNA and insulin level (adults) PFNA and HOMA-IR (adolescents, adults) PFNA and beta-cell function (adults) PFNA and metabolic syndrome (adolescents, adults) PFNA and glucose component of metabolic syndrome (adolescents, adults)

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Lin 2011	Cross-sectional	General community (Taiwan)	Adolescents, young adults	287	PFOA PFOS PFNA PFunDA ΣPFAS	PFNA and greater adiponectin (total, males, 20-30 years, HOMA-IR > 0.57)	PFOA and adiponectin PFOA and glucose PFOA and insulin PFOA and HOMA-IR PFOS and adiponectin PFOS and glucose PFOS and insulin PFOS and HOMA-IR PFNA and adiponectin (females, 12-19 years, HOMA-IR ≤ 0.57) PFNA and glucose PFNA and insulin PFNA and HOMA-IR PFunDA and adiponectin PFunDA and glucose PFunDA and insulin PFunDA and HOMA-IR ΣPFAS and adiponectin ΣPFAS and glucose ΣPFAS and insulin ΣPFAS and HOMA-IR
Metabolic	Lin 2013a	Cross-sectional	General community (Taiwan)	Adolescents, young adults	664	PFOA PFOS PFNA PFunDA	PFNA and lower HOMA-IR	PFOA and HOMA-IR PFOS and HOMA-IR PFunDA and HOMA-IR

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Lind 2014	Cross-sectional	General community (Uppsala, Sweden)	Older adults	1,016	PFHpA PFHxS PFOA PFOS (linear) PFOSA PFNA PFunDA	PFOA and greater proinsulin/insulin ratio PFNA and greater risk of diabetes (model with linear+quadratic terms)	PFHpA and diabetes, proinsulin/insulin ratio, and HOMA-IR PFHxS and diabetes, proinsulin/insulin ratio, and HOMA-IR PFOA and diabetes and HOMA-IR PFOS and diabetes, proinsulin/insulin ratio, and HOMA-IR PFOSA and diabetes, proinsulin/insulin ratio, and HOMA-IR PFNA and proinsulin/insulin ratio and HOMA-IR PFunDA and diabetes, proinsulin/insulin ratio, and HOMA-IR PFAS × sex interactions and diabetes, proinsulin/insulin ratio, and HOMA-IR
Metabolic	Lind 2017b	Prospective cohort	General community (Uppsala, Sweden)	Older adults	452	PFHxS PFHpA PFOA PFOS (linear) PFOSA PFNA PFunDA	None	PFHxS and metabolic syndrome PFHpA and metabolic syndrome PFOA and metabolic syndrome PFOS (linear) and metabolic syndrome PFOSA and metabolic syndrome PFNA and metabolic syndrome PFunDA and metabolic syndrome
Metabolic	Lopez-Espinosa 2016	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Children	1,169 boys 1,123 girls	PFHxS PFOA PFOS PFNA	PFOA and lower insulin-like growth factor 1 level at 6-9 years (girls) PFOS and lower insulin-like growth factor 1 level at 6-9 years (girls, boys) PFNA and lower insulin-like growth factor 1 level at 6-9 years (girls, boys)	PFHxS and insulin-like growth factor 1 at 6-9 years (girls, boys) PFOA and insulin-like growth factor 1 at 6-9 years (boys)
Metabolic	Lundin 2009	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,993	PFOA	PFOA and greater risk of diabetes mortality (ever probable/never definite exposure)	PFOA and diabetes mortality (ever definite exposure, no deaths)

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	MacNeil 2009	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	4,149 cases, 48,776 controls (self-reported, unrestricted) 1,035 cases, 12,106 controls (validated, restricted) 21,642 fasting glucose	PFOA	PFOA and lower risk of type II diabetes (validated, long-term resident of water district of interest; validated, no residency restriction; self-reported, no residency restriction	PFOA and fasting glucose (non-diabetics)
Metabolic	Melzer 2010	Cross-sectional	General community (United States)	Adults	3,974	PFOA PFOS	None	PFOA and diabetes ever PFOS and diabetes ever
Metabolic	Minatoya 2017	Prospective cohort	General community (Hokkaido, Japan)	Newborns	168	PFOA PFOS	PFOS and greater total adiponectin at birth Interaction of PFOS × sex and leptin at birth	PFOA and total adiponectin at birth PFOA and high-molecular-weight adiponectin at birth PFOA and leptin at birth Interaction of PFOA × sex and total adiponectin, high-molecular-weight adiponectin, and leptin at birth PFOS and high-molecular-weight adiponectin at birth PFOS and leptin at birth Interaction of PFOS × sex and total adiponectin and high-molecular-weight adiponectin at birth
Metabolic	Nelson 2010	Cross-sectional	General community (United States)	Adolescents, adults	1,055	PFHxS PFOA PFOS PFNA	PFHxS and lower HOMA-beta (females 12-19 years) PFOA and lower HOMA-beta (females 12-19 years) PFNA and greater HOMA-beta (females 20-59 years)	PFHxS and HOMA-beta (males 12-19 years; men and women 20-59 and 60-80 years; adults 20-80 years) PFOA and HOMA-beta (males 12-19 years; men and women 20-59 and 60-80 years; adults 20-80 years) PFOS and HOMA-beta (males and females 12-19, 20-59, and 60-80 years; adults 20-80 years) PFNA and HOMA-beta (males and females 12-19 years; men 20-59 years; men and women 60-80 years; adults 20-80 years)

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Nolan 2010	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Women	1,548	PFOA	None	PFOA (Little Hocking Water Association only) and maternal diabetes (type not specified) PFOA (partial Little Hocking Water Association) and maternal diabetes (type not specified)
Metabolic	Olsen 2004	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,311	PFOS	None	PFOS and episodes of care for diabetes
Metabolic	Raleigh 2014	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	9,027	PFOA	None	PFOA and diabetes mortality
Metabolic	Shankar 2011a	Cross-sectional	General community (United States)	Adults	4,587	PFOA PFOS	PFOS and greater glycohemoglobin	PFOA and diabetes PFOA and glycohemoglobin PFOS and diabetes
Metabolic	Shankar 2012	Cross-sectional	General community (United States)	Adults	1,216	PFOA	None	PFOA and diabetes
Metabolic	Shapiro 2016	Prospective cohort	General community (Canada)	Women	1,274	PFHxS PFOA PFOS	PFHxS and greater risk of gestational impaired glucose tolerance (quartile 2, not 3 or 4, no trend) PFHxS and greater risk of gestational diabetes mellitus or impaired glucose tolerance (quartile 2, not 3 or 4, no trend)	PFHxS and gestational diabetes mellitus PFOA and gestational diabetes mellitus PFOA and gestational impaired glucose tolerance PFOA and gestational diabetes mellitus or impaired glucose tolerance PFOS and gestational diabetes mellitus PFOS and gestational impaired glucose tolerance PFOS and gestational diabetes mellitus or impaired glucose tolerance
Metabolic	Starling 2017	Cross-sectional	General community (Colorado)	Women	628	PFHxS PFOA PFOS PFNA PFDA	PFHxS and lower glucose PFOA and lower glucose PFNA and lower glucose PFDA and lower glucose	PFOS and glucose
Metabolic	Steenland 2012	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	5,791	PFOA	PFOA and greater risk of diabetes mortality	None
Metabolic	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	None	PFOA and type 2 diabetes

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Su 2016	Cross-sectional	General community (Taipei, Taiwan)	Adults	571	PFOA PFOS PFNA PFunDA	PFOA and lower fasting blood glucose at 0, 30, 60, 90, and 120 minutes PFOA and lower area under the glucose tolerance curve PFOA and lower HbA1c PFOA and lower risk of diabetes PFOS and greater fasting blood glucose at 0, 30, 60, 90, and 120 minutes PFOS and greater area under the glucose tolerance curve PFOS and greater HbA1c PFOS and greater risk of diabetes PFNA and lower fasting blood glucose at 30, 60, 90, and 120 minutes PFNA and lower area under the glucose tolerance curve PFNA and lower risk of diabetes PFunDA and lower fasting blood glucose at 0, 30, 60, 90, and 120 minutes PFunDA and lower area under the glucose tolerance curve PFunDA and lower risk of diabetes	PFNA and fasting blood glucose at 0 minutes PFNA and HbA1c PFunDA and HbA1c
Metabolic	Timmermann 2014	Cross-sectional	General community (Denmark)	Children	495	PFOA PFOS	PFOA and greater insulin at 8-10 years (overweight) PFOA and greater HOMA-beta at 8-10 years (overweight) PFOA and greater HOMA-IR at 8-10 years (overweight) PFOS and greater insulin at 8-10 years (overweight) PFOS and greater HOMA-beta at 8-10 years (overweight) PFOS and greater HOMA-IR at 8-10 years (overweight)	PFOA and adiponectin at 8-10 years PFOA and leptin at 8-10 years PFOA and insulin at 8-10 years (normal weight) PFOA and HOMA-beta at 8-10 years (normal weight) PFOA and HOMA-IR at 8-10 years (normal weight) PFOA and glucose at 8-10 years (normal weight, overweight) PFOS and adiponectin at 8-10 years PFOS and leptin at 8-10 years PFOS and insulin at 8-10 years (normal weight) PFOS and HOMA-beta at 8-10 years (normal weight) PFOS and HOMA-IR at 8-10 years (normal weight) PFOS and glucose at 8-10 years (normal weight, overweight)

Table 10. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and metabolic outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Metabolic	Valvi 2017	Retrospective	Fishing community (Faroe Islands)	Women	604	PFHxS PFOA PFOS PFNA PFDA ΣPFAS (PFHxS+PFOA+PFOS+PFNA+PFDA)	None	PFHxS and gestational diabetes mellitus PFOA and gestational diabetes mellitus PFOS and gestational diabetes mellitus PFNA and gestational diabetes mellitus PFDA and gestational diabetes mellitus ΣPFAS and gestational diabetes mellitus
Metabolic	Zhang 2015	Prospective	General community (Michigan and Texas, United States)	Women	258	PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	PFOA and greater risk of gestational diabetes mellitus	PFOS and gestational diabetes mellitus PFOSA and gestational diabetes mellitus Me-PFOSA-AcOH and gestational diabetes mellitus Et-PFOSA-AcOH and gestational diabetes mellitus PFNA and gestational diabetes mellitus PFDA and gestational diabetes mellitus
Metabolic	Zong 2016	Cross-sectional	General community (United States)	Women	1,029	PFHxS PFOA PFOS PFNA	None	PFHxS and diabetes PFOA and diabetes PFOS and diabetes PFNA and diabetes

Table 11. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and all-cause mortality

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Mortality	Alexander 2003	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	2,083	PFOS	PFOS and lower risk of all-cause mortality	None
Mortality	Consonni 2013	Retrospective cohort	Occupational (Germany, Netherlands, Italy, United Kingdom, New Jersey, West Virginia)	Adults	4,773	PFOA/tetrafluoroethylene (TFE)	PFOA and lower risk of all-cause mortality	None
Mortality	Gilliland 1993	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,537	PFOA	PFOA and lower risk of all-cause mortality (women, men)	PFOA and all-cause mortality (men in chemical division)
Mortality	Leonard 2008	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	6,027	PFOA	PFOA and lower risk of all-cause mortality (vs. US total, West Virginia total, US men, or West Virginia men) PFOA and greater risk of all-cause mortality (vs. DuPont Region 1 women)	PFOA and all-cause mortality (vs. DuPont Region 1 total, DuPont Region 1 men, US female, or West Virginia female)
Mortality	Lundin 2009	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,993	PFOA	PFOA and lower risk of all-cause mortality (ever probable/never definite)	PFOA and all-cause mortality (ever definite)
Mortality	Raleigh 2014	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	9,027	PFOA	PFOA and lower risk of all-cause mortality	None
Mortality	Steenland 2012	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	5,791	PFOA	None	PFOA and all-cause mortality
Mortality	Ubel 1980	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,688	PFOA	None	PFOA and "all the various causes of death"

Table 12. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurodevelopmental outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurodevelopment	Braun 2014	Cross-sectional	General community (United States)	Children	175	PFHxS PFOA PFOS PFNA	PFOS and greater Social Responsiveness Scale total score at 4-5 years (boys)	PFHxS and Social Responsiveness Scale total score at 4-5 years PFOA and Social Responsiveness Scale total score at 4-5 years PFOS and Social Responsiveness Scale total score at 4-5 years (total, girls) PFNA and Social Responsiveness Scale total score at 4-5 years
Neurodevelopment	Chen 2013	Prospective cohort	General community (northern Taiwan)	Toddlers	239	PFOA PFOS	PFOS and lower Comprehensive Developmental Inventory whole test score at 2 years (IQR ln PFOS increase, not continuous or percentiles) PFOS and lower Comprehensive Developmental Inventory gross-motor score at 2 years PFOS and greater risk of poor (lowest 10%) Comprehensive Developmental Inventory gross-motor score at 2 years PFOS and greater risk of poor (lowest 10%) Comprehensive Developmental Inventory cognitive score at 2 years (30th-59th percentile, not 60-89th or ≥ 90th; not IQR or continuous)	PFOA and Comprehensive Developmental Inventory total score at 2 years PFOA and Comprehensive Developmental Inventory cognitive score at 2 years PFOA and Comprehensive Developmental Inventory language score at 2 years PFOA and Comprehensive Developmental Inventory gross-motor score at 2 years PFOA and Comprehensive Developmental Inventory fine-motor score at 2 years PFOA and Comprehensive Developmental Inventory social score at 2 years PFOA and Comprehensive Developmental Inventory self-help score at 2 years PFOA and poor (lowest 10%) Comprehensive Developmental Inventory total, cognitive, language, gross-motor, fine-motor, social, or self-help score at 2 years PFOS and Comprehensive Developmental Inventory cognitive score at 2 years PFOS and Comprehensive Developmental Inventory language score at 2 years PFOS and Comprehensive Developmental Inventory fine-motor score at 2 years PFOS and Comprehensive Developmental Inventory social score at 2 years PFOS and Comprehensive Developmental Inventory self-help score at 2 years PFOS and poor (lowest 10%) Comprehensive Developmental Inventory total, language, fine-motor, social, or self-help score at 2 years
Neurodevelopment	Donauer 2015	Prospective cohort	General community (Cincinnati, Ohio)	Infants	349	PFOA PFOS	PFOA and greater risk of NICU Network Neurobehavioral Scale "hypotonic" profile at 5 weeks	PFOA and NICU Network Neurobehavioral Scale attention, self-regulation, quality of movement, arousal, excitability, special handling required, lethargy, nonoptimal reflexes, asymmetrical reflexes, hypotonicity, and stress/abstinence scores at 5 weeks PFOA and NICU Network Neurobehavioral Scale "difficult" profile at 5 weeks PFOS and NICU Network Neurobehavioral Scale attention, self-regulation, quality of movement, arousal, excitability, special handling required, lethargy, nonoptimal reflexes, asymmetrical reflexes, hypotonicity, and stress/abstinence scores at 5 weeks PFOS and NICU Network Neurobehavioral Scale "difficult" profile at 5 weeks PFOS and NICU Network Neurobehavioral Scale "hypotonic" profile at 5 weeks

Table 12. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurodevelopmental outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurodevelopment	Fei 2008a	Prospective cohort	General community (Denmark)	Toddlers	1,400	PFOA PFOS	PFOS and lower odds of sitting without support (gross motor developmental milestone) at 18 months PFOS and greater odds of not using word-like sounds to tell what he/she wants (language developmental milestone) at 18 months PFOS and lower odds of not using sentences of two words (language developmental milestone) at 18 months	PFOA and gross motor developmental milestones (sit without support; walk without support; did not go up stairs without support) at 18 months PFOA and fine motor developmental milestones (did not take off socks and shoes when asked to do so; did not drink from an ordinary cup without help) at 18 months PFOA and attention developmental milestones (was not occupied alone with the same thing for at least 15 minutes) at 18 months PFOA and cognition developmental milestones (did not bring things when told to; did not make marks on table or paper; did not turn the picture right when looking in a book) at 18 months PFOA and language developmental milestones (did not use word-like sounds to tell what he/she wants; number of things he/she can mention by name; did not use sentences of two words) PFOS and gross motor developmental milestones (walk without support; did not go up stairs without support) at 18 months PFOS and fine motor developmental milestones (did not take off socks and shoes when asked to do so; did not drink from an ordinary cup without help) at 18 months PFOS and attention developmental milestones (was not occupied alone with the same thing for at least 15 minutes) at 18 months PFOS and cognition developmental milestones (did not bring things when told to; did not make marks on table or paper; did not turn the picture right when looking in a book) at 18 months PFOS and language developmental milestones (did not use word-like sounds to tell what he/she wants; number of things he/she can mention by name; did not use sentences of two words)
Neurodevelopment	Fei 2011	Prospective cohort	General community (Denmark)	Childen	787	PFOA PFOS	PFOA and lower risk of high Strengths and Difficulties Questionnaire total difficulties score at ~7 years (quartile 3, not 4 or trend) PFOA and lower risk of high Strengths and Difficulties Questionnaire emotional symptoms score at ~7 years (quartile 2, not 3 or 4 or trend) PFOA and lower risk of high Strengths and Difficulties Questionnaire hyperactivity score at ~7 years (quartiles 2 and 3, not 4 or trend) PFOA and lower Strengths and Difficulties Questionnaire prosocial behavior score at ~7 years PFOS and lower Strengths and Difficulties Questionnaire prosocial behavior score at ~7 years	PFOA and high Strengths and Difficulties Questionnaire emotional symptoms score at ~7 years PFOA and high Strengths and Difficulties Questionnaire conduct problems score at ~7 years PFOA and high Strengths and Difficulties Questionnaire peer problems score at ~7 years PFOA and low Strengths and Difficulties Questionnaire prosocial behavior score at ~7 years PFOA and low Developmental Coordination Disorder Questionnaire score at ~7 years PFOS and high Strengths and Difficulties Questionnaire total difficulties score at ~7 years PFOS and high Strengths and Difficulties Questionnaire emotional symptoms score at ~7 years PFOS and high Strengths and Difficulties Questionnaire hyperactivity score at ~7 years PFOS and high Strengths and Difficulties Questionnaire conduct problems score at ~7 years PFOS and high Strengths and Difficulties Questionnaire peer problems score at ~7 years PFOS and low Strengths and Difficulties Questionnaire prosocial behavior score at ~7 years PFOS and low Developmental Coordination Disorder Questionnaire score at ~7 years
Neurodevelopment	Forns 2015	Prospective cohort	General community (Norway)	Infants, toddlers	843	PFOA PFOS	None	PFOA and Infant-Toddler Symptom Checklist score at 12 months PFOA and Infant-Toddler Symptom Checklist score at 24 months PFOA and abnormal Ages and Stages Questionnaire-II score at 6 months PFOA and abnormal Ages and Stages Questionnaire-II score at 24 months PFOS and Infant-Toddler Symptom Checklist score at 12 months PFOS and Infant-Toddler Symptom Checklist score at 24 months PFOS and abnormal Ages and Stages Questionnaire-II score at 6 months PFOS and abnormal Ages and Stages Questionnaire-II score at 24 months

Table 12. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurodevelopmental outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurodevelopment	Goudarzi 2016b	Prospective cohort	General community (Hokkaido, Japan)	Infants, toddlers	173	PFOA PFOS	PFOA and lower Bayley Scales of Infant Development II Mental Development Index at 6 months (total, girls)	PFOA and Bayley Scales of Infant Development II Mental Development Index at 6 months (boys) PFOA and Bayley Scales of Infant Development II Psychomotor Development Index at 6 months PFOA and Bayley Scales of Infant Development II Mental Development Index at 18 months PFOA and Bayley Scales of Infant Development II Psychomotor Development Index at 18 months PFOS and Bayley Scales of Infant Development II Mental Development Index at 6 months PFOS and Bayley Scales of Infant Development II Psychomotor Development Index at 6 months PFOS and Bayley Scales of Infant Development II Mental Development Index at 18 months PFOS and Bayley Scales of Infant Development II Psychomotor Development Index at 18 months
Neurodevelopment	Gump 2011	Cross-sectional	General community (Oswego County, New York)	Children	79	PFHxS PFOA PFOS PFOSA PFNA PFDA ΣPFAS (PFHxS+PFOA+PFOS+PFOSA+PFNA+PFDA)	PFHxS and shorter inter-response time on differential reinforcement of low rates of responding (DRL) task (interpreted as impaired response inhibition) at 6-10 min at 9-11 y PFOS and shorter inter-response time at 11-15 min at 9-11 y PFOSA and shorter inter-response time at 6-10 min at 9-11 y PFNA and shorter inter-response time at 6-10 min at 9-11 y PFDA and shorter inter-response time at 6-10 and 11-15 min PFDA and poorer overall test performance (less money earned on DRL task) at 9-11 y ΣPFAS and shorter inter-response time at 6-10, 11-15, and 16-20 min at 9-11 y	PFHxS and inter-response time on differential reinforcement of low rates of responding (DRL) task (interpreted as impaired response inhibition) at 0-5, 11-15, and 16-20 min at 9-11 y PFHxS and overall test performance (money earned on DRL task) at 9-11 y PFHxS and Brief Mood Introspection Scale mood dimension at 9-11 y PFOA and inter-response time at 0-5, 6-10, 11-15, and 16-20 min at 9-11 y PFOA and overall test performance at 9-11 y PFOA and Brief Mood Introspection Scale mood dimension at 9-11 y PFOS and inter-response time at 0-5, 6-10, and 16-20 min at 9-11 y PFOS and overall test performance at 9-11 y PFOS and Brief Mood Introspection Scale mood dimension at 9-11 y PFOSA and inter-response time at 0-5, 11-15, and 16-20 min at 9-11 y PFOSA and overall test performance at 9-11 y PFOSA and Brief Mood Introspection Scale mood dimension at 9-11 y PFNA and inter-response time at 0-5, 11-15, and 16-20 min at 9-11 y PFNA and overall test performance at 9-11 y PFNA and Brief Mood Introspection Scale mood dimension at 9-11 y PFDA and inter-response time at 0-5 and 16-20 min at 9-11 y PFDA and Brief Mood Introspection Scale mood dimension at 9-11 y ΣPFAS and inter-response time at 0-5 min at 9-11 y ΣPFAS and overal test performance at 9-11 y ΣPFAS and Brief Mood Introspection Scale mood dimension at 9-11 y
Neurodevelopment	Hoffman 2010	Cross-sectional	General community (United States)	Children	571	PFHxS PFOA PFOS PFNA	PFHxS and greater risk of ADHD at 12-15 years PFOA and greater risk fo ADHD at 12-15 years PFOS and greater risk of ADHD at 12-15 years	PFNA and ADHD at 12-15 years

Table 12. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurodevelopmental outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurodevelopment	Hoyer 2015a	Prospective cohort	Fishing/general community (Greenland; Kharkiv, Ukraine; Warsaw, Poland)	Children	1,106	PFOA PFOS	PFOA and greater Strengths and Difficulties Questionnaire hyperactivity score at 5-9 y (total, Greenland) PFOA and greater risk of abnormal Strengths and Difficulties Questionnaire total score at 5-9 y (total) PFOA and greater risk of abnormal Strengths and Difficulties Questionnaire hyperactivity score at 5-9 y (total, Greenland) PFOA and greater risk of abnormal Strengths and Difficulties Questionnaire prosocial behavior score at 5-9 y (Ukraine) PFOS and greater Strengths and Difficulties Questionnaire total score at 5-9 y (Greenland) PFOS and lower risk of abnormal Strengths and Difficulties Questionnaire emotional symptoms score at 5-9 y (Ukraine)	PFOA and Developmental Coordination Disorder Questionnaire 2007 total score at 5-9 y PFOA and Strengths and Difficulties Questionnaire total score at 5-9 y PFOA and Strengths and Difficulties Questionnaire hyperactivity score at 5-9 y (Ukraine, Poland) PFOA and abnormal Strengths and Difficulties Questionnaire total score at 5-9 y (Greenland, Ukraine) PFOA and abnormal Strengths and Difficulties Questionnaire hyperactivity score at 5-9 y (Ukraine) PFOA and abnormal Strengths and Difficulties Questionnaire emotional symptoms score at 5-9 y PFOA and abnormal Strengths and Difficulties Questionnaire conduct problems score at 5-9 y PFOA and abnormal Strengths and Difficulties Questionnaire peer problems score at 5-9 y PFOA and abnormal Strengths and Difficulties Questionnaire prosocial behavior score at 5-9 y (total, Greenland) PFOS and Developmental Coordination Disorder Questionnaire 2007 total score at 5-9 y PFOS and Strengths and Difficulties Questionnaire total score at 5-9 y (total, Ukraine, Poland) PFOS and Strengths and Difficulties Questionnaire hyperactivity score at 5-9 y PFOS and abnormal Strengths and Difficulties Questionnaire total score at 5-9 y PFOS and abnormal Strengths and Difficulties Questionnaire hyperactivity score at 5-9 y PFOS and abnormal Strengths and Difficulties Questionnaire emotional symptoms score at 5-9 y (total, Greenland) PFOS and abnormal Strengths and Difficulties Questionnaire conduct problems score at 5-9 y PFOS and abnormal Strengths and Difficulties Questionnaire peer problems score at 5-9 y PFOS and abnormal Strengths and Difficulties Questionnaire prosocial behavior score at 5-9 y
Neurodevelopment	Jeddy 2017	Prospective cohort	General community (Avon, United Kingdom)	Toddlers	432	PFHxS PFOA PFOS PFNA	PFHxS and greater verbal comprehension score on MacArthur Communicative Development Inventories at 15 months (mothers 25-30 y) PFHxS and greater nonverbal communication score at 15 months (mothers 25-30 y) PFOA and greater verbal comprehension score at 15 months (mothers > 30 y) PFOA and lower vocabulary comprehension and production score at 15 months (mothers < 25 y) PFOA and lower intelligibility score on MacArthur Communicative Development Inventories at 38 months (overall, mothers > 30 y) PFOA and greater communicative score at 38 months (mothers < 25 y) PFOS and greater verbal comprehension score at 15 months (overall, mothers > 30 y) PFOS and lower vocabulary comprehension and production score at 15 months (mothers < 25 y) PFOS and lower language score at 38 months (overall) PFOS and lower intelligibility score at 38 months (overall, mothers > 30 y) PFOS and greater intelligibility score at 38 months (mothers < 25 y) PFNA and greater verbal comprehension score at 15 months (overall, mothers > 30 y) PFNA and greater vocabulary comprehension and production at 15 months (mothers > 30 y) PFNA and greater social developmental score at 15 months (mothers > 30 y) PFNA and lower language score at 38 months (mothers 25-30 y)	PFHxS and verbal comprehension score (overall, mothers < 25 and > 30 y), vocabulary comprehension and production score (all groups), nonverbal communication score (overall, mothers < 25 and > 30 y), and social developmental score (all groups)on MacArthur Communicative Development Inventories at 15 months PFHxS and language score (all groups), intelligibility score (all groups), and communicative scores (all groups) on MacArthur Communicative Development Inventories at 38 months PFOA and verbal comprehension score (overall, mothers < 25 and 25-30 y), vocabulary comprehension and production score (overall, mothers 25-30 and > 30 y), nonverbal communication score (all groups), and social developmental score (all groups) at 15 months PFOA and language score (all groups), intelligibility score (mothers < 25 and 25-30 y), and communicative score (overall, mothers 25-30 and > 30 y) at 38 months PFOS and verbal comprehension score (mothers < 25 and 25-30 y), vocabulary comprehension and production score (overall, mothers 25-30 and > 30 y), nonverbal communication score (all groups), and social developmental score (all groups) at 15 months PFOS and language score (mothers < 25, 25-30, and > 30 y), intelligibility score (mothers 25-30 y), and communicative score (all groups) at 38 months PFNA and verbal comprehension score (mothers < 25 and 25-30 y), vocabulary comprehension and production score (overall, mothers < 25 and 25-30 y), nonverbal communication score (all groups), and social developmental score (overall, mothers < 25 and 25-30 y) at 15 months PFNA and language score (overall, mothers < 25 and > 30 y), intelligibility score (overall, mothers 25-30 and > 30 y), and communicative score (overall, mothers 25-30 and > 30 y) at 38 months

Table 12. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurodevelopmental outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurodevelopment	Lien 2016	Prospective cohort	General community (Taiwan)	Children	282	PFOA PFOS PFNA PFunDA	PFOA and lower inattention score (50th-74th and 75th-89th pctls), hyperactivity/impulsivity score (50th-74th pctl), and oppositional defiant disorder score (50th-74th and 75th-89th pctls) on Swanson, Nolan, and Pelham IV scale at 7 y (no trends) PFOA and lower internalizing problems score (50th-74th pctl) and total problems score (50th-74th pctl) on Child Behavior Checklist at 7 y (no trends) PFOA and lower emotional symptoms score (≥ 90th pctl), conduct problems score (50th-74th and 75th-89th pctls), peer problems score (50th-74th pctl), total difficulties score (50th-74th pctl), and prosocial behavior score (75th-89th pctl) on Strengths and Difficulties Questionnaire at 7 y (no trends) PFOS and greater prosocial behavior score (≥ 90th pctl) on Strengths and Difficulties Questionnaire at 7 y (no trend) PFNA and lower inattention score (≥ 90th pctl, trend), hyperactivity/impulsivity score (≥ 90th pctl, no trend), and oppositional defiant disorder score (75th-89th and ≥ 90th pctls, trend) on Swanson, Nolan, and Pelham IV scale PFNA and lower hyperactivity/inattention score (trend) on Strengths and Difficulties Questionnaire at 7 y PFunDA and lower inattention score (75th-89th pctl), hyperactivity/impulsivity score (75th-89th pctl), and oppositional defiant disorder score (75th-89th pctl) on Swanson, Nolan, and Pelham IV scale at 7 y (no trends) PFunDA and lower internalizing problems score (75th-89th pctl) on Child Behavior Checklist at 7 y (no trend) PFunDA and lower peer problems score (75th-89th pctl) and greater prosocial behavior score (75th-89th pctl) on Strengths and Difficulties	PFOA and externalizing problems score on Child Behavior Checklist at 7 y PFOA and hyperactivity/inatttention score on Strengths and Difficulties Questionnaire at 7 y PFOS and inattention score, hyperactivity/impulsivity score, and oppositional defiant disorder score on Swanson, Nolan, and Pelham IV scale at 7 y PFOS and internalizing problems score, externalizing problems score, and total problems score on Child Behavior Checklist at 7 y PFOS and emotional symptoms score, conduct problems score, hyperactivity/inattention score, peer problems score, and total difficulties score on Strengths and Difficulties Questionnaire at 7 y PFNA and internalizing problems score, externalizing problems score, and total problems score on Child Behavior Checklist at 7 y PFNA and emotional symptoms score, conduct problems score, peer problems score, and total difficulties score on Strengths and Difficulties Questionnaire at 7 y PFunDA and externalizing problems score and total problems score on Child Behavior Checklist at 7 y PFunDA and emotional symptoms score, conduct problems score, hyperactivity/inattention score, and total difficulties score on Strengths and Difficulties Questionnaire at 7 y
Neurodevelopment	Liew 2014	Nested prospective case-control	General community (Denmark)	Infants, children	156 cases, 550 controls	PFHxS PFHpS PFOA PFOS PFNA PFDA	PFOA and greater risk of congenital cerebral palsy (boys, boys born at term) PFOS and greater risk of congenital cerebral palsy (boys, boys born at term)	PFHxS and congenital cerebral palsy PFHpS and congenital cerebral palsy PFOA and congenital cerebral palsy (girls, girls born at term) PFOS and congenital cerebral palsy (girls, girls born at term) PFNA and congenital cerebral palsy PFDA and congenital cerebral palsy

Table 12. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurodevelopmental outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurodevelopment	Liew 2015	Nested prospective case-control	General community (Denmark)	Children	220 ADHD cases, 220 autism cases, 550 controls	PFHxS PFHpS PFOA PFOS PFNA PFDA	PFHxS and greater risk of childhood autism (quartiles 2 and 3, not 4; not continuous) PFHpS and lower risk of childhood ADHD (quartile 2, not 3 or 4; not continuous) PFOA and greater risk of childhood ADHD (quartiles 3 and 4; not continuous) PFNA and greater risk of childhood ADHD (quartiles 2, 3, 4; not continuous) PFDA and lower risk of childhood ADHD (quartiles 2 and 4, not 3; not continuous)	PFHxS and childhood ADHD PFHpS and childhood autism PFOA and childhood autism PFOS and childhood ADHD PFOS and childhood autism PFNA and childhood autism PFDA and childhood autism
Neurodevelopment	Ode 2014	Nested prospective case-control	General community (Malmö, Sweden)	Children	206 cases, 206 controls	PFOA PFOS PFNA	None	PFOA and childhood ADHD PFOS and childhood ADHD PFNA and childhood ADHD
Neurodevelopment	Oulhote 2016	Prospective cohort and cross-sectional	Fishing community (Faroe Islands)	Children	539	PFHxS PFOA PFOS PFNA PFDA ΣPFAS (PFHxS+PFOA+PFOS+PFNA+PFDA)	PFOA at 5 y and greater peer relationship problems score on Strengths and Difficulties Questionnaire at 7 y PFOA at 5 y and greater autism screening score on Strengths and Difficulties Questionnaire at 7 y PFOA at 5 y and greater internalizing problems score on Strengths and Difficulties Questionnaire at 7 y PFOA at 5 y and greater total difficulties score on Strengths and Difficulties Questionnaire at 7 y PFNA at 5 y and greater externalizing problems score on Strengths and Difficulties Questionnaire at 7 y PFNA at 5 y and greater total difficulties score on Strengths and Difficulties Questionnaire at 7 y PFDA at 5 y and greater externalizing problems score on Strengths and Difficulties Questionnaire at 7 y PFDA at 5 y and greater total difficulties score on Strengths and Difficulties Questionnaire at 7 y ΣPFAS postnatally and greater total difficulties score on Strengths and Difficulties Questionnaire at 7 y	PFHxS or PFOS prenatally, at 5 y, and at 7 y and conduct problems score, emotional symptoms score, hyperactivity/inattention score, peer relationship problems score, prosocial behavior score, autism screening score, externalizing problems score, internalizing problems score, and total difficulties score on Strengths and Difficulties Questionnaire at 7 y PFOA prenatally, at 5 y, and at 7 y and conduct problems score, emotional symptoms score, hyperactivity/inattention score, prosocial behavior score, and externalizing problems score at 7 y PFOA prenatally and at 7 y and peer relationship problems score, autism screening score, internalizing problems score, and total difficulties score at 7 y PFNA prenatally, at 5 y, and at 7 y and conduct problems score, emotional symptoms score, hyperactivity/inattention score, peer relationship problems score, prosocial behavior score, autism screening score, and internalizing problems score at 7 y PFNA prenatally and at 7 y and externalizing problems score and total difficulties score at 7 y PFDA prenatally, at 5 y, and at 7 y and conduct problems score, emotional symptoms score, hyperactivity/inattention score, peer relationship problems score, prosocial behavior score, autism screening score, and internalizing problems score at 7 y PFDA prenatally and at 7 y and externalizing problems score and total difficulties score at 7 y ΣPFAS prenatally and postnatally and conduct problems score, emotional symptoms score, hyperactivity/inattention score, peer relationship problems score, autism screening score, externalizing problems score, and internalizing problems score at 7 y ΣPFAS prenatally and total difficulties score at 7 y
Neurodevelopment	Quaak 2016	Prospective cohort	General community (Zwolle, Netherlands)	Toddlers	59	PFOA PFOS ΣPFAS (PFHxS+PFHpS+PFOA+PFOS+PFNA+PFDA+PFunDA)	PFOA and lower externalizing problem scale on Child Behavior Checklist at 18 months (boys) ΣPFAS and lower externalizing problem scale on Child Behavior Checklist at 18 months (all; girls tertile 2, not 3)	PFOA and attention deficit hyperactivity scale of Child Behavior Checklist at 18 months PFOA and externalizing problem scale of Child Behavior Checklist at 18 months (all, girls) PFOS and attention deficit hyperactivity scale of Child Behavior Checklist at 18 months PFOS and externalizing problem scale of Child Behavior Checklist at 18 months (all, girls, boys) ΣPFAS and attention deficit hyperactivity scale of Child Behavior Checklist at 18 months ΣPFAS and externalizing problem scale of Child Behavior Checklist at 18 months (boys)

Table 12. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurodevelopmental outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurodevelopment	Stein 2011	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Children	10,546 3,571 12-15 years	PFHxS PFOA PFOS PFNA	PFHxS and greater risk of ADHD diagnosis at 5-18 years PFHxS and greater risk of ADHD diagnosis+medication at 5-18 years PFOA and lower risk of ADHD diagnosis at 5-18 years PFOA and lower risk of ADHD diagnosis+medication at 5-18 years PFOS and lower risk of learning problem at 5-18 years (quartiles 2 and 3, not 4) PFOS and lower risk of learning probem at 12-15 years PFNA and lower risk of learning problem at 5-18 years PFNA and lower risk of learning probem at 12-15 years	PFHxS and learning problem at 5-18 years PFHxS and ADHD diagnosis at 12-15 years PFHxS and ADHD diagnosis+medication at 12-15 years PFHxS and learning problem at 12-15 years PFOA and learning problem at 5-18 years PFOA and ADHD diagnosis at 12-15 years PFOA and ADHD diagnosis+medication at 12-15 years PFOA and learning problem at 12-15 years PFOS and ADHD diagnosis at 5-18 years PFOS and ADHD diagnosis+medication at 5-18 years PFOS and ADHD diagnosis at 12-15 years PFOS and ADHD diagnosis+medication at 12-15 years PFNA and ADHD diagnosis at 5-18 years PFNA and ADHD diagnosis+medication at 5-18 years PFNA and ADHD diagnosis at 12-15 years PFNA and ADHD diagnosis+medication at 12-15 years
Neurodevelopment	Stein 2013	Prospective and retrospective cohort	Contaminated community (Mid-Ohio Valley)	Children	320	PFOA	PFOA estimated in utero and greater full scale IQ score (Wechsler Abbreviated Scale of Intelligence) at 6-12 years (quartile 4, no trend) PFOA estimated in utero and greater numerical operations standard score (Wechsler Individual Achievement Test-II) at 6-12 years (overall quartile 3, not 4, no trend) PFOA estimated in utero and lower ADHD clinical confidence index (Connors' Continuous Performance Test-II) at 6-12 years PFOA measured in childhood and greater numerical operations standard score (Wechsler Individual Achievement Test-II) at 6-12 years (boys) PFOA measured in childhood and lower ADHD clinical confidence index (Connors' Continuous Performance Test-II) at 6-12 years (trend, not quartiles)	PFOA estimated in utero and verbal IQ score, performance IQ score (Wechsler Abbreviated Scale of Ingelligence); word reading/pseudoword decoding standard score (Wechsler Individual Achievement Test-II); comprehension of instructions scaled score, design copying total scaled score, narrative memory free and cued recall scaled score, word generation semantic/initial letter scaled score mean (NEPSY-II); omissions T-score, commissions T-score, and hit reaction time T-score (Connors' Continuous Performance Test-II) at 6-12 years PFOA measured in childhood and full scale IQ score, verbal IQ score, performance IQ score (Wechsler Abbreviated Scale of Ingelligence); word reading/pseudoword decoding standard score, numerical operations standard score (overall, girls) (Wechsler Individual Achievement Test-II); comprehension of instructions scaled score, design copying total scaled score, narrative memory free and cued recall scaled score, word generation semantic/initial letter scaled score mean (NEPSY-II); omissions T-score, commissions T-score, and hit reaction time T-score (Connors' Continuous Performance Test-II) at 6-12 years
Neurodevelopment	Stein 2014a	Prospective cohort	Contaminated community (Mid-Ohio Valley)	Children	321	PFOA	PFOA and lower mother survey score on Behavior Rating Inventory of Executive Function (BRIEF) Global Executive Composite at 6-12 years (boys) PFOA × sex interaction and mother survey score on BRIEF Global Executive Composite at 6-12 years PFOA and greater mother survey score on Conners' ADHD DSM-IV Scales-Revised at 6-12 years (girls) PFOA × sex interaction and mother survey score on Conners' ADHD DSM-IV Scales-Revised at 6-12 years PFOA and lower teacher survey score on Conners' ADHD DSM-IV Scales-Revised at 6-12 years (overall, boys) PFOA and greater mother survey score on Behavior Assessment System for Children-2 (BASC) Behavioral Symptom Index at 6-12 years (girls) PFOA × sex interaction and mother survey score on BASC Behavioral Symptom Index at 6-12 years	PFOA and mother survey score on Behavior Rating Inventory of Executive Function (BRIEF) Global Executive Composite at 6-12 years (overall, girls) PFOA and teacher survey score on BRIEF Global Executive Composite at 6-12 years (overall, boys, girls) PFOA and mother survey score on Conners' ADHD DSM-IV Scales-Revised at 6-12 years (overall, boys) PFOA and teacher survey score on Conners' ADHD DSM-IV Scales-Revised at 6-12 years (girls) PFOA and mother survey score on Behavior Assessment System for Children-2 Behavioral Symptom Index at 6-12 years (overall, boys) PFOA and teacher survey score on Behavior Assessment System for Children-2 Behavioral Symptom Index at 6-12 years (overall, boys, girls)

Table 12. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurodevelopmental outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurodevelopment	Strom 2014	Prospective cohort	General community (Aarhus, Denmark)	Children	876	PFOA PFOS	None	PFOA and ADHD in childhood up to 22 years PFOA and depression in childhood up to 22 years PFOA and scholastic achievement in childhood up to 22 years PFOS and ADHD in childhood up to 22 years PFOS and depression in childhood up to 22 years PFOS and scholastic achievement in childhood up to 22 years
Neurodevelopment	Vuong 2016	Prospective cohort	General community (Cincinnati, Ohio)	Children	256	PFHxS PFOA PFOS PFNA PFDA	PFHxS and greater risk of global executive function score ≥ 60 on parent-rated Behavior Rating Inventory of Executive Function (BRIEF) at 5 and/or 8 y PFHxS and greater risk of monitor subscale score ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFOS and greater behavioral regulation index score on parent-rated BRIEF at 5 and/or 8 y PFOS and greater metacognition index score on parent-rated BRIEF at 5 and/or 8 y PFOS and greater global executive function score on parent-rated BRIEF at 5 and/or 8 y PFOS and greater risk of global executive function score ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFOS and greater risk of inhibit, working memory, plan/organize, and monitor subscale scores ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFNA and greater risk of inhibit subscale score ≥ 60 on parent-rated BRIEF at 5 and/or 8 y	PFHxS and behavioral regulation index score, metacognition score, and global executive function score on parent-rated Behavior Rating Inventory of Executive Function (BRIEF) at 5 and/or 8 y PFHxS and behavioral regulation index score and metacognition score ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFHxS and inhibit, shift, emotional control, working memory, plan/organize, initiate, and organization of materials subscale scores ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFOA and behavioral regulation index score, metacognition score, and global executive function score on parent-rated BRIEF at 5 and/or 8 y PFOA and behavioral regulation index score, metacognition score, and global executive function score ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFOA and inhibit, shift, emotional control, working memory, plan/organize, initiate, organization of materials, and monitor subscale scores ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFOS and behavioral regulation index score and metacognition score ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFOS and shift, emotional control, initiate, and organization of materials subscale scores ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFNA and behavioral regulation index score, metacognition score, and global executive function score on parent-rated BRIEF at 5 and/or 8 y PFNA and behavioral regulation index score, metacognition score, and global executive function score ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFNA and shift, emotional control, working memory, plan/organize, initiate, organization of materials, and monitor subscale scores ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFDA and behavioral regulation index score, metacognition score, and global executive function score on parent-rated BRIEF at 5 and/or 8 y PFDA and behavioral regulation index score, metacognition score, and global executive function score ≥ 60 on parent-rated BRIEF at 5 and/or 8 y PFDA and inhibit, shift, emotional control, working memory, plan/organize, initiate, organization of

Table 12. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurodevelopmental outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurodevelopment	Wang 2015	Prospective cohort	General community (central Taiwan)	Children	120	PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA	PFNA and lower verbal IQ on Wechsler Preschool and Primary Scale of Intelligence-Revised at 5 years PFunDA and lower performance IQ on Wechsler Preschool and Primary Scale of Intelligence-Revised at 5 years	PFHxS and full scale, verbal, and performance IQ on Wechsler Preschool and Primary Scale of Intelligence-Revised at 5 years PFHxS and full scale, verbal, and performance IQ on Wechsler Preschool and Primary Scale of Intelligence-Revised at 8 years PFOA and full scale, verbal, and performance IQ on Wechsler at 5 years PFOA and full scale, verbal, and performance IQ on Wechsler at 8 years PFOS and full scale, verbal, and performance IQ on Wechsler at 5 years PFOS and full scale, verbal, and performance IQ on Wechsler at 8 years PFNA and full scale, verbal, and performance IQ on Wechsler at 5 years PFNA and full scale and performance IQ on Wechsler at 8 years PFDA and full scale, verbal, and performance IQ on Wechsler at 5 years PFDA and full scale, verbal, and performance IQ on Wechsler at 8 years PFunDA and full scale and verbal IQ on Wechsler at 5 years PFunDA and full scale, verbal, and performance IQ on Wechsler at 8 years PFdoDA and full scale, verbal, and performance IQ on Wechsler at 5 years PFdoDA and full scale, verbal, and performance IQ on Wechsler at 8 years

Table 13. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurological outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurological	Berk 2014	Cross-sectional	General community (United States)	Adults	15,140	PFBS PFHxS PFHpA PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA PFunDA PFdoDA	PFHxS and lower risk of significant depressive symptoms on Patient Health Questionnaire 9 PFOA and lower risk of significant depressive symptoms on Patient Health Questionnaire 9 PFNA and lower risk of significant depressive symptoms on Patient Health Questionnaire 9 PFDA and loewr risk of significant depressive symptoms on Patient Health Questionnaire 9	PFBS and significant depressive symptoms on Patient Health Questionnaire 9 PFHpA and significant depressive symptoms on Patient Health Questionnaire 9 PFOS and significant depressive symptoms on Patient Health Questionnaire 9 PFOSA and significant depressive symptoms on Patient Health Questionnaire 9 Me-PFOSA-AcOH and significant depressive symptoms on Patient Health Questionnaire 9 Et-PFOSA-AcOH and significant depressive symptoms on Patient Health Questionnaire 9 PFunDA and significant depressive symptoms on Patient Health Questionnaire 9 PFdoDA and significant depressive symptoms on Patient Health Questionnaire 9
Neurological	Gallo 2013	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Older adults	21,024	PFHxS PFOA PFOS PFNA	PFHxS and lower risk of short-term memory impairment (overall, patients without diabetes) PFHxS and lower risk of any memory impairment PFOA and lower risk of short-term memory impairment (overall, patients without diabetes) PFOA × diabetes and risk of short-term memory impairment PFOA and lower risk of any memory impairment PFOS and lower risk of short-term memory impairment (overall, patients without diabetes) PFOS and lower risk of any memory impairment PFNA and lower risk of short-term memory impairment (overall no trend, patients without diabetes) PFNA and lower risk of any memory impairment	PFHxS and short-term memory impairment (patients with diabetes) PFOA and short-term memory impairment (patients with diabetes) PFOS and short-term memory impairment (patients with diabetes) PFNA and short-term memory impairment (patients with diabetes)

Table 13. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurological outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurological	Power 2013	Cross-sectional	General community (United States)	Older adults	1,766	PFHxS PFOA PFOS PFNA	PFHxS and lower risk of limitation due to difficulty remembering or periods of confusion (unmedicated diabetics) PFOS and lower risk of limitation due to difficulty remembering or periods of confusion (unmedicated diabetics) PFNA and lower risk of limitation due to difficulty remembering or periods of confusion (unmedicated diabetics)	PFHxS and limitation due to difficulty remembering or periods of confusion (non-diabetics, diabetics, medicated diabetics) PFHxS and difficulties with activities of daily living due to senility PFHxS and Digit-Symbol Substitution Task performance PFOA and limitation due to difficulty remembering or periods of confusion (non-diabetics, diabetics, medicated diabetics, unmedicated diabetics) PFOA and difficulties with activities of daily living due to senility PFOA and Digit-Symbol Substitution Task performance PFOS and limitation due to difficulty remembering or periods of confusion (non-diabetics, diabetics, medicated diabetics) PFOS and difficulties with activities of daily living due to senility PFOS and Digit-Symbol Substitution Task performance PFNA and limitation due to difficulty remembering or periods of confusion (non-diabetics, diabetics, medicated diabetics) PFNA and difficulties with activities of daily living due to senility PFNA and Digit-Symbol Substitution Task performance

Table 13. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and neurological outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Neurological	Shrestha 2017	Cross-sectional	PCB-contaminated community (Hudson River region, New York)	Older adults	126	PFOA PFOS	PFOA and greater t-score on California Verbal Learning Test PFOA and lower perseverative error score on Wisconsin Card Sorting Test PFOA and lower perseverative response score on Wisconsin Card Sorting Test PFOS and lower visual reproduction delayed recall score on Wechsler Memory Scale PFOS and greater total score on Block Design Subtest Mediation by free T4 of PFOS and short and long delay free recall scores on California Verbal Learning Test Mediation by free and total T4 of PFOS and total score on Block Design Subtest (significant positive T4 effect estimate)	PFOA, PFOS and tests of memory and learning: t-score (PFOS), trial 1 score, short delay free recall, long delay free recall, semantic cluster ratio, learning slope, and perseverations on California Verbal Learning Test; logical memory immediate recall score, logical memory delayed recall score, visual reproduction immedicate recall score, and visual reproduction delayed recall score (PFOA) on Wechsler Memory Scale PFOA, PFOS and test of measures of attention: trail making test part A PFOA, PFOS and tests of executive function: Stroop color word test t-score, trail making test part B, perseverative error (PFOS) and perseverative response (PFOS on Wisconsin Card Sorting Test PFOA, PFOS and tests of visual and spatial function: Block Design Subtest total score (PFOA), digit symbol coding total score PFOA, PFOS and reaction time for dominant hand PFOA, PFOS and tests of affective state: Beck Depression Inventory total score, State-Trait Anxiety Inventory state anxiety and trait anxiety t-scores PFOA, PFOS and tests of motor function: dominant and non-dominant hand on Finger Tapping Test, dominant and non-dominant hand on Grooved Pegboard Test; dominant and non-dominant hand on Static Motor Steadiness Test number of contacts and total time touching Mediation by T4 of PFOA and any neuropsychological test scores Mediation by T4 of PFOS and neuropsychological test scores other than short and long delay free recall scores on California Verbal Learning Test and total score on Block Design Subtest

Table 14. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and pregnancy-related hypertension

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Pregnancy-related hypertension	Avanasi 2016a (reanalysis of Savitz 2012b)	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Women	10,149	PFOA	None	PFOA and preeclampsia (accounting for uncertainty in drinking-water PFOA concentrations)
Pregnancy-related hypertension	Avanasi 2016b (reanalysis of Savitz 2012b)	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Women	10,149	PFOA	PFOA and preeclampsia (accounting for geocoding uncertainty in residential and work addresses)	PFOA and preeclampsia (accounting for geocoding uncertainty in residential addresses)
Pregnancy-related hypertension	Avanasi 2016c (reanalysis of Savitz 2012b)	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Women	10,149	PFOA	None	PFOA and preeclampsia (accounting for uncertainty in water ingestion rates and pharmacokinetic parameters)
Pregnancy-related hypertension	Darrow 2013	Prospective cohort	Contaminated community (Mid-Ohio Valley)	Women	1330	PFOA PFOS	PFOA and greater risk of pregnancy-induced hypertension (total) PFOS and greater risk of pregnancy-induced hypertension (total, parous)	PFOS and pregnancy-induced hypertension (nulliparous) PFOA and pregnancy-induced hypertension (nulliparous and parous)
Pregnancy-related hypertension	Nolan 2010	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Women	1,548	PFOA	None	PFOA (Little Hocking Water Association only) and pregnancy hypertension PFOA (Little Hocking Water Association only) and eclampsia PFOA (partial Little Hocking Water Association) and pregnancy hypertension PFOA (partial Little Hocking Water Association) and eclampsia
Pregnancy-related hypertension	Savitz 2012a	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Women	11,737	PFOA	PFOA and preeclampsia (some models)	PFOA and preeclampsia (some models)
Pregnancy-related hypertension	Savitz 2012b	Retrospective case-control and cross-sectional	Contaminated community (Mid-Ohio Valley)	Women	8,353 (Study I) 4,547 (Study II)	PFOA	Study II: PFOA and pregnancy-induced hypertension (quintile 4 only or quintiles 3 and 4, not 5 (some models), not continuous)	Study I: PFOA and pregnancy-induced hypertension Study II PFOA and pregnancy-induced hypertension (some models)

Table 14. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and pregnancy-related hypertension

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Pregnancy-related hypertension	Starling 2014	Nested prospective case-control study	General community (Norway)	Women	466 cases 510 controls	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA	PFunDA and lower risk of preeclampsia	PFHxS and preeclampsia PFHpS and preeclampsia PFOA and preeclampsia PFOS and preeclampsia PFNA and preeclampsia PFDA and preeclampsia
Pregnancy-related hypertension	Stein 2009	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Newborns	5,262	PFOA PFOS	PFOS and greater risk of preeclampsia	PFOA and preeclampsia

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (fertility)	Bach 2015a	Prospective cohort	General community (Denmark)	Women	1,372	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA	None	PFHxS and fecundability ratio and infertility (nulliparous) PFHpS and fecundability ratio and infertility (nulliparous) PFOA and fecundability ratio and infertility (nulliparous) PFOS and fecundability ratio and infertility (nulliparous) PFNA and fecundability ratio and infertility (nulliparous) PFDA and fecundability ratio and infertility (nulliparous) PFunDA and fecundability ratio and infertility (nulliparous)
Reproductive, female (fertility)	Bach 2015b	Prospective cohort	General community (Denmark)	Women	1,601	PFOA PFOS	PFOA and lower fecundability ratio (overall, sample 2, parous overall, parous sample 1, parous sample, 2, nulliparous sample 2) PFOS and lower fecundability ratio (overall, sample 2, nulliparous overall, nulliparous sample 2)	PFOA and fecundability ratio (sample 1, nulliparous overall, nulliparous sample 1) PFOS and fecundability ratio (sample 1, parous overall, parous sample 1, nulliparous sample 1, parous sample 2)
Reproductive, female (pregnancy outcome)	Bae 2015	Prospective cohort	General community (Michigan and Texas)	Adults	233	PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	None	Maternal PFOA and ratio of male to female births Maternal PFOS and ratio of male to female births Maternal PFOSA and ratio of male to female births Maternal Me-PFOSA-AcOH and ratio of male to female births Maternal Et-PFOSA-AcOH and ratio of male to female births Maternal PFNA and ratio of male to female births Maternal PFDA and ratio of male to female births

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (hormones)	Barrett 2015	Cross-sectional	General community (Tromso, Norway)	Women	178	PFHxS PFOA PFOS PFOSA PFNA PFDA PFunDA	PFOS and lower estradiol level during follicular phase (total, nulliparous) PFOS and lower progesterone level during luteal phase (nulliparous)	PFHxS and estradiol during follicular phase PFHxS and progesterone during luteal phase PFOA and estradiol during follicular phase PFOA and progesterone during luteal phase PFOS and estradiol during follicular phase (parous) PFOS and progesterone during luteal phase (total, parous) PFOSA and estradiol during follicular phase PFOSA and progesterone during luteal phase PFNA and estradiol during follicular phase PFNA and progesterone during luteal phase PFDA and estradiol during follicular phase PFDA and progesterone during luteal phase PFunDA and estradiol during follicular phase PFunDA and progesterone during luteal phase
Reproductive, female (endometriosis)	Buck Louis 2012	Cross-sectional	General community (Salt Lake City, Utah, and San Francisco area, California)	Women	473 operative sample (190 endometriosis) 127 population sample (14 endometriosis)	PFHxS PFOA PFOS PFNA PFDA	None	PFHxS and risk of endometriosis PFOA and risk of endometriosis PFOS and risk of endometriosis PFNA and risk of endometriosis PFDA and risk of endometriosis
Reproductive, female (fertility)	Buck Louis 2013	Prospective cohort	General community (Michigan and Texas)	Women	501	PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	PFOSA (female partner) and significantly lower fecundity odds ratio	PFOA (male or female partner) and fecundity odds ratio PFOS (male or female partner) and fecundity odds ratio PFOSA (male partner) and fecundity odds ratio Me-PFOSA-AcOH (male or female partner) and fecundity odds ratio Et-PFOSA-AcOH (male or female partner) and fecundity odds ratio PFNA (male or female partner) and fecundity odds ratio PFDA (male or female partner) and fecundity odds ratio

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (pregnancy outcome)	Buck Louis 2016	Prospective cohort	General community (Michigan and Texas)	Women	344	PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	Me-PFOSA-AcOH and lower risk of pregnancy loss (continuous, not tertiles) PFNA and lower risk of pregnancy loss (tertiles, not continuous)	PFOA and risk of pregnancy loss PFOS and risk of pregnancy loss PFOSA and risk of pregnancy loss Et-PFOSA-AcOH and risk of pregnancy loss PFDA and risk of pregnancy loss
Reproductive, female (endometriosis)	Campbell 2016	Cross-sectional	General community (United States)	Women	749	PFHxS PFOA PFOS PFNA	PFOA and greater risk of endometriosis PFOS and greater risk of endometriosis PFNA and greater risk of endometriosis	PFHxS and risk of endometriosis
Reproductive, female (fertility, breastfeeding)	Cariou 2015	Cross-sectional	General community (Toulouse, France)	Women	100	PFHxS PFOA PFOS PFNA	PFHxS and lower number of pregnancies PFOA and shorter duration of breastfeeding	PFHxS and time to pregnancy > 2 years PFHxS and duration of breastfeeding PFOA and number of pregnancies PFOA and time to pregnancy > 2 years PFOS and number of pregnancies PFOS and time to pregnancy > 2 years PFOS and duration of breastfeeding PFNA and number of pregnancies PFNA and time to pregnancy > 2 years PFNA and duration of breastfeeding
Reproductive, female (fertility, endometriosis)	Caserta 2013	Case-control	General community (Rome, Ferrara, and Sora, Italy)	Women	48 cases 13 controls	PFOA PFOS	None	PFOA and infertility, endometriosis infertility, or other causes of infertility PFOS and infertility, endometriosis infertility, or other causes of infertility

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (menstruation)	Christensen 2011	Prospective nested case-control	General community (Avon, United Kingdom)	Adolescent girls	218 cases 230 controls	PFHxS PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA Σsulfonamide esters (Me-+Et-PFOSA-AcOH) Σsulfonates (PFHxS+PFOS) Σcarboxylates (PFOA+PFNA)	None	PFHxS and risk of earlier menarche (< 11.5 years) PFOA and risk of earlier menarche (< 11.5 years) PFOS and risk of earlier menarche (< 11.5 years) PFOSA and risk of earlier menarche (< 11.5 years) Me-PFOSA-AcOH and risk of earlier menarche (< 11.5 years) Et-PFOSA-AcOH and risk of earlier menarche (< 11.5 years) PFNA and risk of earlier menarche (< 11.5 years) PFDA and risk of earlier menarche (< 11.5 years) Σsulfonamide esters and risk of earlier menarche (< 11.5 years) Σsulfonates and risk of earlier menarche (< 11.5 years) Σcarboxylates and risk of earlier menarche (< 11.5 years)
Reproductive, female (fertility, hormones)	Crawford 2017	Cross-sectional	General community (North Carolina)	Women	99	PFHxS PFOA PFOS PFNA ΣPFAS (PFHxS+PFOA+PFOS+PFNA)	None	PFHxS and fecundability ratio and anti-Mullerian hormone PFOA and fecundability ratio and anti-Mullerian hormone PFOS and fecundability ratio and anti-Mullerian hormone PFNA and fecundability ratio and anti-Mullerian hormone ΣPFAS and fecundability ratio and anti-Mullerian hormone
Reproductive, female (pregnancy outcome)	Darrow 2014	Prospective cohort	Contaminated community (Mid-Ohio Valley)	Women	1,129 (1,438 pregnancies)	PFOA PFOS	PFOS and greater risk of miscarriage (first pregnancy; first/non-recent pregnancy quintile 3, not 4 or 5; nulligravid at enrollment quintile 2, not 3, 4, or 5; nulliparous at enrollment quintile 2, not 3, 4, or 5)	PFOA and risk of miscarriage (overall, first pregnancy, first pregnancy/non-recent pregnancy, nulligravid at enrollment, nulliparous at enrollment, parous at enrollment) PFOS and risk of miscarriage (overall, parous at enrollment)
Reproductive, female (menstruation)	Dhingra 2016a	Prospective cohort and retrospective cohort	Contaminated community (Mid-Ohio Valley)	Women	8,759 retrospective 3,334 prospective	PFOA	Cumulative PFOA and greater risk of natural menopause (retrospective with hysterectomies excluded and 5-year lagged quintiles, no trend; retrospective with hysterectomies included) Annual PFOA and greater risk of menopause (retrospective with hysterectomies included) Baseline serum PFOA and lower risk of menopause (prospective with hysterectomies included, quintile 3, not 4 or 5)	Cumulative PFOA and [age at] natural menopause (retrospective, prospective; hysterectomies censored or excluded; retrospective with 10-, 15-, or 20-year lag) Baseline serum PFOA and [age at] natural menopause (prospective, hysterectomies censored or excluded)

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (menstruation)	Dhingra 2017	Retrospective cohort and cross-sectional	Contaminated community (Mid-Ohio Valley)	Women	9,192	PFOA	Measured serum PFOA and greater odds of menopause More years since menopause (up to 7 years) and greater measured serum PFOA level	Modeled serum or cumulative PFOA and menopause
Reproductive, female (fertility)	Ding 2014	Prospective cohort (outcome-dependent sampling)	General community (Norway)	Women	910	PFOA	PFOA and greater risk of subfecundity (time to pregnancy > 12 or > 3 months)	PFOA and risk of subfecundity (time to pregnancy > 24 months)
Reproductive, female (fertility)	Fei 2009	Retrospective cohort	General community (Norway)	Women	1,240	PFOA PFOS	PFOA and greater risk of infertility PFOA and greater risk of subfecundity PFOS and greater risk of infertility PFOS and greater risk of subfecundity	None
Reproductive, female (breastfeeding)	Fei 2010b	Prospective cohort	General community (Norway)	Women	1,400	PFOA PFOS	PFOA and shorter duration of breastfeeding (overall, multiparous) PFOA and greater risk of weaning before 3 months (overall, multiparous) PFOA and greater risk of weaning before 6 months (overall, multiparous) PFOA and shorter duration of exclusive breastfeeding (overall, multiparous) PFOA and greater risk of terminating exclusive breastfeeding before 1 month (overall, multiparous) PFOA and greater risk of terminating exclusive breastfeeding before 4 months (overall, multiparous) PFOS and shorter duration of breastfeeding (overall, multiparous) PFOS and greater risk of weaning before 3 months (overall, multiparous) PFOS and greater risk of weaning before 6 months (overall, multiparous, primiparous) PFOS and shorter duration of exclusive breastfeeding (overall, multiparous) PFOS and greater risk of terminating exclusive breastfeeding before 1 month (multiparous) PFOS and greater risk of terminating exclusive breastfeeding before 4 months (overall, multiparous)	PFOA and duration of breastfeeding (primiparous) PFOA and risk of weaning before 3 months (primiparous) PFOA and risk of weaning before 6 months (primiparous) PFOA and duration of exclusive breastfeeding (primiparous) PFOA and risk of terminating exclusive breastfeeding before 1 month (primiparous) PFOA and risk of terminating exclusive breastfeeding before 4 months (primiparous) PFOS and duration of breastfeeding (primiparous) PFOS and risk of weaning before 3 months (primiparous) PFOS and duration of exclusive breastfeeding (primiparous) PFOS and risk of terminating exclusive breastfeeding before 1 month (overall, primiparous) PFOS and risk of terminating exclusive breastfeeding before 4 months (primiparous)
Reproductive, female (fertility)	Fei 2012	Retrospective cohort	General community (Norway)	Women	1,233	PFOA PFOS	PFOA and greater risk of infertility (overall, parous) PFOA and greater risk of subfecundity (overall, nulliparous, parous) PFOS and greater risk of infertility (overall, nulliparous, parous) PFOS and greater risk of subfecundity (overall, nulliparous, parous)	PFOA and risk of infertility (nulliparous)

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (fertility)	Governini 2011	Cross-sectional	General community (Siena, Italy)	Women	16	"PFCs"	PFCs in follicular fluid and lower oocyte fertilization rate PFCs in follicular fluid and lower number of embryos transferred	PFCs in follicular fluid and number of oocytes retrieved PFCs in follicular fluid and percentage of top quality oocytes
Reproductive, female (pregnancy outcome)	Grice 2007	Retrospective cohort	Occupational (Decatur, Alabama)	Women	263	PFOS	None	PFOS and stillbirth
Reproductive, female (hormones)	Itoh 2016	Prospective cohort	General community (Hokkaido, Japan)	Newborns	106	PFOA PFOS	PFOS and lower progesterone level (girls) PFOS and lower prolactin level (girls)	PFOA and estradiol (girls) PFOA and testosterone (girls) PFOA and testosterone:estradiol ratio (girls) PFOA and progesterone (girls) PFOA and sex hormone binding globulin (girls) PFOA and testosterone:sex hormone binding globulin ratio (girls) PFOA and prolactin (girls) PFOS and estradiol (girls) PFOS and testosterone (girls) PFOS and testosterone:estradiol ratio (girls) PFOS and sex hormone binding globulin (girls) PFOS and testosterone:sex hormone binding globulin ratio (girls)
Reproductive, female (pregnancy outcome)	Jensen 2015	Prospective case-control	General community (Odense, Denmark)	Women	56 cases 336 controls	PFHxS PFOA PFOS PFNA PFDA	PFNA and greater risk of miscarriage PFDA and greater risk of miscarriage	PFHxS and risk of miscarriage PFOA and risk of miscarriage PFOS and risk of miscarriage
Reproductive, female (fertility)	Jorgensen 2014	Retrospective cohort	Fishing/general community (Greenland; Kharkiv, Ukraine; Warsaw, Poland)	Women	938	PFHxS PFOA PFOS PFNA	PFOA and greater fecundability ratio (primiparous overall, primiparous Ukraine) PFNA and lower fecundability ratio (overall, Greenland) PFNA and greater risk of infertility (overall, Greenland) Male PFNA and lower fecundability ratio (Greenland)	PFHxS and fecundability ratio (all groups; primiparous women) PFHxS and risk of infertility (all groups; primiparous women) Male PFHxS and fecundability ratio (all groups) PFOA and fecundability ratio (all groups; primiparous Poland and Greenland) PFOA and risk of infertility (all groups; primiparous women) Male PFOA and fecundability ratio (all groups) PFOS and fecundability ratio (all groups; primiparous women) PFOS and risk of infertility (all groups; primiparous women) Male PFOS and fecundability ratio (all groups) PFNA and fecundability ratio (Poland, Ukraine; primiparous women) PFNA and risk of infertility (Poland, Ukraine; primiparous women) Male PFNA and fecundability ratio (Poland, Ukraine)

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (menstruation, hormones)	Knox 2011b	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Women	25,957	PFOA PFOS	PFOA and greater odds of menopause (> 42 to ≤ 51 y (no trend), > 51 to ≤ 65 y (no trend)) PFOS and greater odds of menopause (> 42 to ≤ 51 y (no trend), > 51 to ≤ 65 y) PFOS and lower estradiol level (> 42 to ≤ 51 y, > 51 to ≤ 65 y)	PFOA and odds of menopause (18 to ≤ 42 y) PFOA and estradiol (18 to ≤ 42 y, > 42 to ≤ 51 y, > 51 to ≤ 65 y) PFOS and odds of menopause (18 to ≤ 42 y) PFOS and estradiol (18 to ≤ 42 y)
Reproductive, female (menstruation, hormones)	Kristensen 2013	Prospective cohort	General community (Aarhus, Denmark)	Young women	337	PFOA PFOS	PFOA and later age at menarche	PFOA and menstrual cycle length PFOA and total testosterone PFOA and sex hormone binding globulin PFOA and free androgen index PFOA and dehydroepiandrosterone sulfate PFOA and follicle-stimulating hormone PFOA and luteinizing hormone PFOA and estradiol PFOA and anti-Mullerian hormone PFOA and number of cycles per ovary PFOS and age at menarche PFOS and menstrual cycle length PFOS and total testosterone PFOS and sex hormone binding globulin PFOS and free androgen index PFOS and dehydroepiandrosterone sulfate PFOS and follicle-stimulating hormone PFOS and luteinizing hormone PFOS and estradiol PFOS and anti-Mullerian hormone PFOS and number of cycles per ovary
Reproductive, female (fertility)	La Rocca 2014	Case-control	General community (Rome, Ferrara, and Sora, Italy)	Young women	110 cases 43 controls	PFOA PFOS	None	PFOA and infertility (total, metropolitan, urban, rural) PFOS and infertility (total, metropolitan, urban, rural)
Reproductive, female (hormones)	Lewis 2015	Cross-sectional	General community (United States)	Women, adolescent girls	257 (largest age-sex stratum)	PFHxS PFOA PFOS PFNA	None	PFHxS and testosterone (girls/women 12-<20, 20-<40, 40-<60, 60-80 y) PFOA and testosterone (girls/women 12-<20, 20-<40, 40-<60, 60-80 y) PFOS and testosterone (girls/women 12-<20, 20-<40, 40-<60, 60-80 y) PFNA and testosterone (girls/women 12-<20, 20-<40, 40-<60, 60-80 y)

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (menstruation, hormones)	Lopez-Espinosa 2011	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Girls, adolescent girls	2,903	PFOA PFOS	PFOA and later age at menarche PFOS and later age at menarche PFOS and later age at puberty (menarche or estradiol > 20 pg/mL)	PFOA and age at puberty (menarche or estradiol > 20 pg/mL)
Reproductive, female (hormones)	Lopez-Espinosa 2016	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Girls	1,123	PFHxS PFOA PFOS PFNA	PFOS and lower total testosterone level at 6-9 years	PFHxS and estradiol at 6-9 years PFHxS and total testosterone at 6-9 years PFOA and estradiol at 6-9 years PFOA and total testosterone at 6-9 years PFOS and estradiol at 6-9 years PFNA and estradiol at 6-9 years PFNA and total testosterone at 6-9 years
Reproductive, female (menstruation, fertility)	Lum 2017	Prospective cohort	General community (Michigan and Texas)	Women	483	PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	PFOA and shorter menstrual cycle length	PFOA and fecundity PFOS and menstrual cycle length and fecundity PFOSA and menstrual cycle length and fecundity Me-PFOSA-AcOH and menstrual cycle length and fecundity Et-PFOSA-AcOH and menstrual cycle length and fecundity PFNA and menstrual cycle length and fecundity PFDA and menstrual cycle length and fecundity
Reproductive, female (menstruation)	Lyngso 2014	Cross-sectional	Fishing/general community (Greenland; Kharkiv, Ukraine; Warsaw, Poland)	Women	1,623	PFOA PFOS	PFOA and greater risk of long menstrual cycle PFOA and longer menstrual cycle	PFOA and irregular menstrual cycle PFOA and short menstrual cycle PFOS and long menstrual cycle PFOS and irregular menstrual cycle PFOS and short menstrual cycle
Reproductive, female (hormones)	Maisonet 2015a	Prospective cohort	General community (Avon, United Kingdom)	Adolescent girls	72	PFHxS PFOA PFOS PFNA	PFHxS and greater total testosterone levels (tertile 3, no trend) PFOA and greater total testosterone levels (tertile 3, no trend) PFOS and greater total testosterone levels	PFHxS and sex hormone binding globulin PFOA and sex hormone binding globulin PFOS and sex hormone binding globulin PFNA and total testosterone PFNA and sex hormone binding globulin

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (fertility, hormones)	McCoy 2017	Cross-sectional	General community (South Carolina)	Women	34	PFHxS PFOA PFOS PFNA PFDA PFunDA ΣPFAS (PFHxS+PFOA+PFOS+PFNA+PFDA+PFuNDA)	PFHxS in follicular fluid and lower follicle count PFOS in plasma and lower estradiol level PFDA in follicular fluid and lower blast conversion PFunDA in follicular fluid and lower blast conversion PFunDA in plasma and lower blast conversion	PFHxS in follicular fluid and plasma and estradiol, follicle count (plasma), change in estradiol, change in follicle count, oocytes retrieved, percent fertilization, blast conversion, and pregnancy outcomes PFOA in follicular fluid and plasma and estradiol, follicle count, change in estradiol, change in follicle count, oocytes retrieved, percent fertilization, blast conversion, and pregnancy outcomes PFOS in follicular fluid and plasma and estradiol (follicular fluid), follicle count, change in estradiol, change in follicle count, oocytes retrieved, percent fertilization, blast conversion, and pregnancy outcomes PFNA in follicular fluid and plasma and estradiol, follicle count, change in estradiol, change in follicle count, oocytes retrieved, percent fertilization, blast conversion, and pregnancy outcomes PFDA in follicular fluid and plasma and estradiol, follicle count, change in estradiol, change in follicle count, oocytes retrieved, percent fertilization, blast conversion (plasma), and pregnancy outcomes PFunDA in follicular fluid and plasma and estradiol, follicle count, change in estradiol, change in follicle count, oocytes retrieved, percent fertilization, and pregnancy outcomes ΣPFAS in follicular fluid and plasma and estradiol, follicle count, change in estradiol, change in follicle count, oocytes retrieved, percent fertilization, blast conversion, and pregnancy outcomes

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (pregnancy outcome)	Nolan 2010	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Women	1,548	PFOA	PFOA (Little Hocking Water Association only) and lower risk of any labor and delivery complication PFOA (Little Hocking Water Association only) and greater risk of dysfunctional labor PFOA (Little Hocking Water Association only) and lower risk of other labor complications PFOA (Little Hocking Water Association only) and greater risk of maternal anemia PFOA (partial Little Hocking Water Association) and lower risk of other labor complications PFOA (partial Little Hocking Water Association) and lower risk of previous infant small for gestational age	PFOA (Little Hocking Water Association only) and congenital anomalies: any congenital anomaly, heart malformation, circulatory malformation, other congenital anomaly, anencephalus, spina bifida, tracheoesophageal fistula, omphalocele, cleft lip, polydactyly, Down syndrome, or club foot PFOA (Little Hocking Water Association only) and labor and delivery complications: febrile, meconium, membrane rupture, abruptio placenta, placenta previa, excessive bleeding, seizure, precipitous labor, prolonged labor, breech, cephalopelvic disporportion, cord prolapse, anesthetic complications, or fetal distress PFOA (Little Hocking Water Association only) and maternal risk factors: any maternal risk factor, hydramnios, incompetent cervix, previous infant ≥ 4,000 g, previous infant small for gestational age, uterine bleeding, or other maternal risk factor PFOA (partial Little Hocking Water Association) and congenital anomalies: any congenital anomaly, heart malformation, circulatory malformation, other congenital anomaly, anencephalus, spina bifida, tracheoesophageal fistula, omphalocele, cleft lip, polydactyly, Down syndrome, gastrointestinal anomaly, or urogenital anomaly PFOA (partial Little Hocking Water Association only) and labor and delivery complications: any labor and delivery complication, febrile, meconium, membrane rupture, abruptio placenta, placenta previa, excessive bleeding, seizure, precipitous labor, prolonged labor, dysfunctional labor, breech, cephalopelvic disporportion, cord prolapse, anesthetic complications, or fetal distress PFOA (partial Little Hocking Water Association) and maternal risk factors: any maternal risk factor, anemia, hydramnios, incompetent cervix, previous infant ≥ 4,000 g, uterine bleeding, or other maternal risk factor
Reproductive, female (fertility, pregnancy outcome)	Olsen 2004	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,311	PFOS	None	PFOS and episodes of care for fertility and infertility management PFOS and episodes of care for complicated pregnancy or delivery PFOS and episodes of care for preterm labor PFOS and episodes of care for normal or unspecified pregnancy or delivery PFOS and episodes of care for spontaneous abortion PFOS and episodes of care for pregnancy PFOS and episodes of care for congenital anomalies PFOS and episodes of care for perinatal disorders

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (breastfeeding)	Romano 2016	Prospective cohort	General community (Cincinnati, Ohio)	Women	336	PFHxS PFOA PFOS PFNA	PFOA and greater risk of stopping any breastfeeding by 3 months PFOA and greater risk of stopping any breastfeeding by 6 months PFOS and greater risk of stopping any breastfeeding by 3 months (quartile 3, not 4, no trend)	PFHxS and risk of stopping any breastfeeding by 3 months PFHxS and risk of stopping any breastfeeding by 6 months PFHxS and risk of stopping exclusive breastfeeding by 3 months PFOA and risk of stopping exclusive breastfeeding by 3 months PFOS and risk of stopping any breastfeeding by 6 months PFOS and risk of stopping exclusive breastfeeding by 3 months PFNA and risk of stopping any breastfeeding by 3 months PFNA and risk of stopping any breastfeeding by 6 months PFNA and risk of stopping exclusive breastfeeding by 3 months
Reproductive, female (fertility)	Rylander 2009	Cross-sectional	General community (Khanh Hoa province, southern central Vietnam)	Women	91	PFHxS PFHpS PFOA PFOS PFNA	None	PFHxS and parity PFHpS and parity PFOA and parity PFOS and parity PFNA and parity
Reproductive, female (pregnancy outcome)	Savitz 2012a	Case-control	Contaminated community (Mid-Ohio Valley)	Newborns	8,353 (Study I)	PFOA	None	PFOA and stillbirth
Reproductive, female (pregnancy outcome)	Savitz 2012b	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Newborns	11,737	PFOA	None	PFOA and miscarriage PFOA and stillbirth PFOA and birth defects
Reproductive, female (pregnancy outcome)	Stein 2009	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Newborns	1,845 PFOA 5,262 PFOS	PFOA PFOS	None	PFOA and miscarriage PFOA and brith defects PFOS and miscarriage PFOS and birth defects
Reproductive, female (pregnancy outcome)	Stein 2014b	Retrospective cohort	Contaminated community (Mid-Ohio Valley)	Newborns	10,262	PFOA	PFOA and greater risk of brain defects (interquartile range increase, not percentile categories)	PFOA and risk of gastrointestinal birth defects PFOA and risk of kidney birth defects PFOA and risk of craniofacial birth defects PFOA and risk of eye birth defects PFOA and risk of limb birth defects PFOA and risk of genitourinary birth defects PFOA and risk of heart birth defects

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (menstruation)	Taylor 2014	Cross-sectional	General community (United States)	Women	2,151	PFHxS PFOA PFOS PFNA	PFHxS and earlier age at menopause PFHxS and greater risk of hysterectomy PFOA and earlier age at menopause PFOA and greater risk of hysterectomy PFOS and earlier age at menopause (tertile 2, not 3; no trend) PFOS and greater risk of hysterectomy PFNA and earlier age at menopause PFNA and greater risk of hysterectomy	None
Reproductive, female (breastfeeding)	Timmermann 2017b	Prospective cohort	Fishing community (Faroe Islands)	Women	998	PFHxS PFOA PFOS PFNA PFDA	PFOA and shorter duration of breastfeeding (overall, primiparous, multiparous, older cohort, younger cohort) PFOA and shorter duration of exclusive breastfeeding (overall, multiparous, older cohort, younger cohort) PFOS and shorter duration of breastfeeding (overall, primiparous, multiparous, older cohort, younger cohort) PFOS and shorter duration of exclusive breastfeeding (overall, multiparous, younger cohort) PFNA and shorter duration of breastfeeding (overall, primiparous, multiparous, older cohort, younger cohort) PFNA and shorter duration of exclusive breastfeeding (overall) PFDA and shorter duration of breastfeeding (overall, primiparous, older cohort) PFDA and shorter duration of exclusive breastfeeding (primiparous, younger cohort)	PFHxS and duration of breastfeeding (overall, primiparous, multiparous, older cohort, younger cohort) PFHxS and duration of exclusive breastfeeding (overall, primiparous, multiparous, older cohort, younger cohort) PFOA and shorter duration of exclusive breastfeeding (primiparous) PFOS and shorter duration of exclusive breastfeeding (primiparous, older cohort) PFNA and shorter duration of exclusive breastfeeding (primiparous, multiparous, older cohort, younger cohort) PFDA and duration of breastfeeding (multiparous, younger cohort) PFDA and duration of exclusive breastfeeding (overall, multiparous, older cohort)

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (hormones)	Tsai 2015	Cross-sectional	General community (Taipei, Taiwan)	Adolescents, young adults	65 girls 12-17 y 265 women 18-30 y	PFOA PFOS PFNA PFunDA	PFOA and lower sex hormone binding globulin level (girls 12-17 y) PFOS and lower total testosterone level (girls 12-17 y) PFunDA and lower follicle-stimulating hormone level (girls 12-17 y)	PFOA and follicle-stimulating hormone, total testosterone, estrogen, luteinizing hormone, and free testosterone (girls 12-17 y, women 18-30 y) PFOA and sex hormone binding globulin (women 18-30 y) PFOS and sex hormone binding globulin, follicle-stimulating hormone, estrogen, luteinizing hormone, and free testosterone (girls 12-17 y, women 18-30 y) PFOS and total testosterone (women 18-30 y) PFNA and sex hormone binding globulin, follicle-stimulating hormone, total testosterone, estrogen, luteinizing hormone, and free testosterone (girls 12-17 y, women 18-30 y) PFunDA and sex hormone binding globulin, estrogen, total testosterone, luteinizing hormone, and free testosterone (girls 12-17 y, women 18-30 y) PFunDA and follicle-stimulating hormone (women 18-30 y)
Reproductive, female (fertililty)	Velez 2015	Retrospective cohort	General community (Canada)	Women	1,743	PFHxS PFOA PFOS	PFHxS and lower fecundability ratio PFHxS and greater risk of infertility PFOA and lower fecundability ratio PFOA and greater risk of infertility	PFOS and fecundability ratio PFOS and risk of infertility
Reproductive, female (fertililty)	Vestergaard 2012	Prospective cohort	General community (Denmark)	Women	222	PFHxS PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	PFHxS and greater fecundability ratio	PFHxS and risk of subfecundability PFOA and fecundability ratio and risk of subfecundability PFOS and fecundability ratio and risk of subfecundability PFOSA and fecundability ratio and risk of subfecundability Me-PFOSA-AcOH and fecundability ratio and risk of subfecundability Et-PFOSA-AcOH and fecundability ratio and risk of subfecundability PFNA and fecundability ratio and risk of subfecundability PFDA and fecundability ratio and risk of subfecundability

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (endometriosis)	Wang 2017a	Case-control	General community (Hangzhou, China)	Women	157 cases 178 controls	PFBS PFHxS PFHpA PFOA PFOS PFNA PFDA PFunDA PFDA PFdoDA	PFBS and greater risk of endometriosis-related infertility (overall; nulliparous women; women without other gynecologic pathology) PFHxS and lower risk of endometriosis-related infertility (overall) PFHpA and lower risk of endometriosis-related infertility (overall) PFOS and lower risk of endometriosis-related infertility (women without other gynecologic pathology) PFNA and lower risk of endometriosis-related infertility (overall; women without other gynecologic pathology) PFunDA and lower risk of endometriosis-related infertility (women without other gynecologic pathology) PFdoDA and lower risk of endometriosis-related infertility (women without other gynecologic pathology)	PFOA and risk of endometriosis-related infertility (overall) PFOS and risk of endometriosis-related infertility (overall) PFDA and risk of endometriosis-related infertility (overall) PFunDA and risk of endometriosis-related infertility (overall) PFDA and risk of endometriosis-related infertility (overall) PFdoDA and risk of endometriosis-related infertility (overall)
Reproductive, female (hormones)	White 2011	Cross-sectional	General community (North Carolina)	Women	34	PFOSA Me-PFOSA PFNA Other PFAS	PFOSA and greater estradiol level PFNA and lower estradiol level	PFOSA and prolactin level Me-PFOSA and estradiol level Me-PFOSA and prolactin level PFNA and prolactin level Other PFAS and prolactin level Other PFAS and estradiol level
Reproductive, female (fertility)	Whitworth 2012b	Case-control	General community (Norway)	Women	416 cases 494 controls	PFOA PFOS	PFOA and greater risk of subfecundity (parous women) PFOS and greater risk of subfecundity (parous women)	PFOA and risk of subfecundity (primiparous women) PFOS and risk of subfecundity (primiparous women)

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (fertility)	Whitworth 2016	Retrospective cohort	General community (Norway)	Women	451	PFHxS PFHpS PFOA PFOS PFOSA PFNA PFDA PFunDA PFdoDA PFtrDA	None	PFHxS and fecundability ratio (primiparous women) PFHpS and fecundability ratio (primiparous women) PFOA and fecundability ratio (primiparous women) PFOS and fecundability ratio (primiparous women) PFOSA and fecundability ratio (primiparous women) PFNA and fecundability ratio (primiparous women) PFDA and fecundability ratio (primiparous women) PFunDA and fecundability ratio (primiparous women) PFdoDA and fecundability ratio (primiparous women) PFtrDA and fecundability ratio (primiparous women)
Reproductive, female (pregnancy outcome)	Wu 2012	Cross-sectional	Contaminated community (Guiyu Town, Shantou City, Guangdong Province, China)	Newborns	167	PFOA	PFOA and greater risk of stillbirth	None
Reproductive, female (hormones)	Zhou 2016	Cross-sectional	General community (northern Taiwan)	Adolescent girls	123	PFBS PFHxA PFHxS PFOA PFOS PFNA PFDA PFdoDA PFteDA	PFdoDA and lower testosterone level	PFBS and testosterone and estradiol PFHxA and testosterone and estradiol PFHxS and testosterone and estradiol PFOA and testosterone and estradiol PFOS and testosterone and estradiol PFNA and testosterone and estradiol PFDA and testosterone and estradiol PFdoDA and estradiol PFteDA and testosterone and estradiol

Table 15. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and female reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, female (menstruation)	Zhou 2017a	Cross-sectional	General community (Shanghai, China)	Women	950	PFBS	PFHxS and greater risk of irregular menstrual cycle	PFBS and irregular menstrual cycle, long menstrual cycle, short menstrual cycle, menorrhagia, hypomenorrhea
						PFHxS	PFHxS and greater risk of long menstrual cycle	
						PFHpA	PFHxS and lower risk of menorrhagia	PFHxS and short menstrual cycle
						PFOA	PFHxS and greater risk of hypomenorrhea (quartiles 2 and 4, no trend)	
						PFOS		PFHpA and irregular menstrual cycle, long menstrual cycle, short menstrual cycle, menorrhagia, hypomenorrhea
						PFOSA		
						PFNA	PFOA and greater risk of irregular menstrual cycle	PFOA and short menstrual cycle
						PFDA	PFOA and greater risk of long menstrual cycle	
						PFunDA	PFOA and lower risk of menorrhagia	PFOS and irregular menstrual cycle, short menstrual cycle, hypomenorrhea
						PFdoDA	PFOA and greater risk of hypomenorrhea (quartile 3, not 4, no trend)	
								PFOSA and irregular menstrual cycle, long menstrual cycle, short menstrual cycle, menorrhagia, hypomenorrhea
							PFOS and greater risk of long menstrual cycle	
							PFOS and lower risk of menorrhagia	PFNA and short menstrual cycle
							PFNA and greater risk of irregular menstrual cycle	
							PFNA and greater risk of long menstrual cycle	PFDA and irregular menstrual cycle, long menstrual cycle, short menstrual cycle, menorrhagia, hypomenorrhea
							PFNA and lower risk of menorrhagia	
							PFNA and greater risk of hypomenorrhea (quartiles 2 and 3, not 4, no trend)	PFunDA and irregular menstrual cycle, long menstrual cycle, short menstrual cycle, menorrhagia, hypomenorrhea
								PFdoDA and irregular menstrual cycle, long menstrual cycle, short menstrual cycle, menorrhagia, hypomenorrhea

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Bae 2015	Prospective cohort	General community (Michigan and Texas)	Adults	233	PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	Paternal Me-PFOSA-AcOH and lower ratio of male to female births (tertiles, not continuous; joint couple model only) Paternal PFNA and lower ratio of male to female births (tertile 2, not 3 or continuous; joint couple model only)	Paternal PFOA and ratio of male to female births Paternal PFOS and ratio of male to female births Paternal PFOSA and ratio of male to female births Paternal Me-PFOSA-AcOH and ratio of male to female births (paternal model) Paternal Et-PFOSA-AcOH and ratio of male to female births Paternal PFNA and ratio of male to female births Paternal PFDA and ratio of male to female births
Reproductive, male	Buck Louis 2015	Cross-sectional (repeated)	General community (Michigan and Texas)	Men	462	PFOA PFOS PFOSA Me-PFOSA-AcOH Et-PFOSA-AcOH PFNA PFDA	PFOA and greater sperm curvilinear velocity PFOA and greater acrosome area of sperm head PFOA and lower percentage of sperm with coiled tail PFOS and greater straw distance PFOS and lower percentage of sperm with coiled tail PFOSA and smaller sperm head area PFOSA and smaller sperm head perimeter PFOSA and greater percentage bicephalic sperm PFOSA and greater number of immature sperm PFOSA and lower percentage sperm with high DNA stainability Me-PFOSA-AcOH and greater percentage sperm with neck or midpiece abnormalities Me-PFOSA-AcOH and greater number of immature sperm Me-PFOSA-AcOH and lower percentage sperm with high DNA stainability PFNA and greater percent normal sperm morphology, strict criteria PFNA and lower percentage of sperm with coiled tail PFDA and shorter sperm head length PFDA and lower percentage of sperm with coiled tail	PFOA, PFOS, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, and PFDA and general semen characteristics (semen volume, sperm viability, total sperm count, sperm concentration, average path velocity, straight line velocity, curvilinear velocity (except PFOA), amplitude head displacement, beat cross frequency, straightness, linearity, and percent motility) PFOA, PFOS, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, and PFDA and sperm head characteristics (length (except PFDA), area (except PFOSA), width, perimeter (except PFOSA), elongation factor, acrosome area of head (except PFOA)) PFOA, PFOS, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, and PFDA and straw distance (except PFOS) PFOA, PFOS, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, and PFDA and sperm morphology (percent normal strict criteria (except PFNA), percent normal WHO criteria, amorphous, round, pyriform, bicephalic (except PFOSA), tapered, megalo head, micro head, neck of midpiece abnormalities (except Me-PFOSA-AcOH), coiled tail (except PFOA, PFOS, PFNA and PFDA), other tail abnormalities, cytoplasmic droplet, immature sperm (except PFOSA and Me-PFOSA-AcOH)) PFOA, PFOS, PFOSA, Me-PFOSA-AcOH, Et-PFOSA-AcOH, PFNA, and PFDA and sperm chromatin stability (DNA fragmentation index, high DNA stainability (except PFOSA, Me-PFOSA-AcOH))

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Costa 2009	Cross-sectional	Occupational (Trissino, Italy)	Men	53	PFOA	None	PFOA and estradiol level PFOA and prostate-specific antigen level PFOA and testosterone level
Reproductive, male	Den Hond 2015	Case-control	General community (Belgium)	Men	163	PFOA PFOS	None	PFOA and infertility PFOA and sperm concentration, sperm motility, and sperm morphology PFOA and sex hormone binding globulin, testosterone, free testosterone, estradiol, free estradiol, luteinizing hormone, follicle-stimulating hormone, or inhibin B PFOS and infertility PFOS and sperm concentration, sperm motility, and sperm morphology PFOS and sex hormone binding globulin, testosterone, free testosterone, estradiol, free estradiol, luteinizing hormone, follicle-stimulating hormone, or inhibin B
Reproductive, male	Ducatman 2015	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Men	25,412	PFHxS PFOA PFOS PFNA	None	PFHxS and prostate-specific antigen PFOA and prostate-specific antigen PFOS and prostate-specific antigen PFNA and prostate-specific antigen
Reproductive, male	Goudarzi 2017a	Prospective cohort	General community (Sapporo, Japan)	Newborns	185	PFOA PFOS	PFOA and lower dehydroepiandrosterone level PFOS and greater dehydroepiandrosterone level	PFOA and androstenedione level PFOS and androstenedione level
Reproductive, male	Governini 2015	Cross-sectional	General community (Siena, Italy)	Men	59	PFOA and/or PFOS	PFOA and/or PFOS positivity in whole blood or seminal plasma and greater risk of 18 disomy PFOA and/or PFOS positivity in whole blood or seminal plasma and greater risk of gonosome disomy PFOA and/or PFOS positivity in whole blood or seminal plasma and greater risk of diploidy PFOA and/or PFOS positivity in whole blood or seminal plasma and greater risk of aneuploidy PFOA and/or PFOS positivity in whole blood or seminal plasma and greater percentage PI^{dim} population PFOA and/or PFOS positivity in whole blood or seminal plasma and greater percentage PI^{br}+PI^{dim} population	PFOA and/or PFOS positivity in whole blood or seminal plasma and greater percentage PI^{br} population PFOA and/or PFOS positivity in whole blood or seminal plasma and greater percentage M540 population PFOA and/or PFOS positivity in whole blood or seminal plasma and other measures of sperm quality, sperm aneuploidy, and sperm DNA fragmentation (not individually identified)

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Grice 2007	Retrospective cohort	Occupational (Decatur, Alabama)	Men	1,137	PFOS	None	PFOS and benign prostatic hyperplasia PFOS and prostatitis
Reproductive, male	Itoh 2016	Prospective cohort	General community (Hokkaido, Japan)	Newborn boys	83	PFOA PFOS	PFOA and greater inhibin B level PFOS and greater estadiol level PFOS and lower testosterone:estradiol ratio PFOS and lower progesterone level PFOS and lower inhibin B level	PFOA and estradiol PFOA and testosterone PFOA and testosterone:estradiol ratio PFOA and progesterone PFOA and luteinizing hormone PFOA and follicle-stimulating hormone PFOA and sex hormone binding globulin PFOA and testosterone:sex hormone binding globulin ratio PFOA and prolactin PFOA and insulin-like factor 3 PFOS and testosterone PFOS and luteinizing hormone PFOS and follicle-stimulating hormone PFOS and sex hormone binding globulin PFOS and testosterone:sex hormone binding globulin ratio PFOS and prolactin PFOS and insulin-like factor 3

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Joensen 2009	Cross-sectional	General community (Copenhagen, Denmark)	Men	105	PFOA PFOS PFOA+PFOS	PFOA+PFOS and lower percent morphologically normal sperm PFOA+PFOS and lower total number of morphologically normal sperm	PFOA and testosterone level, estradiol level, sex hormone binding globulin level, luteinizing hormone level, follicle-stimulating hormone level, inhibin-B level, free androgen index (testosterone × 100/sex hormone binding globulin), testosterone/luteinizing hormone ratio, free androgen index/luteinizing hormone ratio, estradiol/testosterone ratio, inhibin B/follicule-stimulating hormone ratio, semen volume, semen concentration, total sperm count, and percent motile sperm PFOA and percent and number of morphologically normal sperm PFOS and testosterone level, estradiol level, sex hormone binding globulin level, luteinizing hormone level, follicle-stimulating hormone level, inhibin-B level, free androgen index (testosterone × 100/sex hormone binding globulin), testosterone/luteinizing hormone ratio, free androgen index/luteinizing hormone ratio, estradiol/testosterone ratio, inhibin B/follicule-stimulating hormone ratio, semen volume, semen concentration, total sperm count, and percent motile sperm PFOS and percent and number of morphologically normal sperm PFOA+PFOS and testosterone level, estradiol level, sex hormone binding globulin level, luteinizing hormone level, follicle-stimulating hormone level, inhibin-B level, free androgen index (testosterone × 100/sex hormone binding globulin), testosterone/luteinizing hormone ratio, free androgen index/luteinizing hormone ratio, estradiol/testosterone ratio, inhibin B/follicule-stimulating hormone ratio, semen volume, semen concentration, total sperm count, and percent motile sperm

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Joensen 2013	Cross-sectional	General community (Denmark)	Men	247	PFHxS PFHpS PFOA PFOS PFNA PFDA	PFHpS and lower percent progressively motile sperm PFOS and lower testosterone level PFOS and lower free androgen index PFOS and lower free testosterone PFOS and lower free testosterone/luteinizing hormone ratio PFOS and lower free androgen index/luteinizing hormone ratio PFOS and lower testosterone/luteinizing hormone ratio PFNA and lower estradiol level	PFHxS, PFHpS, PFOA, PFOS, PFNA, and PFDA and semen quality parameters: semen volume, sperm concentration, total sperm count, progressively motile sperm (except PFHpS), morphologically normal sperm, and total normal sperm count PFHxS, PFHpS, PFOA, PFOS, PFNA, and PFDA and reproductive hormones: testosterone (except PFOS), free androgen index (except PFOS), free testosterone (except PFOS), free testosterone/luteinizing hormone ratio (except PFOS), free androgen index/luteinizing hormone ratio (except PFOS), testosterone/luteinizing hormone ratio (except PFOS), testosterone/estradiol ratio, estradiol (except PFNA), sex hormone binding globulin, luteinizing hormone, follicle-stimulating hormone, inhibin-B, and inhibin-B/follicle-stimulating hormone ratio
Reproductive, male	Jorgensen 2014	Retrospective cohort	Fishing/general community (Greenland; Kharkiv, Ukraine; Warsaw, Poland)	Men	401	PFHxS PFOA PFOS PFNA	PFNA and lower fecundability ratio (Greenland)	PFHxS and fecundability ratio PFOA and fecundability ratio PFOS and fecundability ratio PFNA and fecundablity ratio (Ukraine, Poland)
Reproductive, male	Kvist 2012	Cross-sectional	Fishing/general community (Greenland; Kharkiv, Ukraine; Warsaw, Poland)	Men	607	PFOA PFOS	PFOS and greater sperm Y:X chromosome ratio (total) PFOS and lower sperm Y:X chromosome ratio (Greenland)	PFOA and sperm Y:X chromosome ratio PFOS and sperm Y:X chromosome ratio (Ukraine, Poland)
Reproductive, male	La Rocca 2015	Case-control	General community (Rome, Ferrara, and Sora, Italy)	Men	70 cases 83 controls	PFOA PFOS	PFOS in blood and greater risk of infertility (metropolitan)	PFOA in blood and infertility PFOA in semen and infertility PFOS in blood and infertility (total, urban, rural) PFOS in semen and infertility

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Lenters 2015	Cross-sectional	Fishing/general community (Greenland; Kharkiv, Ukraine; Warsaw, Poland)	Men	602	PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA	None	PFHxS, PFOA, PFOS, PFNA, PFDA, PFunDA, and PFdoDA and reproductive hormones: follicle-stimulating hormone, luteinizing hormone, inhibin B, sex hormone binding globulin, total testosterone, free testerone, and estradiol PFHxS, PFOA, PFOS, PFNA, PFDA, PFunDA, and PFdoDA and conventional semen characteristics: semen volume, sperm concentration, total sperm count, percent morphologically normal sperm, and percent progressive motility PFHxS, PFOA, PFOS, PFNA, PFDA, PFunDA, and PFdoDA and sperm chromatin integrity: sperm DNA fragmentation by sperm chromatin structure assay, sperm DNA fragmentation by terminal deoxynucleotidyl transferase dUTP nick end-labeling, and high DNA stainability PFHxS, PFOA, PFOS, PFNA, PFDA, PFunDA, and PFdoDA and apoptotic markers: Fas positivity, Bcl-xL positivity PFHxS, PFOA, PFOS, PFNA, PFDA, PFunDA, and PFdoDA and epididymal and accessory sex gland function: neutral alpha-glucosidase, prostate-specific antigen, zinc, and fructose in ejaculate PFHxS, PFOA, PFOS, PFNA, PFDA, PFunDA, and PFdoDA and percentage Y chromosome sperm cells
Reproductive, male	Lewis 2015	a	General community (United States)	Men, adolescent boys	268 (largest age-sex stratum)	PFHxS PFOA PFOS PFNA	None	PFHxS and testosterone (boys/men 12-<20, 20-<40, 40-<60, 60-80 y) PFOA and testosterone (boys/men 12-<20, 20-<40, 40-<60, 60-80 y) PFOS and testosterone (boys/men 12-<20, 20-<40, 40-<60, 60-80 y) PFNA and testosterone (boys/men 12-<20, 20-<40, 40-<60, 60-80 y)
Reproductive, male	Lopez-Espinosa 2011	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Boys, adolescent boys	3,072	PFOA PFOS	PFOS and later age at puberty (total testosterone > 50 ng/dL) PFOS and later age at puberty (free testosterone > 5 pg/mL)	PFOA and age at puberty (total testosterone > 50 ng/dL) PFOA and age at puberty (free testosterone > 5 pg/mL)

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Lopez-Espinosa 2016	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Boys, adolescent boys	1,169	PFHxS PFOA PFOS PFNA	PFOA and lower total testosterone level at 6-9 years PFOS and lower estradiol level at 6-9 years PFOS and lower total testosterone level at 6-9 years	PFHxS and estradiol at 6-9 years PFHxS and total testosterone at 6-9 years PFOA and estradiol at 6-9 years PFNA and estradiol at 6-9 years PFNA and total testosterone at 6-9 years
Reproductive, male	Olsen 1998	Cross-sectional	Occupational (Cottage Grove, Minnesota)	Men	111	PFOA	PFOA and greater prolactin level (1993) PFOA and greater 17-hydroxyprogesterone level (1995)	PFOA and dehydroepiandrosterone sulfate PFOA and estradiol PFOA and 17-hydroxyprogesterone (1993) PFOA and luteinizing hormone PFOA and prolactin (1995) PFOA and sex hormone binding globulin PFOA and bound testosterone PFOA and free testosterone
Reproductive, male	Olsen 2004	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,311	PFOS	None	PFOS and episodes of care for fertility and infertility management PFOS and episodes of care for complicated pregnancy or delivery PFOS and episodes of care for preterm labor PFOS and episodes of care for normal or unspecified pregnancy or delivery PFOS and episodes of care for spontaneous abortion PFOS and episodes of care for pregnancy PFOS and episodes of care for congenital anomalies PFOS and episodes of care for perinatal disorders

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Raymer 2012	Cross-sectional	General community (Durham, North Carolina)	Men	252	PFOA PFOS	Plasma PFOA and greater free testosterone level Plasma PFOA and greater luteinizing hormone level	Plasma PFOA and semen volume, semen pH, sperm concentration, semen white blood cell concentration, percent motile, initial total motile, percent swim-up overnight motility, swim-up concentration, percent swim-up motility, swim-up total motile, abnormal liquefaction, abnormal viscosity, abnormal volume, abnormal sperm concentration, and abnormal directional motility Plasma or semen PFOS and semen volume, semen pH, sperm concentration, semen white blood cell concentration, percent motile, initial total motile, percent swim-up overnight motility, swim-up concentration, percent swim-up motility, swim-up total motile, abnormal liquefaction, abnormal viscosity, abnormal volume, abnormal sperm concentration, and abnormal directional motility Plasma PFOA and estradiol, prolactin, follicle-stimulating hormone, and total testosterone Plasma or semen PFOS and estradiol, prolactin, follicle-stimulating hormone, free testosterone, total testosterone, and luteinizing hormone

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Specht 2012	Cross-sectional	Fishing/general community (Greenland; Kharkiv, Ukraine; Warsaw, Poland)	Men	604	PFHxS PFOA PFOS PFNA	PFOA and greater DNA fragmentation based on percentage of in situ terminal deoxynucleotidyl transferase dUTP nick-end labeling (TUNEL)-positive sperm cells (Greenland) PFOA and greater sex hormone binding globulin level (Poland) PFOS and greater semen apoptotic markers based on percentage of sperm cells positive for Fas (Poland)	PFHxS and DNA fragmentation in sperm cells (based on sperm chromatin structure assay (SCSA) or in situ terminal deoxynucleotidyl transferase dUTP nick-end labeling (TUNEL) assay) or apoptotic markers in semen (based on positivity for Fas or Bcl-xL) PFHxS and sex hormone binding globulin, testosterone, estradiol, follicule-stimulating hormone, and luteinizing hormone PFOA and DNA fragmentation in sperm cells (based on SCSA assay or TUNEL assay; TUNEL overall, Ukraine, and Poland) or apoptotic markers in semen (based on positivity for Fas or Bcl-xL) PFOA and sex hormone binding globulin (overall, Greenland, Ukraine), testosterone, estradiol, follicule-stimulating hormone, and luteinizing hormone PFOS and DNA fragmentation in sperm cells (based on SCSA assay or TUNEL assay) or apoptotic markers in semen (based on positivity for Fas or Bcl-xL; Fas overall, Greenland, Ukraine) PFOS and sex hormone binding globulin, testosterone, estradiol, follicule-stimulating hormone, and luteinizing hormone PFNA and DNA fragmentation in sperm cells (based on SCSA assay or TUNEL assay) or apoptotic markers in semen (based on positivity for Fas or Bcl-xL) PFNA and sex hormone binding globulin, testosterone, estradiol, follicule-stimulating hormone, and luteinizing hormone

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Toft 2012	Cross-sectional	Fishing/general community (Greenland; Kharkiv, Ukraine; Warsaw, Poland)	Men	588	PFHxS PFOA PFOS PFNA	PFHxS and lower percentage of morphologically normal sperm (tertile 3, no trend; overall) PFOA and greater percentage of motile sperm (overall, Greenland) PFOS and greater sperm concentration (tertile 2, no trend, Poland) PFOS and greater total sperm count (tertile 2, no trend, Poland) PFOS and lower percentage of morphologically normal sperm (tertiles 2 and 3, no trend; overall) PFOS and greater percentage sperm with tail defect (tertile 2, no trend, Poland)	PFHxS and sperm concentration, semen volume, total sperm count, and percentage motile sperm PFOA and sperm concentration, semen volume, total sperm count, percentage motile sperm (Ukraine, Poland), and percentage morphologically normal cells PFOS and sperm concentration (overall, Greenland, Ukraine), semen volume, total sperm count (overall, Greenland, Ukraine), percentage motile sperm, percentage morphologically normal cells (Greenland, Poland, Ukraine), and percentage with head defect, midpiece defect, cytoplasm drop, or immature spermatozoa PFNA and sperm concentration, semen volume, total sperm count, percentage motile sperm, and percentage morphologically normal cells
Reproductive, male	Toft 2016	Nested prospective case-control	General community (Denmark)	Newborn boys	270 cryptorchidism cases 75 hypospadias cases 300 controls	PFOS	PFOS and greater testosterone level PFOS and greater androstenedione level PFOS and greater progesterone level PFOS and greater 17-OH progesterone level PFOS and lower insulin-like factor 3 level (absolute concentration, not multiple of median values)	PFOS and dehydroepiandrosterone sulfate PFOS and risk of cryptorchidism PFOS and risk of hypospadias
Reproductive, male	Tsai 2015	Cross-sectional	General community (Taipei, Taiwan)	Adolescents, young adults	30 boys 12-17 y 180 men 18-30 y	PFOA PFOS PFNA PFunDA	PFOS and lower follicle-stimulating hormone level (boys 12-17 y)	PFOA and sex hormone binding globulin, follicle-stimulating hormone, total testosterone, estrogen, luteinizing hormone, and free testosterone (boys 12-17 y, men 18-30 y) PFOS and sex hormone binding globulin, total testosterone, estrogen, luteinizing hormone, and free testosterone (boys 12-17 y, men 18-30 y) PFOS and follicle-stimulating hormone (men 18- 30 y) PFNA and sex hormone binding globulin, follicle-stimulating hormone, total testosterone, estrogen, luteinizing hormone, and free testosterone (boys 12-17 y, men 18-30 y) PFunDA and sex hormone binding globulin, follicle-stimulating hormone, total testosterone, estrogen, luteinizing hormone, and free testosterone (boys 12-17 y, men 18-30 y)

Table 16. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and male reproductive outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Reproductive, male	Vested 2013	Prospective and retrospective cohort	General community (Aarhus, Denmark)	Men	169	PFOA PFOS	PFOA and lower sperm concentration PFOA and lower total sperm count PFOA and lower percentage progressive spermatozoa (computer-assisted semen analysis) PFOA and greater luteinizing hormone level PFOA and greater follicle-stimulating hormone level	PFOA and history of reproductive tract disease (cryptorchidism, hypospadias, inguinal hernia, varicocele, testicular hydrocele, incarcerated hernia, phimosis, testicular torsion, chlamydia, gonorrhea, or epididymitis) PFOA and semen volume, percentage progressive spermatozoa (manual semen analysis), percentage morphologically normal spermatozoa, and mean testicular volume PFOA and testosterone, estradiol, inhibin B, sex hormone binding globulin, and free androgen index PFOS and history of reproductive tract disease (cryptorchidism, hypospadias, inguinal hernia, varicocele, testicular hydrocele, incarcerated hernia, phimosis, testicular torsion, chlamydia, gonorrhea, or epididymitis) PFOS and sperm concentration, total sperm count, semen volume, percentage progressive spermatozoa, percentage morphologically normal spermatozoa, and mean testicular volume PFOS and testosterone, estradiol, luteinizing hormone, follicle-stimulating hormone, inhibin B, sex hormone binding globulin, and free androgen index
Reproductive, male	Vesterholm Jensen 2014	Case-control	General community (Denmark and Finland)	Infant boys	107 cases 108 controls	PFOA PFOS	PFOA and lower risk of cryptorchidism (Finland)	PFOA and risk of cryptorchidism (overall, Denmark) PFOS and risk of cryptorchidism (overall, Denmark, Finland)
Reproductive, male	Zhou 2016	Cross-sectional	General community (northern Taiwan)	Adolescent boys	102	PFBS PFHxA PFHxS PFOA PFOS PFNA PFDA PFdoDA PFteDA	PFHxA and lower testosterone level PFHxS and greater estradiol level PFOA and greater estradiol level PFOS and lower testosterone level PFNA and lower testosterone level PFDA and lower testosterone level	PFBS and testosterone and estradiol PFHxA and estradiol PFHxS and testosterone PFOA and testosterone PFOS and estradiol PFNA and estradiol PFDA and estradiol PFdoDA and testosterone and estradiol PFteDA and testosterone and estradiol

Table 17. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and respiratory outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Respiratory	Alexander 2003	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	2,083	PFOS	None	PFOS and mortality from non-malignant respiratory disease
Respiratory	Anderson-Mahoney 2008	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	566	PFOA	Residence in PFOA water district and greater risk of chronic bronchitis Residence in PFOA water district and greater risk of shortness of breath on stairs	None
Respiratory	Consonni 2013	Retrospective cohort	Occupational (Germany, Netherlands, Italy, United Kingdom, New Jersey, West Virginia)	Adults	4,773	PFOA/tetrafluoroethylene (TFE)	PFOA and lower risk of mortality from non-malignant respiratory disease	None
Respiratory	Gilliland 1993	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,537	PFOA	None	PFOA and mortality from respiratory disease (men, men in chemical division)
Respiratory	Leonard 2008	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	6,027	PFOA	PFOA and lower risk of mortality from nonmalignant respiratory disease (vs. US or West Virginia) PFOA and lower risk of mortality from influenza and pneumonia (vs. US or West Virginia) PFOA and lower risk of mortality from bronchitis, emphysema, and asthma (vs. US or West Virginia) PFOA and lower risk of mortality from bronchitis (vs. West Virginia) PFOA and lower risk of mortality from other respiratory disease (vs. US or West Virginia)	PFOA and mortality from nonmalignant respiratory disease (vs. DuPont Region 1) PFOA and mortality from influenza and pneumonia (vs. DuPont Region 1) PFOA and mortality from bronchitis, emphysema, and asthma (vs. DuPont Region 1) PFOA and mortality from bronchitis (vs. US or DuPont Region 1) PFOA and mortality from emphysema (vs. US, West Virginia, or DuPont Region 1) PFOA and mortality from other respiratory disease (vs. DuPont Region 1)
Respiratory	Lundin 2009	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,993	PFOA	PFOA and lower risk of mortality from non-malignant respiratory disease (ever probable/never definite exposure) PFOA and lower risk of mortality from other non-malignant respiratory disease (ever probable/never definite exposure)	PFOA and mortality from tuberculosis PFOA and mortality from non-malignant respiratory disease (ever definite exposure) PFOA and mortality from influenza and pneumonia PFOA and mortality from bronchitis PFOA and mortality from emphysema PFOA and mortality from asthma PFOA and mortality from other non-malignant respiratory disease (ever definite exposure)
Respiratory	Melzer 2010	Cross-sectional	General community (United States)	Adults	3,974	PFOA PFOS	PFOS and lower risk of COPD ever	PFOA and COPD ever

Table 17. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and respiratory outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Respiratory	Nolan 2010	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Women	1,548	PFOA	None	PFOA (Little Hocking Water Association only) and maternal lung disease PFOA (partial Little Hocking Water Association) and maternal lung disease
Respiratory	Steenland 2012	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	5,791	PFOA	None	PFOA and mortality from chronic obstructive pulmonary disease
Respiratory	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	None	PFOA and chronic obstructive pulmonary disease PFOA and medicated asthma

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Anderson-Mahoney 2008	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	566	PFOA	Residence in PFOA water district and greater risk of thyroid problems (total, 18-34 (women; men unobserved), 35-49, and 50-64 years)	Residence in PFOA water district and thyroid problems (65+ years)
Thyroid	Audet-Delage 2013	Cross-sectional	Fishing community (Nunavik, Arctic Quebec, Canada)	Women	120	PFOS	None	PFOS and T4 bound to transthyretin (T4 transport protein)
Thyroid	Berg 2015	Prospective cohort	General community (northern Norway)	Women	375	PFHxS	PFOS and greater TSH level	PFHxS and TSH, T3, free T3, T4, and free T4
						PFHpS	PFOS and greater TSH:T4 or free T4 ratio	
						PFOA	PFOS and greater TSH:T3 or free T3 ratio	PFHpS and TSH, T3, free T3, T4, and free T4
						PFOS		
						PFNA	PFDA and lower T3 level	PFOA and TSH, T3, free T3, T4, and free T4
						PFDA		
						PFunDA	PFunDA and lower free T3 level	PFOS and T3, free T3, T4, and free T4
								PFNA and TSH, T3, free T3, T4, and free T4
								PFDA and TSH, free T3, T4, and free T4
								PFunDA and TSH, T3, T4, and free T4
Thyroid	Berg 2017	Prospective cohort	General community (northern Norway)	Newborns	370	PFHxS	PFOS and greater maternal TSH level	PFHxS and maternal TSH, T3, free T3, T4, and free T4 PFHxS and newborn TSH, T3, free T3, T4, and free T4
						PFHpS		
						PFOA	PFDA and lower maternal T3 level	
						PFOS		PFHpS and maternal TSH, T3, free T3, T4, and free T4
						PFNA	PFunDA and lower maternal free T3 level	PFHpS and newborn TSH, T3, free T3, T4, and free T4
						PFDA		
						PFunDA		PFOA and maternal TSH, T3, free T3, T4, and free T4 PFOA and newborn TSH, T3, free T3, T4, and free T4
						ΣPFAS (PFHxS+PFHpS+PFOA+PFOS+PFNA+PFDA+PFunDA)		PFOS and maternal TSH, T3, free T3, T4, and free T4 PFOS and newborn TSH, T3, free T3, T4, and free T4
								PFNA and maternal TSH, T3, free T3, T4, and free T4 PFNA and newborn TSH, T3, free T3, T4, and free T4
								PFDA and maternal TSH, free T3, T4, and free T4 PFDA and newborn TSH, T3, free T3, T4, and free T4
								PFunDA and maternal TSH, T3, T4, and free T4 PFunDA and newborn TSH, T3, free T3, T4, and free T4
								ΣPFAS and maternal TSH, T3, free T3, T4, and free T4 ΣPFAS and newborn TSH, T3, free T3, T4, and free T4

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Bloom 2010	Cross-sectional	Fishing community (Wisconsin anglers)	Men	31	PFHxS PFOA PFOS PFNA PFDA PFunDA ΣPFAS (PFHxS+PFOA+PFOS+PFNA+PFDA+PFunDA)	None	PFHxS and TSH and free T4 PFOA and TSH and free T4 PFOS and TSH and free T4 PFNA and TSH and free T4 PFDA and TSH and free T4 PFunDA and TSH and free T4 ΣPFAS and TSH and free T4
Thyroid	Chan 2011	Case-control	General community (Edmonton, Alberta, Canada)	Women	96 cases, 175 controls	PFHxS PFOA PFOS ΣPFAS (PFHxS+PFOA+PFOS)	None	PFHxS and hypothyroxinemia (normal TSH, no evidence of hyperthyroidism, lowest 10th percentile of free T4 in population sample) PFOA and hypothyroxinemia PFOS and hypothyroxinemia ΣPFAS and hypothyroxinemia
Thyroid	Christensen 2016b	Cross-sectional	Fishing community (Wisconsin anglers)	Men	154	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA ΣPFAS (PFHxS+PFHpS+PFOA+PFOS+PFNA+PFDA+PFunDA)	None	PFHxS and thyroid disease PFHpS and thyroid disease PFOA and thyroid disease PFOS and thyroid disease PFNA and thyroid disease PFDA and thyroid disease PFunDA and thyroid disease ΣPFAS and thyroid disease
Thyroid	Crawford 2017	Cross-sectional	General community (North Carolina)	Women	99	PFHxS PFOA PFOS PFNA ΣPFAS (PFHxS+PFOA+PFOS+PFNA)	PFOA and greater T3 level PFNA and greater T3 level PFNA and greater free T4 level ΣPFAS and greater T3 level	PFHxS and TSH, T3, T4, and free T4 PFOA and TSH, T4, and free T4 PFOS and TSH, T3, T4, and free T4 PFNA and TSH and T4 ΣPFAS and TSH, T4, and free T4

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Dallaire 2009	Cross-sectional	Fishing community (Nunavik, Arctic Quebec, Canada)	Adults	623	PFOS	PFOS and lower TSH level PFOS and lower T3 level PFOS and greater free T4 level PFOS and lower thyroid-binding globulin level	None
Thyroid	de Cock 2014	Prospective cohort	General community (Zwolle, Netherlands)	Newborns	83	PFOA PFOS	PFOA and greater T4 level (girls)	PFOA and T4 (boys) PFOS and T4
Thyroid	Emmett 2006	Cross-sectional	Contaminated community (Mid-Ohio Valley)	All (children, adolescents, adults)	371	PFOA	None	PFOA and TSH PFOA and self-reported thyroid disease
Thyroid	Espino-Hernandez 2011 (reanalysis of Chan 2011)	Case-control	General community (Edmonton, Alberta, Canada)	Women	96 cases, 175 controls	PFHxS PFOA PFOS	None	PFHxS and hypothyroxinemia (normal TSH, no evidence of hyperthyroidism, lowest 10th percentile of free T4 in population sample), correcting for PFAS measurement error PFOA and hypothyroxinemia, correcting for PFAS measurement error PFOS and hypothyroxinemia, correcting for PFAS measurement error
Thyroid	Inoue 2004	Prospective cohort and cross-sectional	General community (Hokkaido, Japan)	Newborns	15	PFOS	None	PFOS and TSH PFOS and free T4
Thyroid	Jain 2013	Cross-sectional	General community (United States)	Adolescents, adults	1,540	PFHxS PFOA PFOS Me-PFOSA-AcOH PFNA PFDA	PFHxS and greater total T4 level (categorical, not continuous) PFOA and greater TSH level (categorical, not continuous) PFOA and greater total T3 level	PFHxS and TSH, total T3, free T3, free T4, and thyroglobulin PFOA and free T3, total T4, free T4, and thyroglobulin PFOS and TSH, total T3, free T3, total T4, free T4, and thyroglobulin Me-PFOSA-AcOH and TSH, total T3, free T3, total T4, free T4, and thyroglobulin PFNA and TSH, total T3, free T3, total T4, free T4, and thyroglobulin PFDA and TSH, total T3, free T3, total T4, free T4, and thyroglobulin

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Ji 2012	Cross-sectional	General community (Siheung, South Korea)	Adolescents, adults	633	PFHxS PFHpS PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA	PFtrDA and lower T4 level (total, females) PFtrDA and greater TSH level (total, females) PFtrDA×PFOS interaction and TSH level (males) PFOS and lower TSH level in model with PFtrDA×PFOS interaction (males) PFtrDA and greater TSH level in model with PFtrDA×PFOS interaction (males)	PFHxS and TSH and T4 PFHpS and TSH and T4 PFOA and TSH and T4 PFOS and TSH and T4 PFNA and TSH and T4 PFDA and TSH and T4 PFunDA and TSH and T4 PFdoDA and TSH and T4 PFtrDA and TSH (males) and T4 (males)
Thyroid	Kato 2016	Prospective cohort and retrospective (mothers)	General community (Hokkaido, Japan)	Mothers, newborns	392	PFOA PFOS	PFOS and lower maternal TSH level PFOS and greater infant TSH level	PFOA and maternal TSH PFOA and maternal free T4 PFOA and infant TSH PFOA and infant free T4 PFOS and maternal free T4 PFOS and infant free T4
Thyroid	Kim 2011a	Prospective cohort and cross-sectional	General community (Seoul, Cheongju, and Gumi, South Korea)	Newborns	35	PFHxS PFOA PFOS PFtrDA ΣPFAS (PFHxS+PFOA+PFOS+PFtrDA)	PFOA (maternal) and greater TSH level PFOS (maternal) and lower T3 level PFtrDA (maternal) and lower T3 level PFtrDA (maternal) and lower T4 level ΣPFAS (maternal) and lower T3 level	PFHxS (maternal) and T3, T4, and TSH levels PFHxS (fetal) and T3, T4, and TSH levels PFOA (maternal) and T3 and T4 levels PFOA (fetal) and T3, T4, and TSH levels PFOS (maternal) and T4 and TSH levels PFOS (fetal) and T3, T4, and TSH levels PFtrDA (maternal) and TSH level PFtrDA (fetal) and T3, T4, and TSH levels ΣPFAS (maternal) and T4 and TSH levels ΣPFAS (fetal) and T3, T4, and TSH levels

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Kim 2016a	Case-control	General community (Seoul, South Korea)	Infants	27 cases, 13 controls	PFBA PFBS PFPA PFHxA PFHxS PFHpA PFHpS PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA ΣPFSA (PFBS+PFHxS+PFHpS+PFOS) ΣPFAS (PFBA+PFBS+PFPA+PFHxA+PFHxS+PFHpA+PFHpS+PFOA+PFOS+PFNA+PFDA+PFunDA+PFdoDA+PFtrDA)	PFPA and greater levels of relevant microsomal antibodies (controls) PFHxS and lower levels of thyroid-stimulating immunoglobulin (cases) PFHpA and greater levels of relevant microsomal antibodies (controls) PFOA and greater risk of congenital hypothyroidism PFOA and lower levels of thyroid-stimulating immunoglobulin (cases) PFNA and greater risk of congenital hypothyroidism PFDA and greater risk of congenital hypothyroidism PFunDA and greater risk of congenital hypothyroidism PFtrDA and lower levels of relevant microsomal antibodies (cases) ΣPFSA and lower levels of thyroid-stimulating immunoglobulin (cases) ΣPFAS and greater risk of congenital hypothyroidism ΣPFAS and lower levels of thyroid-stimulating immunoglobulin (cases)	PFBA, PFBS, PFHxA, PFHpS, PFOS, PFdoDA, and congenital hypothyroidism, TSH, total T3, free T4, thyroglobulin antibody, relevant microsomal antibodies, and thyroid-stimulating immunoglobulin PFPA and congenital hypothyroidism, TSH, total T3, free T4, thyroglobulin antibody, relevant microsomal antibodies (cases), and thyroid-stimulating immunoglobulin PFHxS and congenital hypothyroidism, TSH, total T3, free T4, thyroglobulin antibody, relevant microsomal antibodies, and thyroid-stimulating immunoglobulin (controls) PFHpA and congenital hypothyroidism, TSH, total T3, free T4, thyroglobulin antibody, relevant microsomal antibodies (cases), and thyroid-stimulating immunoglobulin PFOA and TSH, total T3, free T4, thyroglobulin antibody, relevant microsomal antibodies, and thyroid-stimulating immunoglobulin (controls) PFtrDA and congenital hypothyroidism, TSH, total T3, free T4, thyroglobulin antibody, relevant microsomal antibodies (controls), and thyroid-stimulating immunoglobulin ΣPFSA and TSH, total T3, free T4, thyroglobulin antibody, relevant microsomal antibodies, and thyroid-stimulating immunoglobulin (controls) ΣPFAS and TSH, total T3, free T4, thyroglobulin antibody, relevant microsomal antibodies, and thyroid-stimulating immunoglobulin (controls)
Thyroid	Knox 2011a	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults, older adults	16,193 women 20-50 y 14,944 men 20-50 y 8,854 women > 50 y 10,122 men > 50 y	PFOA PFOS	PFOA×sex and TSH PFOA and lower T3 uptake (women 20-50 y, women > 50 y, men > 50 y) PFOA×sex and T3 uptake level PFOA and greater total T4 level (women 20-50 y, women > 50 y, men > 50 y) PFOA and greater albumin level (women 20-50 y, women > 50 y, men 20-50 y, men > 50 y) PFOA×sex and albumin level PFOS and lower T3 uptake (women 20-50 y, women > 50 y, men 20-50 y, men > 50 y) PFOS×sex and T3 uptake level PFOS and greater total T4 level (women 20-50 y, women > 50 y, men 20-50 y, men > 50 y) PFOS×sex and total T4 level PFOS and greater albumin level (women 20-50 y, women > 50 y, men 20-50 y, men > 50 y)	PFOA and TSH (women 20-50 y, women > 50 y, men 20-50 y, men > 50 y) PFOA and T3 uptake (men 20-50 y) PFOA and total T4 (men 20-50 y) PFOA×sex and total T4 PFOS and TSH (women 20-50 y, women > 50 y, men 20-50 y, men > 50 y)

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Lewis 2015	Cross-sectional	General community (United States)	Adults, adolescents	268 (largest age-sex stratum)	PFHxS PFOA PFOS PFNA	PFHxS and greater total T3 level (women 50-80 y) PFOA and greater free T3 level (women 60-80, 50-80 y) PFOA and greater total T3 level (women 60-80, 50-80 y) PFOA and greater free T4 level (women 20-<40, 20-<50 y) PFOA and lower TSH level (girls 12-<20 y) PFOS and greater TSH level (boys 12-<20 y) PFOS and greater free T4 level (women 20-<40, 20-<50 y) PFNA and greater TSH level (boys 12-<20 y) PFNA and greater free T4 level (women 20-<40, 20-<50 y)	PFHxS and free T3, total T3, free T4, total T4, and TSH (boys/men 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFHxS and free T3, free T4, total T4, and TSH (girls/women 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFHxS and total T3 (girls/women 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50 y) PFOA and free T3, total T3, free T4, total T4, and TSH (boys/men 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFOA and total T4 (girls/women 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFOA and free T3 and total T3 (girls/women 12-<20, 20-<40, 40-<60 y; 20-<50 y) PFOA and free T4 (girls/women 12-<20, 40-<60, 60-80 y; 50-80 y) PFOA and TSH (women 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFOS and free T3, total T3, free T4, and total T4 (boys/men 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFOS and TSH (men 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFOS and free T3, total T3, total T4, and TSH (girls/women 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFOS and free T4 (girls/women 12-<20, 40-<60, 60-80 y; 50-80 y) PFNA and free T3, total T3, free T4, and total T4 (boys/men 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFNA and TSH (men 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFNA and free T3, total T3, and total T4 (girls/women 12-<20, 20-<40, 40-<60, 60-80 y; 20-<50, 50-80 y) PFNA and free T4 (girls/women 12-<20, 40-<60, 60-80 y; 50-80 y)
Thyroid	Li 2017	Cross-sectional	General community (southern China)	All	202	PFBA PFBS PFPA PFPrA (pentafluoropropionic acid) PFHxA PFHxS PFOA PFOS ΣPFAS (PFBA+PFBS+PFPA+PFPrA+PFHxA+PFHxS+PFOA+PFOS)	PFBA and lower TSH level (normal thyroid) PFPA and lower TSH level (hyperthyroidism) PFHxA and greater anti-thyroglobulin antibody level (total) PFHxA and greater anti-thyroid microsomal antibody level (total) PFHxA and greater anti-thyroglobulin antibody level (normal thyroid) PFOA and lower free T4 level (Hashimoto's disease) PFOS and lower free T3 level (total) PFOS and lower free T4 level (total) PFOS and greater TSH level (total) ΣPFAS and lower free T3 level (hyperthyroidism) ΣPFAS and lower free T4 level (hyperthyroidism)	PFBS, PFPrA, and PFHxS and free T3, free T4, TSH, anti-thyroglobulin antibody, and anti-microsomal antibody (total, normal thyroid, hyperthyroidism, hypothyroidism, Hashimoto's disease) PFBA, PFBS, PFPA, PFPrA, PFHxA, PFHxS, PFOA, PFOS, and ΣPFAS and free T3, free T4, TSH, anti-thyroglobulin antibody, and anti-microsomal antibody (hypothyroidism) Other associations between PFBA, PFPA, PFHxA, PFOA, PFOS, and ΣPFAS and free T3, free T4, TSH, anti-thyroglobulin antibody, and anti-microsomal antibody (total, normal thyroid, hyperthyroidism, hypothyroidism, Hashimoto's disease), except as shown at left

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Lin 2013b	Cross-sectional	General community (Taipei, Taiwan)	Adolescents, young adults	551	PFOA PFOS PFNA PFunDA	PFNA and greater free T4 level (total, males 20-30 y, BMI ≥ 24 mg/m^2, ever smoked, current or no hypertension)	PFOA and free T4, TSH, hypothyroidism (TSH above normal range) (total, males and females 12-19 y, males and females 20-30 y, subgroups by BMI, smoking, and hypertension) PFOS and free T4, TSH, hypothyroidism (total, males and females 12-19 y, males and females 20-30 y, subgroups by BMI, smoking, and hypertension) PFNA and free T4 (males and females 12-19 y, females 20-30 y, BMI < 24 kg/m^2, never smoked) PFNA and TSH, hypothyroidism (total, males and females 12-19 y, males and females 20-30 y) PFunDA and free T4, TSH, hypothyroidism (total, males and females 12-19 y, males and females 20-30 y, subgroups by BMI, smoking, and hypertension)
Thyroid	Lopez-Espinosa 2012a	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	50	PFOA PFOS	None	PFOA and free T4 PFOS and free T4
Thyroid	Lopez-Espinosa 2012b	Retrospective cohort and cross-sectional	Contaminated community (Mid-Ohio Valley)	Children, adolescents	10,725	PFOA PFOS PFNA	PFOA (modeled in utero) and greater total T4 level (all 1-5 y) PFOA (cross-sectional) and lower TSH level (all 1-5 y, girls 1-5 y) PFOA (cross-sectional) and greater total T4 level (girls 6-10 y) PFOA (cross-sectional) and greater risk of self-reported thyroid disease PFOS (cross-sectional) and greater total T4 level (all 6-10 y, all > 10 y, all 1-17 y; boys > 10 y, boys 1-17 y; girls 6-10 y, girls > 10 y, girls 1-17 y) PFNA (cross-sectional) and greater total T4 level (all 6-10 y, all > 10 y, all 1-17 y; boys > 10 y, boys 1-17 y; girls 6-10 y, girls 1-17 y)	PFOA (modeled in utero) and TSH (all age/sex subgroups) PFOA (modeled in utero) and total T4 (all age/sex subgroups except all 1-5 y PFOA (modeled in utero) and risk of self-reported thyroid disease or hypothyroidism, and subclinical hypothyroidism or hyperthyroidism PFOA (cross-sectional) and TSH (all age/sex subgroups except all age 1-5 y and girls 1-5 y) PFOA (cross-sectional) and total T4 (all age/sex subgroups except girls 6-10 y) PFOA (cross-sectional) and risk of self-reported hypothyroidism, and subclinical hypothyroidism or hyperthyroidism PFOS (cross-sectional and TSH) (all age/sex subgroups) PFOS (cross-sectional) and total T4 (all 1-5 y; boys 1-5 or 6-10 y; girls 1-5 y) PFOS (cross-sectional) and risk of self-reported thyroid disease or hypothyroidism, and subclinical hypothyroidism or hyperthyroidism PFNA (cross-sectional and TSH) (all age/sex subgroups) PFNA (cross-sectional) and total T4 (all 1-5 y; boys 1-5 or 6-17 y; girls 1-5 or > 10 y) PFNA (cross-sectional) and risk of self-reported thyroid disease or hypothyroidism, and subclinical hypothyroidism or hyperthyroidism

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Melzer 2010	Cross-sectional	General community (United States)	Adolescents, adults	2,066 women 1,900 men	PFOA PFOS	PFOA and greater risk of thyroid disease ever (women) PFOA and greater risk of thyroid disease current with medication (women) PFOS and greater risk of thyroid disease current with medication (men)	PFOA and thyroid disease ever (men) PFOA and thyroid disease current with medication (men) PFOS and thyroid disease ever (women, men) PFOS and thyroid disease current with medication (women)
Thyroid	Olsen 1998	Cross-sectional	Occupational (Cottage Grove, Minnesota)	Men	111	PFOA	PFOA and greater TSH level (1995)	PFOA and TSH (1993)
Thyroid	Olsen 2003a	Prospective cohort and cross-sectional	Occupational (Decatur, Alabama; Antwerp, Belgium)	Adults	263 Decatur 255 Antwerp	PFOS	PFOS and greater T3 level (men)	PFOS and TSH PFOS and T3 (women) PFOS and T4 PFOS and free T4
Thyroid	Olsen 2004	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,311	PFOS	None	PFOS and episodes of care for thyroid disorders
Thyroid	Olsen 2007	Cross-sectional	Occupational (Antwerp, Belgium; Cottage Grove, Minnesota; Decatur, Alabama)	Men	506	PFOA	PFOA and lower free T4 level (all) PFOA and greater T3 level (all, Antwerp, Decatur)	PFOA and TSH PFOA and T4 PFOA and free T4 (Antwerp, Cottage Grove, Decatur) PFOA and T3 (Cottage Grove)
Thyroid	Pirali 2009	Case-control	General community (Pavia, Italy)	Adults	9-12 goiter cases 8-10 autoimmune thyroid cases 7-10 controls	PFOA PFOS	None	PFOA and toxic/nontoxic multinodular goiter PFOA and Hashimoto's thyroiditis or Graves' disease PFOS and toxic/nontoxic multinodular goiter PFOS and Hashimoto's thyroiditis or Graves' disease
Thyroid	Raymer 2012	Cross-sectional	General community (Durham, North Carolina)	Men	252	PFOA PFOS	Plasma PFOS and greater T3 level	Plasma PFOA and TSH, T4, and T3 Plasma PFOS and TSH and T4 Semen PFOS and T3

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Shah-Kulkarni 2016	Cross-sectional	General community (Seoul, South Korea)	Newborns	279	PFPA PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA PFtrDA PFteDA	PFPA and lower total T3 level (boys) PFPA and greater total T4 level (total, girls) PFHxS and greater T3 level (girls) PFNA and lower TSH level (girls)	PFPA and total T3 (total, girls), total T4 (boys), and TSH (all) PFHxS and total T3 (total, boys), total T4 (all), and TSH (all) PFOA and total T3, total T4, and TSH (all) PFOS and total T3, total T4, and TSH (all) PFNA and total T3 (all), total T4 (all), and TSH (total, boys) PFDA and total T3, total T4, and TSH (all) PFunDA and total T3, total T4, and TSH (all) PFdoDA and total T3, total T4, and TSH (all) PFtrDA and total T3, total T4, and TSH (all) PFteDA and total T3, total T4, and TSH (all)
Thyroid	Shrestha 2015	Cross-sectional	PCB-contaminated community (Hudson River region, New York)	Older adults	87	PFOA PFOS	PFOA×age and free T4 level PFOA×age and total T4 PFOS and greater total T4 level PFOS and greater free T4 level	PFOA and TSH PFOA and total T3 PFOA and total T4 PFOA and free T4 PFOA×age and TSH PFOA×age and total T3 PFOS and TSH PFOS and total T3
Thyroid	Steenland 2015	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	3,713	PFOA	None	PFOA and male thyroid disease PFOA and female thyroid disease
Thyroid	Tsai 2017	Cross-sectional	General community (Taipei, Taiwan)	Newborns	118	PFOA PFOS PFNA PFunDA	PFOS and greater TSH level (total, boys) PFOS and lower total T4 level (total, boys) PFNA and lower total T4 level (boys) PFunDA and greater total T3 level (total, 60-89th pctl, not ≥ 90th or continuous)	PFOA and TSH (total, girls, boys) PFOA and total T3 (total, girls, boys) PFOA and total T4 (total, girls, boys) PFOS and TSH (girls) PFOS and total T3 (total, girls, boys) PFOS and total T4 (girls) PFNA and TSH (total, girls, boys) PFNA and total T3 (total, girls, boys) PFNA and total T4 (total, girls) PFunDA and TSH (total, girls, boys) PFunDA and total T3 (girls, boys) PFunDA and total T4 (total, girls, boys)

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Wang 2013	Cross-sectional	General community (Norway)	Women	903	PFHpS PFHxS PFOA PFOS PFNA PFDA PFunDA	PFOS and greater TSH level	PFHpS and TSH PFHxS and TSH PFOA and TSH PFNA and TSH PFDA and TSH PFunDA and TSH
Thyroid	Wang 2014	Prospective cohort and cross-sectional	General community (Taiwan)	Newborns, women	116 newborns 285 women	PFHxS PFOA PFOS PFNA PFDA PFunDA PFdoDA	PFHxS and greater maternal TSH level PFNA and lower maternal free T4 level PFNA and lower maternal total T4 level PFNA and lower neonatal total T4 level PFNA and lower neonatal total T3 level PFDA and greater maternal total T3 level PFDA and lower neonatal total T3 level PFunDA and lower maternal free T4 level PFunDA and lower maternal total T4 level PFunDA and lower neonatal total T4 level PFunDA and lower neonatal total T3 level PFdoDA and lower maternal free T4 level PFdoDA and lower maternal total T4 level PFdoDA and lower neonatal total T4 level PFdoDA and lower neonatal total T3 level	PFHxS and maternal free T4, total T4, and total T3 PFHxS and neonatal free T4, total T4, total T3, and TSH PFOA and maternal free T4, total T4, total T3, and TSH PFOA and neonatal free T4, total T4, total T3, and TSH PFOS and maternal free T4, total T4, total T3, and TSH PFOS and neonatal free T4, total T4, total T3, and TSH PFNA and maternal total T3 and TSH PFNA and neonatal free T4 and TSH PFDA and maternal free T4, total T4, and TSH PFDA and neonatal free T4, total T4, and TSH PFunDA and maternal total T3 and TSH PFunDA and neonatal free T4 and TSH PFdoDA and maternal total T3 and TSH PFdoDA and neonatal free T4 and TSH
Thyroid	Webster 2014	Cross-sectional (repeated)	General community (Vancouver, Canada)	Women	152	PFHxS PFOA PFOS PFNA	PFOA and greater TSH level (high anti-thyroid peroxidase antibody) PFOS and greater TSH level (high anti-thyroid peroxidase antibody) PFNA and greater TSH level (all, high anti-thyroid peroxidase antibody)	PFHxS and free T4 (all, normal or high anti-thyroid peroxidase antibody) PFHxS and total T4 (all, normal or high anti-thyroid peroxidase antibody) PFHxS and TSH (all, normal or high anti-thyroid peroxidase antibody) PFOA and free T4 (all, normal or high anti-thyroid peroxidase antibody) PFOA and total T4 (all, normal or high anti-thyroid peroxidase antibody) PFOA and TSH (all, normal anti-thyroid peroxidase antibody) PFOS and free T4 (all, normal or high anti-thyroid peroxidase antibody) PFOS and total T4 (all, normal or high anti-thyroid peroxidase antibody) PFOS and TSH (all, normal anti-thyroid peroxidase antibody) PFNA and free T4 (all, normal or high anti-thyroid peroxidase antibody) PFNA and total T4 (all, normal or high anti-thyroid peroxidase antibody) PFNA and TSH (normal high anti-thyroid peroxidase antibody)

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Webster 2016	Cross-sectional	General community (United States)	Adults	1,525	PFHxS PFOA PFOS PFNA	PFHxS and greater total T3 level (high anti-thyroid peroxidase antibody, normal urinary iodine) PFOA and greater free T3 level (normal anti-thyroid peroxidase antibody and urinary iodine) Among subjects with high anti-thyroid peroxidase antibody and low urinary iodine: PFHxS and greater free T3 level PFHxS and lower free T4 level PFHxS and greater free T3/free T4 ratio PFHxS and greater TSH level PFHxS and greater total T3 level PFOA and greater free T3 level PFOA and greater free T3/free T4 ratio PFOA and greater TSH level PFOA and greater total T3 level PFOS and greater free T3 level PFOS and lower free T4 level PFOS and greater free T3/free T4 ratio PFOS and greater TSH level PFOS and greater total T3 level PFNA and greater free T3 level PFNA and greater free T3/free T4 ratio PFNA and greater TSH level PFNA and greater total T3 level	PFHxS and free T3 (all groups except high antibody/low iodine) PFHxS and free T4 (all groups except high antibody/low iodine) PFHxS and free T3/free T4 ratio (all groups except high antibody/low iodine) PFHxS and TSH (all groups except high antibody/low iodine) PFHxS and total T3 (all groups except high antibody/normal iodine and high antibody/low iodine) PFHxS and total T4 (all groups) PFOA and free T3 (all groups except normal antibody/normal iodine and high antibody/low iodine) PFOA and free T4 (all groups) PFOA and free T3/free T4 ratio (all groups except high antibody/low iodine) PFOA and TSH (all groups except high antibody/low iodine) PFOA and total T3 (all groups except high antibody/low iodine) PFOA and total T4 (all groups) PFOS and free T3 (all groups except high antibody/low iodine) PFOS and free T4 (all groups except high antibody/low iodine) PFOS and free T3/free T4 ratio (all groups except high antibody/low iodine) PFOS and TSH (all groups except high antibody/low iodine) PFOS and total T3 (all groups except high antibody/low iodine) PFOS and total T4 (all groups) PFNA and free T3 (all groups except high antibody/low iodine) PFNA and free T4 (all groups) PFNA and free T3/free T4 ratio (all groups except high antibody/low iodine) PFNA and TSH (all groups except high antibody/low iodine) PFNA and total T3 (all groups except high antibody/low iodine) PFNA and total T4 (all groups)

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Wen 2013	Cross-sectional	General community (United States)	Adults	672 men 509 women	PFHxS PFOA PFOS PFNA	PFHxS and greater total T4 level (women) PFHxS and lower free T4 level (men) PFHxS and greater total T3 level (women) PFHxS and greater risk of subclinical hypothyroidism (women) PFHxS and greater risk of subclinical hyperthyroidism (women) PFOA and greater total T3 level (women) PFOA and greater risk of subclinical hypothyroidism (women) PFOA and lower risk of subclinical hyperthyroidism (men) PFOS and greater risk of subclinical hypothyroidism (men, women) Adjusting for all 4 PFAS*: PFHxS and greater total T4 level (women) PFHxS and greater total T3 level (women) PFOS and lower free T3 level (women) PFOS and greater thyroglobulin level (women) *All other associations tested when adjusting for other PFAS were statistically non-significant.	PFHxS and total T4 (men) PFHxS and free T4 (women) PFHxS and total T3 (men) PFHxS and free T3 (men, women) PFHxS and TSH (men, women) PFHxS and thyroglobulin (men, women) PFHxS and subclinical hypothyroidism (men) and subclinical hyperthyroidism (men) PFOA and total T4 (men, women) PFOA and free T4 (men, women) PFOA and total T3 (men) PFOA and free T3 (men, women) PFOA and TSH (men, women) PFOA and thyroglobulin (men, women) PFOA and subclinical hypothyroidism (men) and subclinical hyperthyroidism (women) PFOS and total T4 (men, women) PFOS and free T4 (men, women) PFOS and total T3 (men, women) PFOS and free T3 (men, women) PFOS and TSH (men, women) PFOS and thyroglobulin (men, women) PFOS and subclinical hyperthyroidism (men, women) PFNA and total T4 (men, women) PFNA and free T4 (men, women) PFNA and total T3 (men, women) PFNA and free T3 (men, women) PFNA and TSH (men, women) PFNA and thyroglobulin (men, women)

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Winquist 2014b	Prospective and retrospective cohort	Contaminated community (Mid-Ohio Valley) and occupational (Parkersburg, West Virginia)	Adults	32,254	PFOA	PFOA (cumulative, retrospective) and greater risk of validated functional thyroid disease (total, women) PFOA (cumulative, retrospective) and greater risk of validated hypothyroidism (total, women) PFOA (annual, retrospective) and greater risk of validated functional thyroid disease (total, women) PFOA (annual, retrospective) and greater risk of validated hyperthyroidism (total, women) PFOA (annual, retrospective) and greater risk of validated hypothyroidism (total, women) PFOA (cumulative, prospective) and greater risk of validated hypothyroidism (men) PFOA (annual, prospective) and greater risk of validated functional thyroid disease (men in quintile 3, not 4 or 5) PFOA (annual, prospective) and greater risk of validated hypothyroidism (men in quintiles 3 and 4, not 5) PFOA (serum, baseline) and lower risk of validated hyperthyroidism (men in quintiles 4 and 5, no trend)	PFOA (cumulative, retrospective) and risk of validated functional thyroid disease (men) PFOA (cumulative, retrospective) and risk of validated hyperthyroidism (total, women, men) PFOA (cumulative, retrospective) and risk of validated hypothyroidism (men) PFOA (annual, retrospective) and risk of validated functional thyroid disease (men) PFOA (annual, retrospective) and risk of validated hyperthyroidism (men) PFOA (annual, retrospective) and risk of validated hypothyroidism (men) PFOA (cumulative, prospective) and risk of validated functional thyroid disease (total, women, men) PFOA (cumulative, prospective) and risk of validated hyperthyroidism (total, women, men) PFOA (cumulative, prospective) and risk of validated hypothyroidism (total, women) PFOA (annual, prospective) and risk of validated functional thyroid disease (total, women) PFOA (annual, prospective) and risk of validated hyperthyroidism (total, women, men) PFOA (annual, prospective) and risk of validated hypothyroidism (total, women) PFOA (serum, baseline) and risk of validated functional thyroid disease (total, women, men) PFOA (serum, baseline) and risk of validated hyperthyroidism (total, women) PFOA (serum, baseline) and risk of validated hypothyroidism (total, women, men)

Table 18. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and thyroid outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Thyroid	Yang 2016	Cross-sectional	General community (Beijing, China)	Newborns, women	157	PFHxS PFOA PFOS Me-PFOSA-AcOH PFNA PFDA PFunDA PFdoDA 6:2 fluorotelomer sulfonates (6:2 FTS)	Fetal PFHxS and lower maternal free T3 and maternal total T3 levels Fetal PFOA and lower maternal free T3 level Maternal PFOS and lower maternal TSH level Fetal PFOS and lower maternal free T3, maternal total T3, and maternal TSH levels Maternal Me-PFOSA-AcOH and greater maternal free T3 level Maternal PFNA and lower maternal TSH level Fetal PFNA and lower maternal free T3 and maternal total T3 levels Maternal PFDA and lower maternal TSH level Fetal PFDA and lower maternal free T3 and maternal total T3 levels Maternal PFunDA and lower maternal TSH level Fetal PFunDA and lower maternal free T3 and maternal total T3 levels Maternal PFdoDA and lower maternal free T3, lower maternal free T4, lower maternal total T3, lower maternal total T4, and lower maternal TSH levels Fetal PFdoDA and lower maternal free T3 and maternal total T3 levels Maternal 6:2 FTS and lower maternal free T3, lower maternal free T4, lower maternal total T3, and lower maternal total T4 levels	Maternal PFHxS and maternal free T3, maternal free T4, maternal total T3, maternal total T4, and maternal TSH Fetal PFHxS and maternal free T4, maternal total T4, and maternal TSH Maternal PFOA and maternal free T3, maternal free T4, maternal total T3, maternal total T4, and maternal TSH Fetal PFOA and maternal free T4, maternal total T3, maternal total T4, and maternal TSH Maternal PFOS and maternal free T3, maternal free T4, maternal total T3, and maternal total T4 Fetal PFOS and maternal free T4 and maternal total T4 Maternal Me-PFOSA-ACOH and maternal free T4, maternal total T3, maternal total T4, and maternal TSH Fetal Me-PFOSA-ACOH and maternal free T3, maternal free T4, maternal total T3, maternal total T4, and maternal TSH Maternal PFNA and maternal free T3, maternal free T4, maternal total T3, and maternal total T4 Fetal PFNA and maternal free T4, maternal total T4, and maternal TSH Maternal PFDA and maternal free T3, maternal free T4, maternal total T3, and maternal total T4 Fetal PFDA and maternal free T4, maternal total T4, and maternal TSH Maternal PFunDA and maternal free T3, maternal free T4, maternal total T3, and maternal total T4 Fetal PFunDA and maternal free T4, maternal total T4, and maternal TSH Fetal PFdoDA and maternal free T4, maternal total T4, and maternal TSH Maternal 6:2 FTS and maternal TSH Fetal 6:2 FTS and maternal free T3, maternal free T4, maternal total T3, maternal total T4, and maternal TSH

Table 19. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and other health outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Other	Caserta 2013	Case-control	General community (Rome, Ferrara, and Sora, Italy)	Women	111	PFOA PFOS	PFOA and lower gene expression of aryl hydrocarbon receptor (*AHR*) (infertile women) PFOA and lower gene expression of pregnane X receptor (*PXR)* (fertile women) PFOS and greater gene expression of androgen receptor (*AR*) (infertile women) PFOS and greater gene expression of *PXR* (infertile women)	PFOA and gene expression of estrogen receptor alpha (*ER-alpha*) (infertile and fertile women) PFOA and gene expression of *ER-beta* (infertile and fertile women) PFOA and gene expression of *AR* (infertile and fertile women) PFOA and gene expression of *PXR* (infertile women) PFOA and gene expression of *AHR* (fertile women) PFOA and gene expression of peroxisome proliferator-activated receptor gamma (PPAR-gamma) (infertile and fertile women) PFOS and gene expression of *ER-alpha* (infertile and fertile women) PFOS and gene expression of *ER-beta* (infertile and fertile women) PFOS and gene expression of *AR* (fertile women) PFOS and gene expression of *PXR* (fertile women) PFOS and gene expression of *AHR* (infertile and fertile women) PFOS and gene expression of *PPAR-gamma* (infertile and fertile women)
Other	Consonni 2013	Retrospective cohort	Occupational (Germany, Netherlands, Italy, United Kingdom, New Jersey, West Virginia)	Adults	4,773	PFOA/tetrafluoroethylene (TFE)	None	PFOA and benign/uncertain/unspecified tumor mortality PFOA and digestive disease mortality

Table 19. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and other health outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Other	Fletcher 2013	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	290	PFOA PFOS	PFOA and lower expression of *ABCG1* (cholesterol transport gene) (total, men) PFOA and lower expression of *APOA1* (cholesterol transport gene) (postmenopausal) PFOA and greater expression of *NCEH1* (cholesterol mobilization gene) (women) PFOA and lower expression of *NPC1* (cholesterol mobilization gene) (total, men) PFOA and lower expression of *NR1H2 (LXRB)* (transcriptional activation of PPAR-alpha target gene) (total, women, premenopausal) PFOA and lower expression of *PPARA* (peroxisome proliferation gene) (men) PFOA and lower expression of *PPARD* (peroxisome proliferation gene) (premenopausal) PFOS and lower expression of *NCEH1* (cholesterol mobilization gene) (total, women, premenopausal) PFOS and lower expression of *NR1H3 (LXRA)* (transcriptional activation of PPAR-alpha target gene) (total, premenopausal) PFOA and lower expression of *PPARA* (peroxisome proliferation gene) (women, premenopausal)	PFOA and expression of *ABCA1* PFOA and expression of *ABCG1* (women) PFOA and expression of *ACAT1* PFOA and expression of *APOA1* (total, premenopausal) PFOA and expression of *HMGCR* PFOA and expression of *NCEH1* (total, men) PFOA and expression of *NPC1* (women) PFOA and expression of *NR1H2 (LXRB)* (men, postmenopausal) PFOA and expression of *NR1H3 (LXRA)* PFOA and expression of *PPARA* (total, women) PFOA and expression of *PPARD* (total, postmenopausal) PFOA and expression of *PPARGC1A* (peroxisome proliferator-activated receptor gamma coactivator 1-alpha) PFOA and expression of *PPARG* PFOS and expression of *ABCA1* PFOS and expression of *ABCG1* PFOS and expression of *ACAT1* PFOS and expression of *APOA1* PFOS and expression of *HMGCR* PFOS and expression of *NCEH1* (men, postmenopausal) PFOS and expression of *NPC1* PFOS and expression of *NR1H2 (LXRB)* PFOS and expression of *NR1H3 (LXRA)* (women, men, postmenopausal) PFOS and expression of *PPARA* (total, men, postmenopausal) PFOS and expression of *PPARD* PFOS and expression of *PPARGC1A* PFOS and expression of *PPARG*
Other	Franken 2017	Cross-sectional	General community (Flanders and two industrial areas, Genk-Zuid and Menen, Belgium)	Adolescents	606	PFOA	PFOA and greater % DNA migration by alkaline comet assay (not significant after p value adjustment for false discovery rate)	PFOA and % DNA migration by formamidopyrimidine DNA glycosylase (FPG)-modified comet assay PFOA and urinary 8-hydroxydeoxyguanosine
Other	Gilliland 1993	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,537	PFOA	PFOA and lower risk of mortality from gastrointestinal disease (men)	PFOA and mortality from gastrointestinal disease (women, men in chemical division)
Other	Goudarzi 2017a	Prospective cohort	General community (Sapporo, Japan)	Newborns	185	PFOA PFOS	PFOS and lower cortisol level PFOS and lower cortisone level PFOS and greater cortisol/cortisone ratio PFOS and lower cortisol/dehydroepiandrosterone ratio PFOS and lower glucocorticoid/androgenic hormones ratio	PFOA and cortisol level PFOA and cortisone level PFOA and cortisol/cortisone ratio PFOA and cortisol/dehydroepiandrosterone ratio PFOA and glucocorticoid/androgenic hormones ratio

Table 19. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and other health outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Other	Grice 2007	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,400	PFOS	None	PFOS and cystitis PFOS and bladder calculi PFOS and colon polyps PFOS and gastric ulcer PFOS and benign prostatic hyperplasia PFOS and prostatitis
Other	Kim 2016b	Randomized controlled trial	General community (Seoul, South Korea)	Older adults	126	PFOS PFdoDA	PFOS and greater malondialdehyde level (baseline and placebo) PFOS and greater 8-hydroxy-2'-deoxyguanosine level (baseline and placebo) PFdoDA and greater 8-hydroxy-2'-deoxyguanosine level (baseline and placebo)	PFOS and malondialdehyde (vitamin C supplementation) PFOS and 8-hydroxy-2'-deoxyguanosine (vitamin C supplementation) PFdoDA and malondialdehyde (baseline and placebo; vitamin C supplementation) PFdoDA and 8-hydroxy-2'-deoxyguanosine (vitamin C supplementation)
Other	Kingsley 2017	Prospective cohort	General community (Cincinnati, Ohio)	Infants	44	PFOA	PFOA and cord blood leukocyte methylation at 7 CpG sites in 3 genes (*RASA3* , *UCK1* , and *OPRD1*), especially in promoter regions	PFOA and cord blood leukocyte methylation at any CpG site
Other	Kobayashi 2017	Prospective cohort	General community (Hokkaido, Japan)	Newborns	177	PFOA PFOS	PFOA and lower insulin growth factor 2 (*IGF2*) methylation in cord blood	PFOA and *H19* (*IGF2* locus) methylation in cord blood PFOA and long interspersed element 1 (*LINE1*) methylation in cord blood PFOS and *IGF2* methylation in cord blood PFOS and *H19* methylation in cord blood PFOS and *LINE1* methylation in cord blood
Other	La Rocca 2015	Case-control	General community (Rome, Ferrara, and Sora, Italy)	Men	70 cases 83 controls	PFOA PFOS	PFOA in blood and lower nuclear receptor gene expression (estrogen receptor-alpha, estrogen receptor-beta, androgen receptor, aryl hydrocarbon receptor, and pregnane X receptor)	PFOA in blood and peroxisome proliferator-activated receptor-gamma nuclear receptor gene expression PFOS in blood and nuclear receptor gene expression (estrogen receptor-alpha, estrogen receptor-beta, androgen receptor, aryl hydrocarbon receptor, pregnane X receptor, and peroxisome proliferator-activated receptor-gamma nuclear receptor gene expression)
Other	Leonard 2008	Retrospective cohort	Occupational (Parkersburg, West Virginia)	Adults	6,027	PFOA	None	PFOA and mortality from benign neoplasms

Table 19. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and other health outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Other	Lind 2017a	Prospective cohort	General community (Odense, Denmark)	Infants	299 boys 212 girls	PFHxS PFOA PFOS PFNA PFDA	PFHxS and shorter anogenital distance to posterior base of scrotum (boys) PFHxS and shorter anogenital distance to posterior fourchette (girls) PFOS and greater anogenital distance to posterior base of scrotum (boys) PFOS and shorter anogenital distance to posterior fourchette (girls) PFNA and shorter anogenital distance to posterior fourchette (girls) PFDA and greater anogenital distance to posterior base of scrotum (boys) PFDA and shorter anogenital distance to posterior fourchette (girls)	PFHxS and anogenital distance to cephalad insertion of penis and penile width (boys) PFHxS and anogenital distance to top of clitoris (girls) PFOA and anogenital distance to posterior base of scrotum, anogenital distance to cephalad insertion of penis, and penile width (boys) PFOA and anogenital distance to posterior fourchette and anogenital distance to top of clitoris (girls) PFOS and anogenital distance to cephalad insertion of penis and penile width (boys) PFOS and anogenital distance to top of clitoris (girls) PFNA and anogenital distance to posterior base of scrotum, anogenital distance to cephalad insertion of penis, and penile width (boys) PFNA and anogenital distance to top of clitoris (girls) PFDA and anogenital distance to cephalad insertion of penis and penile width (boys) PFDA and anogenital distance to top of clitoris (girls)
Other	Lundin 2009	Retrospective cohort	Occupational (Cottage Grove, Minnesota)	Adults	3,993	PFOA	None	PFOA and mortality from benign neoplasms PFOA and mortality from ulcer of stomach
Other	Olsen 2000	Cross-sectional	Occupational (Cottage Grove, Minnesota)	Men	74	PFOA	PFOA and lower cholecystokinin level in 1997	None
Other	Olsen 2004	Retrospective cohort	Occupational (Decatur, Alabama)	Adults	1,311	PFOS	PFOS and greater risk of episodes of care for benign colonic polyps (long-term chemical workers) PFOS and greater risk of episodes of care for cystitis (long-term chemical workers) PFOS and greater risk of episodes of care for urinary tract infections, not specified (long-term chemical workers)	PFOS and episodes of care for benign colonic polyps (shorter-term chemical workers) PFOS and episodes of care for benign neoplasm of skin PFOS and episodes of care for other benign/unspecified neoplasms PFOS and episodes of care for acute pancreatitis PFOS and episodes of care for cystitis (shorter-term chemical workers) PFOS and episodes of care for urinary tract infections, not specified (shorter-term chemical workers) PFOS and episodes of care for calculus of urinary tract PFOS and episodes of care for upper urinary tract infections PFOS and episodes of care for prostatic hyperplasia PFOS and episodes of care for acute prostatitis

Table 19. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and other health outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Other	Pennings 2016	Prospective cohort	General community (Norway)	Infants, young children	NR	PFHxS PFOA PFOS PFNA	27 genes overlapping between those correlated with number of common cold episodes up to 3 years and those correlated with PFAS exposure, including 3 related to immunological and/or hematopoietic functions and 6 involved in development and/or morphogenesis 26 genes overlapping between those correlated with anti-rubella antibody level at 3 years and those correlated with PFAS exposure, including 3 related to immunological and/or hematopoietic functions and 7 involved in development and/or morphogenesis	553 other genes correlated with number of common cold episodes up to 3 years 1,205 other genes correlated with anti-rubella antibody level at 3 years PFAS exposure and other genes involved in development and/or morphogenesis at age 3 years (not described)
Other	Toft 2016	Nested prospective case-control	General community (Denmark)	Newborn boys	270 cryptorchidism cases 75 hypospadias cases 300 controls	PFOS	PFOS and greater cortisol level	None
Other	Vagi 2014	Case-control	General community (Los Angeles, California)	Women	52 cases 50 controls	PFHxS PFOA PFOS PFNA	PFOA and greater risk of polycystic ovary syndrome PFOS and greater risk of polycystic ovary syndrome	PFHxS and polycystic ovary syndrome PFNA and polycystic ovary syndrome
Other	Vriens 2017	Cross-sectional	General community (Flanders, Belgium)	Newborns	233	PFHxS PFOA PFOS PFNA	None	PFHxS and placental mitochondrial DNA content PFOA and placental mitochondrial DNA content PFOS and placental mitochondrial DNA content PFNA and placental mitochondrial DNA content
Other	Wang 2012	Cross-sectional	Occupational and contaminated community (Changshu City, Jiangsu Province, China)	NR (includes adults)	55 workers 132 residents	PFOA	PFOA and greater levels of circulating miR-24, miR-26b, miR-30b, miR-30c, miR-92a, miR-106b, miR-127, miR-199a-3p, and miR-601	None

Table 19. Published epidemiologic studies of associations between perfluoroalkyl/polyfluoroalkyl substances (PFAS) and other health outcomes

Group	Reference	Study Design	Study Setting	Age Group	Total N (Max)	Exposure	Significant Associations	Nonsignificant Associations
Other	Wang 2017b	Cross-sectional	General community (Shandong, China)	Men	181	PFOA PFOS ΣPFAS (PFHxS+PFOA+PFOS+PFNA+PFDA+PFunDA)+(PFBA+PFBS+PFHxA+PFHpA+PFdoDA, undetectable)	PFOA and deoxyarabinohexonic acid (fatty acid metabolism), alpha-carboxyethyl hydroxychromanol (alpha-tocopherol metabolism), arachidonic acid (fatty acid metabolism), D-glucurono-6,3-lactone (fatty acid and ascorbate metabolism), hypoxanthine (purine metabolism), oxoglutaric acid (Krebs cycle), pyroglutamic acid (glutathione cycle), tetrahydrobiopterin (nitric oxide generation), and xanthine (purine metabolism) PFOS and deoxyarabinohexonic acid, D-glucurono-6,3-lactone, hydroxybutyric acid (glutathione cycle), hypoxanthine, oxoglutaric acid, pyroglutamic acid, tetrahydrobiopterin, and xanthine ΣPFAS and deoxyarabinohexonic acid, D-glucurono-6,3-lactone, hydroxybutyric acid, hypoxanthine, oxoglutaric acid, pyroglutamic acid, tetrahydrobiopterin, and xanthine	PFOA, PFOS, ΣPFAS and other serum metabolome biomarkers (number and types not stated)
Other	Watkins 2014	Cross-sectional	Contaminated community (Mid-Ohio Valley)	Adults	685	PFHxS PFOA PFOS PFNA	PFOS and greater % LINE-1 DNA methylation	PFHxS and % LINE-1 DNA methylation PFOA and % LINE-1 DNA methylation PFNA and % LINE-1 DNA methylation

Appendix B

Curriculum Vitae of Ellen T. Chang, Sc.D.

Exponent®
Engineering & Scientific Consulting

Ellen Chang, Sc.D.

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Professional Profile

Dr. Chang has 18 years of experience in designing, conducting, and interpreting epidemiologic studies, with a particular focus on studies of cancer and other chronic diseases. She provides scientific consultation on the potential human health effects of various chemicals (such as dioxins, chlorinated solvents, pesticides, PCBs, and perfluoroalkyl and polyfluoroalkyl substances), air pollutants, metals and metalloids, fibers, pharmaceuticals, medical devices, electromagnetic fields, and nutrients. She has expertise in qualitatively and quantitatively synthesizing the weight of epidemiologic evidence on causal effects of environmental exposures.

Dr. Chang's recent projects include evaluations of the epidemiologic evidence on glyphosate, TCDD, and perchloroethylene in association with non-Hodgkin lymphoma and other cancers; perfluoroalkyl and polyfluoroalkyl substances in association with immune-related and other health conditions; fine particulate matter and ozone in association with all-cause and cause-specific mortality; and organophosphate insecticides in association with birth and developmental outcomes. Dr. Chang also frequently conducts and coordinates analyses of cancer incidence, mortality, and survival in population-based cancer registries.

Dr. Chang has led original research studies of cancers of the head and neck, nasopharynx, stomach, liver, lung and bronchus, skin, breast, uterus, ovary, prostate, thyroid, and lymphatic system. These studies focused on a wide range of exposures including genetic variation, physical activity, body size, diet and nutrition, alcohol consumption, tobacco smoking, ultraviolet radiation, immunologic biomarkers, microbial infections, use of nonsteroidal anti-inflammatory drugs and other medications, use of hormone therapy and oral contraceptives, reproductive factors, medical history, family structure, and demographic characteristics. In addition, Dr. Chang has conducted cancer surveillance research at one of the U.S. National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) registries, and contributed to community-based research on hepatitis B and liver cancer awareness, detection, prevention, and medical management at the Asian Liver Center at Stanford University.

Dr. Chang earned her undergraduate degree in English and American literature and language from Harvard College. She earned her Sc.D. (Doctor of Science) in epidemiology with a minor in biostatistics from the Harvard School of Public Health, and she completed a post-doctoral fellowship at the Karolinska Institute. She is a member of the Stanford Cancer Institute and a former Consulting Assistant Professor in the Division of Epidemiology, Department of Health Research and Policy at the Stanford University School of Medicine. Dr. Chang has published more than 160 peer-reviewed research articles and reviews, and 11 book chapters.

Academic Credentials & Professional Honors

Sc.D., Epidemiology, Harvard University, 2003

A.B., English and American Literature and Language, Harvard University, 1998

National Cancer Institute Minority Investigators Workshop on Behavioral Methodologies Fellowship, 2007

New York Academy of Sciences (NYAS) Science Alliance Program Membership, 2005-2006

National Institutes of Health Ruth L. Kirschstein National Research Service Award, 2004-2005

American Association for the Advancement of Science (AAAS)/Science Program for Excellence in Science Membership, 2004-2005

Harvard University Sheldon Traveling Fellowship, 2003-2004

Harvard School of Public Health Department of Epidemiology Seiden Scholarship, 2001-2003

Harvard University Pforzheimer Public Service Fellowship, 1999-2003

National Cancer Institute/Harvard School of Public Health Cancer Epidemiology Pre-Doctoral Training Program Fellowship, 1998-2002

Academic Appointments

Member, Stanford Cancer Institute, 2005-present

Prior Experience

Consulting Assistant Professor, Division of Epidemiology, Department of Health Research and Policy, Stanford University School of Medicine, 2005-2016

Research Scientist, Cancer Prevention Institute of California, 2005-2012

Consulting Assistant Investigator, Department of Health Policy Research, Palo Alto Medical Foundation Research Institute, 2008-2012

Chief Epidemiologist, Asian Liver Center at Stanford University, 2006-2011

Professional Affiliations

American Association for Cancer Research

Society for Epidemiologic Research

Languages

Mandarin

French

Publications

Chen V, Le A, Podlaha O, Estevez J, Li B, Vutien P, Chang E, Rosenberg-Hasson Y, Pflanz S, Jiang Z, Ge D, Gaggar A, Nguyen M. Soluble intercellular adhesion molecule-1 is associated with hepatocellular carcinoma risk: multiplex analysis of serum markers. Scientific Reports 2017; 7(1): 11169.

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Kurtz SM, Lau E, Chang E, Son M, Zimmerli W, Parvizi J. Are we winning or losing the war with PJI: trends in PJI and mortality risk for the Medicare population. Abstract and presentation at The Knee Society 2017, Naples, Florida, September 14-16, 2017.

Chen VL, Podlaha O, Estevez J, Li B, Le A, Vutien P, Chang ET, Pflanz S, Jiang Z, Ge D, Gaggar A, Nguyen MH. High serum soluble intracellular adhesion molecule 1 (sICAM-1) concentration is assocated with hepatocellular carcinoma development in hepatitis B virus, hepatitis C virus, and non-viral liver disease: Multiplex analysis of 51 cytokines and other serum markers. Abstract and presentation at Digestive Disease Week 2016, San Diego, California, May 21-24, 2016.

Estevez J, Chen VL, Podlaha O, Li B, Le A, Vutien P, Chang ET, Jiang Z, Pflanz S, Ge D, Gaggar A, Nguyen MH. Differential cytokine profiles in patients with hepatocellular carcinoma related to hepatitis B virus (HBV) and hepatitis C virus (HCV) infection. Abstract and presentation at Digestive Disease Week 2016, San Diego, California, May 21-24, 2016.

Chang ET. Weight-of-evidence synthesis in epidemiology: What's the bottom line? Presentation at DRI Toxic Torts and Environmental Law Seminar, New Orleans, Louisiana, March 17-18, 2016.

Chen VL, Vutien P, Li B, Podlaha O, Chang ET, Jiang Z, Ge D, Gaggar A, Nguyen MH. Differential serum cytokine profiles in patients with hepatitis B virus (HBV), hepatitis C virus (HCV), and non-viral non-autoimmune liver disease, with or without hepatocellular carcinoma (HCC). Abstract and poster at The AASLD Liver Meeting 2015, San Francisco, California, November 13-17, 2015.

Plenary speaker. NPC international incidence and risk factors. 7th International Biannual Symposium on Nasopharyngeal Carcinoma 2015, Yogyakarta, Indonesia, June 3-6, 2015.

Session chair for plenary session on genetics and epigenetics of nasopharyngeal carcinoma. 7th International Biannual Symposium on Nasopharyngeal Carcinoma 2015, Yogyakarta, Indonesia, June 3-6, 2015.

Invited speaker. Reanalysis of the Diesel Exhaust in Miners Study (DEMS) cohort. Mario Negri Institute for Pharmacological Research, Milan, Italy, March 12, 2015.

Epstein MM, Chang ET, Zhang Y, Fung T, Batista JL, Ambinder RF, Zheng T, Mueller NE, Birmann BM. Diet patterns and risk of Hodgkin lymphoma in a population-based case-control study. Abstract and

presentation at 2014 InterLymph Annual Meeting, Los Angeles, CA, June 17-20, 2014.

Hjalgrim H, Monnereau A, Glaser SL, Chang E. Risk factors for classical Hodgkin lymphoma. Presentation at 2013 InterLymph Annual Meeting, Dijon, France, June 24-26, 2013.

Birmann BM, Epstein MM, Chang ET, Zhang Y, Fung T, Kasperzyk J, Ambinder RF, Zheng T, Mueller NE. Dietary patterns and risk of Hodgkin lymphoma in a population-based case-control study [Abstract 491-S]. American Journal of Epidemiology 2013; 177(11Suppl): S123. Poster at 46th Annual Society for Epidemiologic Research (SER) Meeting, Boston, MA, June 18-21, 2013.

Gao L, Chang E, Nelson D, Vutien P, Rosenberg-Hassan Y, Nguyen MH. Serum cytokine profiles and hepatocellular carcinoma (HCC) in patients with chronic hepatitis C. Poster at Digestive Disease Week, Orlando, FL, May 18–21, 2013.

Vutien P, Chang E, Nelson D, Gao L, Rosenberg-Hassan Y, Nguyen MH. Serum cytokine profiles in patients with hepatitis B virus (HBV) infection and associated hepatocellular carcinoma (HCC). Poster at Digestive Disease Week, Orlando, FL, May 18–21, 2013.

Monnereau A, Glaser SL, Chang ET. Ultraviolet radiation (UVR) and risk of Hodgkin lymphoma: A pooled analysis. Presentation at InterLymph Consortium Annual Meeting, Washington, DC, June 7, 2012.

Invited speaker. Gastric cancer incidence patterns in California Hispanics. Northern California Cancer Registrars Association Conference, Fremont, CA, December 14, 2011.

Ai W, Chang E, Fu K, Fish K, Weisenburger DD, Keegan T. Racial/ethnic patterns of NK/T cell lymphoma in California: A population-based study. Abstract at International Conference on Malignant Lymphoma, Lugano, Switzerland, June 15-18, 2011.

Colevas AD, Clarke CA, Lichtensztajn D, Chang ET. A population-based evaluation of incidence trends in oropharynx cancer focusing on socioeconomic status, sex, and race/ethnicity. Poster at American Society of Clinical Oncology (ASCO) Annual Meeting, Chicago, IL, June 4-8, 2010.

Clément-Duchêne C, Xu X, Gomez SL, Chang ET, West DW, Wakelee HA, Gould MK. Survival among never and ever smokers with lung cancer in the Cancer Care Outcomes Research and Surveillance Consortium (CanCORS) study. Poster at American Society of Clinical Oncology (ASCO) Annual Meeting, Chicago, IL, June 4-8, 2010.

Chang ET. Ultraviolet radiation exposure & sensitivity and risk of Hodgkin lymphoma: A pooled analysis. Presentation at InterLymph Consortium 9th Annual Meeting, Washington, DC, April 15, 2010.

Chang ET, Nguyen BH, So SK. Motivations for hepatitis B and liver cancer prevention in Bay Area Chinese Americans. Poster at Stanford Cancer Center Members' Retreat, Menlo Park, CA, April 7, 2010.

Wakelee HA, Chang ET, Shema SJ, Reynolds P, Clément-Duchêne C, Wiencke J, Gomez SL. Survival after non-small cell lung cancer in never-smoking Asian/Pacific Islander and Latina women. Journal of Thoracic Oncology 2009; 4(9 Suppl 1): s310 (Abstr#A7.6). Oral presentation at 13th World Conference on Lung Cancer, San Francisco, CA, July 31-August 4, 2009.

Chang ET, Kasperzyk JL, Birmann BM, Kraft P, Zheng T, Mueller NE. One-carbon metabolism nutrients and genes and Hodgkin lymphoma risk. Oral presentation at InterLymph Consortium 8th Annual Meeting, Vancouver, British Columbia, July 19-22, 2009.

Marshall SF, Chang ET, Clarke CA, Cress R, Deapen D, Horn-Ross PL, Largent J, Neuhausen S, Reynolds P, Templeman C, Bernstein L. Hormone therapy before diagnosis and breast cancer survival in the 10 California Teachers Study. Abstract at San Antonio Breast Cancer Symposium, San Antonio, TX,

December 10-14, 2008.

Telli ML, Kurian AW, Chang ET, Keegan THM, McClure LA, Ford JM, Gomez SL. Differences in breast cancer subtype distribution exist among ethnic subgroups of Asian women in California. Abstract at San Antonio Breast Cancer Symposium, San Antonio, TX, December 10-14, 2008.

Telli ML, Kurian AW, Chang ET, Keegan THM, Ford JM, Gomez SL. Asian race and breast cancer subtypes: a study from the California Cancer Registry. Poster at 44th American Society for Clinical Oncology (ASCO) Annual Meeting, Chicago, IL, May 30-June 3, 2008.

Invited speaker. Hepatitis B and liver cancer prevention in Asian/Pacific Islander Americans. Department of Epidemiology, Harvard School of Public Health, Boston, MA, April 1, 2008.

Chen JJ, Bergin M, Chang ET, So SK. A model HBV catch-up immunization and education project in Qinghai, China. Workshop presentation at 42nd National Immunization Conference, Atlanta, GA, March 17-20, 2008.

Bergin M, Rao A, Chang ET, So SK. Motivating youth to take action in public health: 5th Annual Youth Leadership Conference on Asian and Pacific Islander Health. Poster at 42nd National Immunization Conference, Atlanta, GA, March 17-20, 2008.

Invited speaker. Integration of population sciences with clinical research. Cancer Clinical Trials Forum, Stanford University School of Medicine, Stanford, California, July 18, 2007.

Chang ET, Nguyen BH, So SK. Determinants of hepatitis B awareness and prevention in Chinese Americans. Poster and presentation at Stanford Cancer Center retreat, Menlo Park, CA, March 12, 2007.

Chang ET, Lin SY, So SK. The Jade Ribbon Campaign: Hepatitis B virus screening and education in Asian/Pacific Islander Americans. Presentation at 2006 National Asian American Pacific Islander Health Summit, San Jose, CA, September 15, 2006.

Chang ET, Canchola AJ, Lee VS, Clarke CA, Reynolds P, Horn-Ross PL, and the California Teachers Study Investigators. Wine and other alcohol consumption and risk of ovarian cancer in the California Teachers Study cohort. Abstract and poster at 2nd North American Congress of Epidemiology, Seattle, WA, June 21-24, 2006.

Invited speaker. The role of the Epstein-Barr virus in Hodgkin lymphoma. Viruses and Cancer Symposium, Harvard School of Public Health, Boston, MA, May 5, 2006.

Invited speaker. The changing racial/ethnic burden of liver cancer in the Greater San Francisco Bay Area. Greater Bay Area Cancer Registry Certified Tumor Registrars Meeting, Fremont, CA, April 5, 2006.

Invited speaker. The role of the Epstein-Barr virus in Hodgkin lymphoma. Department of Epidemiology, University of Michigan School of Public Health, Ann Arbor, MI, January 12, 2005.

Invited speaker. Department of Epidemiology, University of Washington School of Public Health and Community Medicine, and Seattle Epidemiologic Research and Information Center, Seattle, WA, August 17, 2004.

Levin LI, Lennette ET, Ambinder RF, Chang ET, Rubertone M, Mueller NE. Prediagnosis Epstein-Barr virus serologic patterns in relation to the molecular status of Hodgkin's lymphoma in young adults. Abstract and poster at American Association for Cancer Research International Conference on Molecular and Genetic Epidemiology of Cancer, Waikoloa, HI, January 18-23, 2003.

Chang ET, Ambinder RF, Weir EG, Borowitz M, Mann RB, Zheng T, Mueller NE. Inverse association

between nursery school and Hodgkin's lymphoma, independent of EBV tumor status. Abstract and poster at 10th Biennial Meeting of the International Association for Research on Epstein-Barr Virus and Associated Diseases, Cairns, Australia, July 16-21, 2002.

Levin LI, Lennette ET, Ambinder RF, Chang ET, Rubertone M, Mueller NE. Prediagnosis Epstein-Barr virus serologic patterns in EBV-positive and EBV-negative Hodgkin's lymphoma. Abstract and poster at 10th Biennial Meeting of the International Association for Research on Epstein-Barr Virus and Associated Diseases, Cairns, Australia, July 16-21, 2002.

Chang ET, Ambinder RF, Zheng T, Mueller NE. Inverse association between childhood history of nursery school and Hodgkin's lymphoma in a population-based case-control study. Leukemia & Lymphoma 2001; 42(Suppl 2): 39-40. Abstract and poster at 5th International Symposium on Hodgkin's Lymphoma, Cologne, Germany, September 22-25, 2001.

Chang ET, Ambinder RF, Zheng T, Mueller NE. Inverse association between aspirin use and Hodgkin's lymphoma in a population-based case-control study. Leukemia & Lymphoma 2001; 42(Suppl 2): 41. Abstract and poster at 5th International Symposium on Hodgkin's Lymphoma, Cologne, Germany, September 22-25, 2001.

Chang ET, Birmann B, Ambinder RF, Zheng T, Mueller NE. Serum sCD23 levels in Hodgkin's disease patients are higher in EBV genome-positive than EBV genome-negative cases. Abstract, poster, and presentation at 9th Biennial Meeting of the International Association for Research on Epstein-Barr Virus and Associated Diseases, New Haven, Connecticut, June 22-27, 2000.

Advisory Appointments

Member, Faculty of 1000 Medicine, 2007-present

Member, California Teachers Study Steering Committee, 2007-2012

Scientific Member, Institutional Review Board, Cancer Prevention Institute of California, 2006-2012

Ad hoc member, ZRG1 PSE-P (02) M: NIH Special Emphasis Panel/Scientific Review Group for EPIC (Epidemiology of Cancer), March 2014

Ad hoc member, EPIC (Epidemiology of Cancer), NIH Population Sciences and Epidemiology Integrated Review Group, February and October 2010, June 2012

Ad hoc member, ZCA1 SRLB-3 (J1): NIH-Supported Centers for Population Health and Health Disparities Special Emphasis Panel, October 2009

Reviewer, Academia Sinica Investigator Award, 2009

Peer Reviewer

American Journal of Epidemiology

American Journal of Public Health

Annals of Epidemiology

Blood

Breast Cancer Research

Cancer

Cancer Causes & Control

Cancer Epidemiology

Cancer Epidemiology, Biomarkers & Prevention

Cancer Research

Clinical Cancer Research

Critical Reviews in Toxicology

Epidemiology

European Journal of Epidemiology

Hepatology

International Journal of Cancer

JAMA

Journal of Medical Virology

Journal of the National Cancer Institute

Leukemia Research

Molecular Carcinogenesis

Nutrition and Cancer

PLoS ONE

Risk Analysis

Social Science & Medicine

Appendix C

Testimony History of Ellen T. Chang, Sc.D.

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Ellen Chang, Sc.D.

Testimony history

Deposition testimony
Date: 11/25/2014
Case name: Battista et al. v. Enviro Tech International, Inc. et al. (Superior Court of New Jersey, Law Division – County of Essex, Docket No. L-681-10)
Law firms: King & Spalding LLP, McGivney & Kluger PC, Mound Cotton Wollan & Greengrass LLP

Deposition testimony
Date: 11/15/2015
Case name: Whitlock et al. v. PepsiAmericas et al. (United States District Court for the Northern District of California, Case No. C-08-2742 SI)
Law firm: Morgan, Lewis and Bockius, LLP

Deposition testimony
Date: 01/05/2016
Case name: Sanchez-Knutson v. Ford Motor Company (United States District Court for the Southern District of Florida, Case No. 0:14-cv-61344)
Law firm: Lankford Crawford Moreno & Ostertag, LLP

Deposition testimony
Date: 10/27/2017
Case name: Christopher Lightfoot v. Georgia-Pacific Wood Products LLC, et al. (United States District Court for the Eastern District of North Carolina, Southern Division, Case No. 7:16-cv-00244-FL)
Law firm: Forman Watkins & Krutz, LLP