Australian National University

PFAS HEALTH STUDY

PFAS Health Study

Component three: Cross-sectional survey of self-reported physical and mental health outcomes and associations with blood serum PFAS

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Contents

Plain Language Summary

Per- and polyfluoroalkyl substances (PFAS) are man-made chemicals that may be harmful to the environment and human health. The aim of the PFAS Health Study Cross-sectional Survey was to examine health conditions and concerns among people who had lived or worked in Australian communities with known PFAS contamination. This included Katherine in the Northern Territory, Oakey in Queensland, and Williamtown in New South Wales (the 'exposed communities').

We surveyed people in these communities who provided a blood sample for PFAS testing in the Australian Government-funded Voluntary Blood Testing Program. We also surveyed people in similar communities in Australia not known to have PFAS contamination. This included Alice Springs in the Northern Territory, Dalby in Queensland, and Kiama and Shellharbour in New South Wales (the 'comparison communities'). We did this through Services Australia, who sent invitations to a random sample of people on the Medicare Enrolment File in the comparison communities, on behalf of the PFAS Health Study team.

We asked people in exposed and comparison communities to complete an online or paper survey that asked about their demographic details; where they had lived and worked; whether they had ever been diagnosed with any of 32 health conditions; and the state of their mental health. We also asked people in exposed communities about their health concerns and use of healthcare related to the PFAS contamination.

We measured levels of PFAS in blood to see how health conditions varied with different levels of PFAS. We focused on three PFAS that were found in the blood of most participants in the exposed communities: perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA), and perfluorohexane sulfonic acid (PFHxS).

In total, 917 people from the exposed communities and 801 from the comparison communities completed the survey. We compared the percentage of people with different health conditions in the exposed and comparison communities.

People in Katherine were more likely to report cancer (especially breast cancer) and liver disease (especially fatty liver disease) than people in Alice Springs.

In Williamtown, people were more likely to report rheumatoid arthritis, hypercholesterolaemia (high cholesterol), type II diabetes, and problems with fertility compared to people in Kiama and Shellharbour.

In Oakey and Dalby, the numbers of people surveyed were too small to make reliable comparisons.

While we observed differences between exposed and comparison communities, the findings were not consistent across exposed and comparison community pairs. The reported health conditions could have occurred at any time, even before a person lived or worked in a community exposed to PFAS. In addition, health conditions were self-reported by survey participants and may not have been diagnosed by a health professional.

We found that people with higher PFAS levels were not more likely to report most diseases. However, the results varied across the different communities and PFAS. For example, for a doubling of the blood level of PFOS, people in Katherine were 29% less likely to report breast cancer, whereas in Williamtown people were 15% more likely to report breast cancer. For one chemical, PFOA, which was not elevated in people in exposed communities compared to people in comparison communities in the PFAS Health Study Blood Serum Study, we found that a doubling of blood levels was associated with more people with high cholesterol, gout, and hypothyroidism in different exposed communities.

People living in exposed communities reported much higher levels of mental distress and worry than people in comparison communities. People who worked with firefighting foams containing PFAS and people who used bore water on their properties reported higher levels of worry and concern than people who did not.

In the exposed communities, one in three people reported being ‘very’ or ‘extremely’ concerned about their health and one in five people had serious concerns about their mental health. People surveyed in these communities also reported concerns about their finances, the stigma of living in exposed communities, and uncertainty about the future.

The survey participants from the exposed communities were not randomly sampled, rather people chose to participate. The results may therefore represent the experiences of people who were more worried about PFAS or were more likely to believe an illness was related to PFAS because of their known exposure. The results may not represent the experience of all people living in the communities. In the comparison communities, we randomly sampled people, but a very small number of the invited people completed the survey (only 3%). In addition, some of the reported results could be due to chance.

Because this is a cross-sectional survey, we cannot draw conclusions about whether PFAS could have caused health conditions. While survey participants reported higher percentages of some health conditions in individual communities, these findings were not consistent across communities, and were not clearly related to levels of PFAS in blood. In contrast, there was consistency when looking at mental health. We observed higher levels of distress and worry in people from exposed communities, particularly among those who may have been exposed to PFAS at work, than in people from comparison communities.

Technical Summary

Background

Per- and polyfluoroalkyl substances (PFAS) are synthetic chemicals classified as contaminants of emerging concern due to their potential adverse effects on the environment and human health. From 2013 to 2017, the Australian Government identified PFAS contamination in the local environments of Katherine in the Northern Territory, Oakey in Queensland, and Williamtown in New South Wales — known as the PFAS Management Areas. The primary aim of the PFAS Health Study Cross-sectional Survey was to identify health conditions and concerns reported by people residing or working in the PFAS Management Areas. We compared the prevalence of physical and mental health conditions in the exposed communities with the prevalence in similar communities not known to have PFAS contamination (the 'comparison communities'), and quantified relationships between PFAS concentrations in blood serum and the reported health conditions.

Methods

In 2019 to 2020, we conducted a cross-sectional survey of the exposure history, physical health, mental health, and sociodemographic characteristics of individuals in exposed and comparison communities. To survey individuals who resided or worked in the PFAS Management Areas, we invited individuals who had their blood tested for PFAS in the Australian Government Voluntary Blood Testing Program (VBTP) and consented to the PFAS Health Study Blood Serum Study to complete a paper or online survey.

We chose three comparison communities that were of sufficient population size and similar to the PFAS Management Areas based on area-level sociodemographic characteristics, remoteness, and proportion of residents who identified as Aboriginal and/or Torres Strait Islander. The comparison communities were: Alice Springs in the Northern Territory, Dalby in Queensland, and Kiama and Shellharbour in New South Wales. Services Australia randomly sampled 30,000 people (16 years of age and older) who resided in the comparison communities from the Australian Government Medicare Enrolment File and sent invitations to participate in the Cross-sectional Survey on behalf of the PFAS Health Study team. Participants completed a paper or online survey. At the time of recruitment, we also invited comparison participants to provide a blood sample for the Blood Serum Study.

In the survey, participants reported whether they had ever been diagnosed with any of 32 health conditions, whether they had ever experienced problems with their fertility, and the age that female participants started menopause. Participants reported mental health symptoms through four validated measures of psychological distress (Patient Health Questionnaire-15, Kessler-6, Distress Questionnaire-5, and Generalised Anxiety Disorder-7). Participants were also asked to rate their concerns related to living or working in the PFAS Management Areas and changes in health-related behaviour.

We compared the prevalence of self-reported health outcomes between exposed and comparison communities, including those relating to psychological distress. We also quantified the association between self-reported health outcomes and blood serum concentrations of three PFAS (perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA), and perfluorohexane sulfonic acid (PFHxS)) that were measured in the Blood Serum Study. We estimated prevalence ratios (PR), taking into account sociodemographic and health-related confounders. In exposed communities, we assessed participant concerns regarding their physical and mental health, along with participant perceptions of the usefulness of the blood tests for PFAS in the VBTP.

Results

In total, 917 individuals from the exposed communities completed the survey, along with 801 individuals from comparison communities.

The prevalence of self-reported breast cancer was higher in Katherine than in Alice Springs (adjusted PR = 3.09, 95% confidence interval (CI) 1.17 to 8.17), as was the prevalence of any of 10 cancers combined (adjusted PR = 3.78, 95% CI 1.66 to 8.60), fatty liver disease (adjusted PR = 2.42, 95% CI 0.97 to 6.02), and any of three liver conditions combined (adjusted PR = 2.39, 95% CI 1.06 to 5.40). The prevalence of self-reported rheumatoid arthritis was higher in Williamtown than in Kiama and Shellharbour (PR = 2.25, 95% CI 1.04 to 4.90), as was hypercholesterolaemia (high cholesterol) (adjusted PR = 1.25, 95% CI 0.98 to 1.59), type II diabetes (adjusted PR = 1.77, 95% CI 1.01 to 3.09), and problems with fertility (adjusted PR = 2.15, 95% CI 1.30 to 3.54). In Oakey compared to Dalby, prevalence ratios were imprecisely estimated and uninformative.

PFAS concentrations in blood serum were not clearly associated with most self-reported health conditions. We observed higher prevalence of gout, hypercholesterolaemia, and hypothyroidism per doubling in blood serum concentrations of PFOA (e.g., in Williamtown: gout and PFOA adjusted PR = 1.54, 95% CI 1.06 to 2.24), which is a relatively minor component of the firefighting foams used in Australia, compared to PFOS and PFHxS. In contrast, small inverse associations were observed for cancer, gout, and hypothyroidism per doubling in blood serum concentrations of PFOS and/or PFHxS in different exposed communities (e.g., in Williamtown: gout and PFOS adjusted PR = 0.74, 95% CI 0.62 to 0.90), which were the predominant PFAS chemicals identified in the blood serum of participants in exposed communities in the Blood Serum Study.

We observed substantially higher levels of self-reported psychological distress in exposed communities than in comparison communities (e.g., in Katherine compared to Alice Springs: clinically-significant self-reported anxiety scores, adjusted PR = 2.82, 95% CI 1.16 to 6.89). We found limited evidence to suggest that psychological distress was associated with PFAS serum concentrations in the exposed communities. However, psychological distress was higher among participants who were occupationally exposed to firefighting foam, among participants who used bore water on their properties, and among participants who were concerned about their health.

Of the participants in the PFAS Management Areas who responded to the survey questions on health concerns, 32% (270/835) reported being 'very' or 'extremely' concerned about their health and 19% (156/829) reported being 'very' or 'extremely' concerned about their mental health. Sixty-eight percent (562/832) of participants reported feeling uncertainty about the future, 48% (399/825) reported concern about their finances, and 42% (347/829) reported concern about stigma.

Our findings should be interpreted with awareness of the study weaknesses. Selection bias was a particular issue, as the survey respondents in the exposed communities were self-selected and were not representative of the population in each of the communities. We used self-reported measures of health that were not validated using medical records and exposure measurement may have occurred after disease onset. As this was a cross-sectional study, our findings cannot be used to determine cause and reverse causation is a possibility. In addition, some of the findings may be due to chance.

Conclusions

Overall, we found that the prevalence of several self-reported health conditions was higher in participants from the PFAS Management Areas than in participants from the comparison communities. When we examined the prevalence of these health outcomes per doubling in PFAS concentrations in blood serum, there was limited evidence of associations and low consistency across the three PFAS Management Areas. This study demonstrated the high levels of psychological distress among participants from the PFAS Management Areas.

Abbreviations

AFFF	Aqueous film-forming foam
BMI	Body Mass Index
DQ5	Distress Questionnaire-5
eGFR	Estimated Glomerular Filtration Rate
GAD-7	Generalised Anxiety Disorder-7 scale
GP	General Practitioner
K6	Kessler-6 scale
NHANES	National Health and Nutrition Examination Survey (USA)
NSW	New South Wales
NT	Northern Territory
PFAS	Per- and polyfluoroalkyl substances
PFBS	Perfluorobutane sulfonic acid
PFDA	Perfluorodecanoic acid
PFHpA	Perfluoroheptanoic acid
PFHxA	Perfluorohexanoic acid
PFHxS	Perfluorohexane sulfonic acid
PFNA	Perfluorononanoic acid
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulfonic acid
PHQ-15	Patient Health Questionnaire-15
PR	Prevalence ratio
Qld	Queensland
RAAF	Royal Australian Air Force
US	United States (of America)
USA	United States of America
VBTP	Voluntary Blood Testing Program
6:2 FTS	6:2 fluorotelomer sulfonic acid

Introduction

Per- and polyfluoroalkyl substances (PFAS) are synthetic chemicals classified as contaminants of emerging concern due to their potential to adversely affect the environment and human health. Concerns over the widespread use and global distribution of PFAS have led to considerable scientific investigation and public interest regarding the effects of exposure to these chemicals on human health.[1] PFAS have been extensively used in industrial and consumer products since the 1950s and are universally detected in blood serum samples due to their persistent and bioaccumulative properties.[1-4] However, communities in areas with contaminated water sources and land have been shown to have higher exposure to PFAS, which has the potential for both immediate impacts on psychological health and latent effects on physical health.[5] Understanding exposure to PFAS and the associated health effects is vital to informing public health responses and addressing community concerns in areas affected by environmental contamination.[6]

PFAS overview

PFAS are a group of more than 4,000 fluorinated, organic chemicals that contain at least one carbon atom that has all of its hydrogen substituents replaced by fluorine atoms.[7] Structurally, most PFAS consist of a carbon chain (alkyl chain) and a functional end group, such as an acid group.[7] PFAS vary in their properties depending on the structure and length of the carbon chain.[7] Perfluoroalkyl substances have a carbon chain that contains only fluorinated carbons.[7] Due to the strength of the carbon fluorine bonds, perfluoroalkyl substances remain stable under a variety of biological, chemical and thermal conditions.[7] In contrast, polyfluoroalkyl substances contain a carbon chain that has at least one fluorinated and one non-fluorinated carbon atom. Under specific conditions, some polyfluoroalkyl substances can break down into stable perfluoroalkyl substances.[7,8]

Many PFAS contain a functional end group that attracts water (hydrophilic), opposing the properties of the fluorinated carbon chain, which repels water (hydrophobic) and oil (oleophobic). As a result, PFAS have unique surface-active properties which make them effective in reducing surface tension and resistant to heat, oil, stains, grease and water.[7,9] Due to their stability and properties, PFAS have a wide range of uses.[9] Initially, PFAS were manufactured for use in consumer products, such as fabric protectant and non-stick cookware. Later applications of PFAS include a range of industrial products, including aqueous film-forming foams (AFFF) that were used to extinguish liquid fuel fires in aviation settings. The extensive use of PFAS for household and industrial purposes since the 1950s, and the subsequent movement of PFAS through water sources and land, led to environmental contamination across the world.[10-12]

Perfluorooctane sulfonic acid (PFOS), perfluorohexane sulfonic acid (PFHxS) and perfluorooctanoic acid (PFOA) are the most widely studied PFAS and have been found to bioaccumulate in wildlife and humans.[7] The presence of these long-chain perfluoroalkyl substances (defined as ≥6–8 perfluoroalkyl carbons) in the environment is driven by environmental release from industrial and consumer products and the subsequent breakdown of larger polyfluoroalkyl substances.[7,13] The common use and widespread distribution of PFAS has led to increasing concerns about potential effects on the environment and human health.[1] In response, manufacturers have phased out of production many long-chain perfluoroalkyl substances over the past two decades.[1,7]

In Australia, studies of pooled blood serum samples of the general population have shown declines in PFOS, PFHxS, and PFOA concentrations over time following the phase-out of their production and use.[2] However, exposure to PFAS through environmental contamination in specific populations remains a public health concern. Worldwide, studies show significantly higher exposure to PFAS in populations living in areas affected by environmental contamination, compared to the general

population.[14-17] In response, governments and international agencies have prioritised the remediation of environments contaminated with PFAS to reduce potential exposure. However, the stability of PFAS under varying environmental conditions and their sources are a current challenge to remediation efforts, requiring the development of innovative methods.[18-20]

Human health effects

Concerns over the potential for PFAS to adversely affect human health arise from the ease with which they are absorbed into and distributed through the body.[14,21] Human exposure to PFAS occurs predominantly through ingestion and absorption into the blood stream via the digestive tract, but may also occur through inhalation or absorption through the skin (dermal). Following exposure, several PFAS have been found to bind to serum albumin (a protein present in blood serum) resulting in accumulation in tissues with large blood supply, such as the kidneys and liver.[21,22] The elimination half-life[1] in human blood varies with the type of PFAS, ranging from 3–5 years for PFOS to 5–8 years for PFHxS and 2–3 years for PFOA.[23-25] However, ongoing exposure to PFAS through industrial and consumer products may affect the estimates of the half-lives[2] of these chemicals.

A rise in scientific and public interest in the potential health effects of PFAS exposure has led to substantial epidemiological and toxicological investigations, which indicate a range of potential effects on metabolism, immunity, reproduction, and development. Systematic reviews of the epidemiological literature suggest that these effects include higher serum lipid levels (e.g., high cholesterol, known as hypercholesterolaemia), abnormal thyroid hormone levels, and the suppression of some immune responses.[14,26-32] Reviews suggest associations of PFAS with adverse changes to liver function (through disruptions to bile acid uptake and lipid accumulation) and reductions in kidney function, as measured by the estimated Glomerular Filtration Rate (eGFR).[27,33] There is additional evidence of an association between higher serum PFOA concentrations and hyperuricaemia, through a disruption to uric acid metabolism.[14] In addition to changes in metabolism, scoping reviews suggest an association between PFOA exposure and increased risk of testicular and kidney cancers.[33,34] Potential adverse effects on reproduction include decreased fertility through changes to testosterone levels in males and disruption to ovarian function in females.[35-37]

Key epidemiological studies

Epidemiological studies of communities affected by environmental contamination may provide insight into the potential health impacts of PFAS exposure. The results of individual studies, however, are not considered conclusive and must be weighed against studies of similar outcomes through systematic review. The C8 Health Project was conducted from 2005 to 2013 in approximately 69,000 residents of areas in Ohio and West Virginia who consumed drinking water contaminated with PFOA.[16] The C8 Health Project found evidence for increased risks of the following health outcomes associated with blood serum concentrations of PFOA: hypercholesterolaemia; pregnancy-induced hypertension; thyroid disease; testicular and kidney cancer; and ulcerative colitis.[38] Similarly, an epidemiological study of PFOA exposure in residents of the Veneto region of Northern Italy reported a range of adverse health effects: increased all-cause and cause-specific mortality rates, including COVID-19 mortality rates; changes in

[1] The elimination half-life of a substance in the body is a measurement of the length of time required for the body to eliminate half of the substance by normal physiological processes.

[2] In many studies, the elimination half-life is estimated by monitoring the rate of elimination from the body, without considering potential ongoing exposure (or other physiological changes). In such studies, the observed half-life is often referred to as an 'apparent half-life' (where the elimination rate is a result of ongoing exposure, adsorption and distribution in the body, as well as elimination). If there is an ongoing exposure, the apparent half-life is likely to be longer compared to the 'intrinsic' (true) elimination half-life (estimated from the elimination alone).

cholesterol levels of women during pregnancy, including an increase in total cholesterol in the first trimester; delayed or irregular menstruation in young women; and decreased biochemical markers of fertility in young men.[17,39-42]

Individual epidemiological studies across Sweden show evidence of increased serum PFOS and PFHxS concentrations in communities affected by environmental contamination from AFFF. The Prospective Investigation of the Vasculature in Uppsala Seniors (PIVUS) Study, conducted from 2001 to 2014, investigated the health effects of PFAS exposure in elderly residents of Uppsala, Sweden. Residents were exposed to drinking water contaminated with AFFF from a nearby military airport.[43] A study of the affected residents found a positive association between serum concentrations of several PFAS, including PFOS, and carotid atherosclerosis.[43] Another cohort study of pregnant women and their offspring conducted from 1996 to 2017 in Uppsala reported potential adverse effects on fetal and childhood development. Higher serum PFOA, PFOS, and PFHxS concentrations in mothers were associated with higher Body Mass Index (BMI) in their children at 3–5 years.[44] A study of approximately 63,000 people who lived in Ronneby, Sweden from 1980 to 2013 reported an association between higher serum PFAS concentrations and higher total cholesterol and low-density lipoprotein concentrations.[15] Further studies reported an association between higher serum PFAS concentrations and changes to gene expression related to the development of cardiovascular disease, dementia and cancers.[45,46] Individual epidemiological studies of environmental contamination from historic AFFF use in the United States of America (USA) also reported higher serum PFOS and PFHxS concentrations in residents and workers of the affected communities, compared to the general population. However, investigations of the health risks in these populations are ongoing, with the results not yet published.[47-49]

PFAS contamination in Australia

In Australia, PFAS contamination has occurred in environments surrounding firefighting training grounds, airports and military bases where AFFF were in frequent use. From the 1970s, AFFF were used at Australian Defence Force bases for fire emergencies and training purposes.[50-53] Predominantly, Australian Defence Force bases used the product 3M Light Water™, which contains PFOS and PFHxS as the main active ingredients.[50,51,53,54] In 2002, the 3M Company ceased the production of Light Water™ due to environmental and human health concerns. The Department of Defence discontinued use of Light Water™ across Australian military bases over the following years, replacing the product with Ansulite™ — a fluorotelomer-based foam.[55,56]

PFAS Management Areas

From 2013 to 2017, the Australian Government identified PFAS contamination affecting the environment surrounding the Royal Australian Air Force (RAAF) Bases at Tindal in Katherine, Northern Territory (NT) and Williamtown in New South Wales (NSW), and the Army Aviation Centre in Oakey in Queensland (Qld).[57-59] Environmental investigations of PFAS in groundwater, surface water, sediment and soil showed the extent of contamination on the military bases and off-base areas, including surrounding residential properties.[60-62] The affected environments, referred to as PFAS Management Areas, contain varying concentrations of PFAS depending on the historic use of AFFF and other factors, including the direction of groundwater flow through aquifers and the spread of surface water through drains, waterways and flooding events.[60-62] However, PFAS concentrations were highest in water sources and land located in close proximity to the military bases, represented by Primary Zones within the PFAS Management Areas.[50-52]

The main PFAS exposure pathways in Katherine, Oakey, and Williamtown are the consumption of local bore water (extracted from groundwater) — including incidental consumption via bathing and swimming — and the consumption of local produce watered with bore water or grown in contaminated soil, which may have also been affected by surface water.[50-52] Consumption of fish

or crustaceans sourced from local rivers and waterways is an additional exposure pathway for the affected communities.[50-52] The Australian Government and state and territory governments provided advice to residents of these PFAS Management Areas to minimise potential sources of exposure to PFAS. These precautions were informed by risk assessments incorporating the environmental site investigations of groundwater, surface water and local produce, including livestock, poultry, seafood, and fresh fruit and vegetables.[50-52] Contamination of the local environment in Williamtown, Oakey, and Katherine led to substantial community concern and public interest in the potential human health effects.

PFAS Health Study

In response to the contamination events, the Australian Department of Health commissioned the Australian National University (ANU) to conduct an epidemiological study to investigate exposure to PFAS and the related health effects in Katherine, Oakey, and Williamtown. To coincide with the epidemiological study, the Department of Health introduced the Voluntary Blood Testing Program (VBTP) for PFAS for people who had ever lived or worked in these PFAS Management Areas. The PFAS Health Study was conducted in two phases.

In Phase I, the PFAS Health Study team conducted a systematic review to examine the health effects of PFAS in humans as reported in literature published until February 2017.[63] The review reported sufficient evidence for an association of higher blood serum concentrations of PFOA and PFOS with increased serum total cholesterol concentrations. The review identified limited evidence for a positive association of serum PFOA and PFOS with serum uric acid concentrations, an inverse association between serum PFOA and PFOS and eGFR, and a positive association of serum PFOA and PFOS with prevalence of chronic kidney disease. Together, these findings suggest a potential association between high serum PFAS concentrations and impairment of kidney function in humans. The review further reported limited evidence for a positive association between exposure to PFOA and kidney and testicular cancer, and an inverse association between exposure to a range of PFAS (including PFOS and PFOA) and antibody levels of diphtheria and rubella after vaccination.

Phase II included an epidemiological study of the three PFAS-affected communities noted above, comprising four studies which are detailed below.

Focus Groups Study

The PFAS Health Study team conducted the Focus Groups Study to understand the views, experiences and concerns regarding PFAS among individuals who lived or worked in the towns of Katherine, Oakey, and Williamtown. Three focus groups of 29 people in Katherine, 36 in Oakey and 46 in Williamtown occurred between January and August 2018. Additional focus groups were held in three local Aboriginal communities in Katherine, with 69 participants in August 2018. The findings of the focus group discussions were published in a report released in February 2019 and subsequently, in a peer-reviewed journal article.[6,64]

Blood Serum Study

The Blood Serum Study compared blood serum PFAS concentrations in residents and workers from the three PFAS Management Areas (the exposed communities) and residents of three communities not affected by environmental PFAS contamination (the comparison communities): Alice Springs in the NT, Dalby in Qld, and Kiama and Shellharbour in NSW.[65] Participants from the exposed communities were a sub-sample of people who undertook blood testing through the VBTP between 2016 and 2019. Participants from the comparison communities were randomly selected to participate in the PFAS Health Study from the Medicare Enrolment File in 2020. A pathology laboratory tested blood serum samples of participants from the exposed and

comparison communities for a range of PFAS, as well as several biochemical markers of health, including serum lipids and markers of kidney, liver and thyroid function.

Cross-sectional Survey

The Cross-sectional Survey (detailed in this report) investigated the health of residents and workers from the exposed communities and residents of the comparison communities. Participants completed a survey about whether or not they had ever experienced any of a range of health outcomes. The survey also assessed psychological well-being and distress and collected data on sociodemographic characteristics. Participants from the exposed communities completed the survey in 2019, following the end of the VBTP. Participants from the comparison communities were invited to participate in the Cross-sectional Survey in 2020, at the same time as the Blood Serum Study in 2020.

Data Linkage Study

The Data Linkage Study examined whether rates of adverse health outcomes were higher among people who had lived in the PFAS Management Areas than among people who had lived in similar areas in Australia not affected by environmental contamination.[66] Using linked administrative data collected over time, the study investigated maternal and infant (perinatal) health, childhood development, cancer, and cause-specific mortality outcomes.

Cross-sectional Survey report

In this report, we detail the methods, results and conclusions of the PFAS Health Study Cross-sectional Survey.

Aims and objectives

The aims of the Cross-sectional Survey were to identify health conditions and concerns of people residing or working in the PFAS Management Areas of Katherine, Oakey, and Williamtown. Specifically, we sought to compare the prevalence of physical and mental health conditions between the exposed and comparison communities, and to quantify potential associations between PFAS concentrations in blood serum and health outcomes. A further aim was to report participant perceptions of the VBTP.

Research questions

The research questions specified in our protocol were:

1. What are the main self-reported health outcomes among people living or working in the PFAS Management Areas in Katherine, Oakey, and Williamtown?
2. What are the current levels of psychological distress and how do these relate to PFAS blood serum concentrations and location of residence or work?
3. What are the main concerns regarding health associated with living or working in the PFAS Management Areas in Katherine, Oakey, and Williamtown?

The additional questions that we answered were:

4. Is the prevalence of self-reported health conditions and psychological distress in the exposed communities higher than the prevalence in the comparison communities, after taking into account sociodemographic differences?
5. Are self-reported health outcomes and psychological distress levels associated with PFAS serum concentrations?
6. What are the experiences and perceptions of the VBTP among people living or working in the PFAS Management Areas in Katherine, Oakey, and Williamtown?

Report structure and content

In this report, we detail the methods, results and conclusions of the third component of the PFAS Health Study — the Cross-sectional Survey. The study draws on data collected in the Blood Serum Study, including the measurements of PFAS exposure and biochemical markers in blood serum. The Blood Serum Study and the Cross-sectional Survey were undertaken contemporaneously, with participants in the exposed and comparison communities invited to complete the survey at the time of, or after, blood sample collection for the Blood Serum Study. The results of the Cross-sectional Survey are presented in four sections: analyses of self-reported health; analyses of self-reported psychological distress; health concerns among participants in the exposed communities; and participant perceptions and experiences of the PFAS blood testing through the VBTP. Throughout this report, we cross-reference the methods, results and conclusions of the Blood Serum Study report,[65] which presents findings on blood serum PFAS concentrations in exposed and comparison communities, risk factors for elevated serum PFAS concentrations; and associations between PFAS and biochemical markers of health.

Methods

Study design and recruitment

To investigate PFAS exposure and the potential health effects associated with PFAS exposure in Australian communities affected by environmental contamination, we conducted a cross-sectional survey of exposure history, physical health, mental health, and sociodemographic characteristics of exposed and comparison populations.

Exposed population

The exposed population was recruited from participants of the Blood Serum Study who had agreed to be contacted by the PFAS Health Study team for future research. Invitations were sent directly from the PFAS Health Study team. Participants in the Blood Serum Study were recruited through the VBTP; details on the recruitment can be found in the Blood Serum Study report.[65] In addition, current and previous community members who had not taken part in the Blood Serum Study were able to contact the PFAS Health Study team to enrol as a participant in the Survey.

Through the consent procedures for the Cross-sectional Survey, we invited participants to have their blood sample tested for biochemical markers of kidney, liver, and thyroid function and for lipids (e.g., cholesterol). In December 2020, we invited participants from the Blood Serum Study who had not completed the survey to complete a shorter version of the survey (the 'mini-survey') and to consent to have their blood sample tested for biomarkers of health. The mini-survey was hosted online only; however, participants were able to contact the study team by phone to provide their consent to participate.

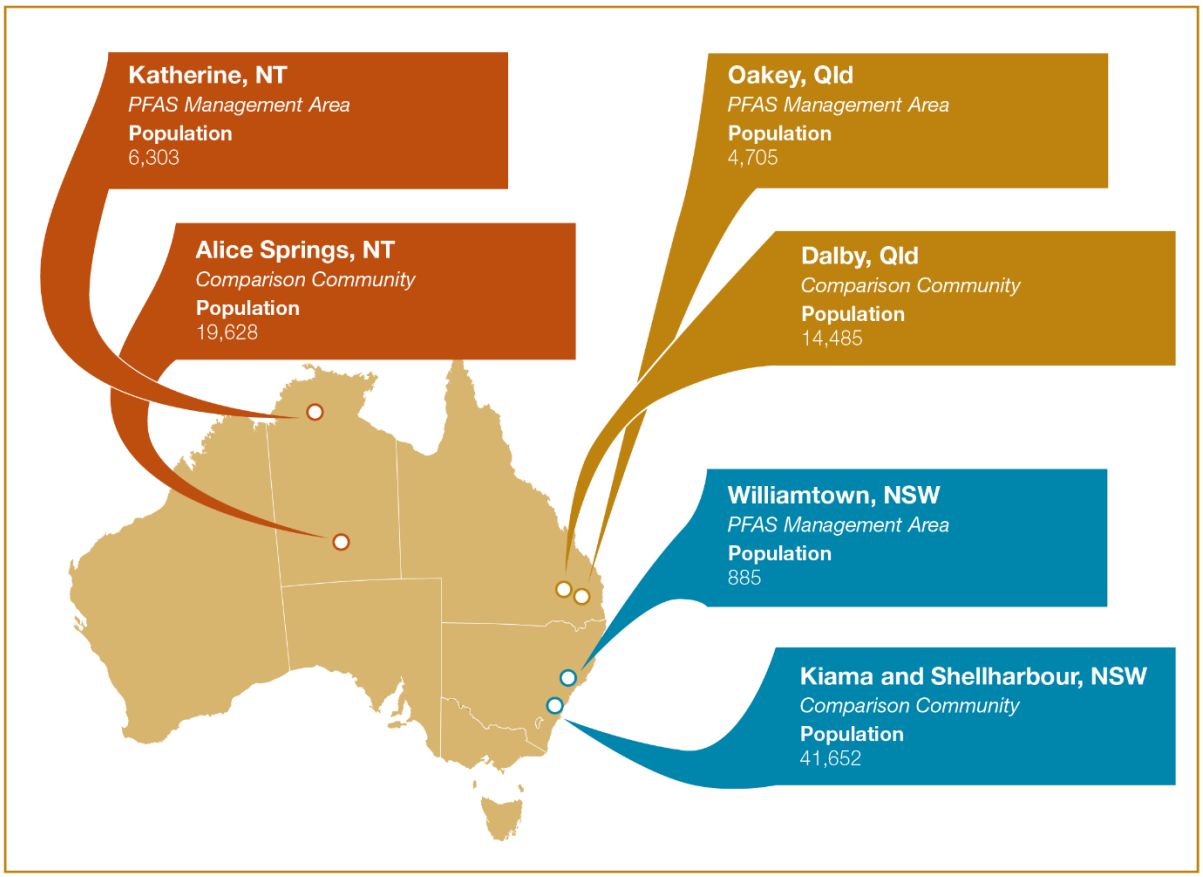
Comparison population

The target comparison population for the study was current residents of communities not affected by environmental PFAS contamination, according to the water quality guideline values for PFAS developed by the Australian Government Department of Health, Food Standards Australia New Zealand, and the National Health and Medical Research Council.[67] We chose three comparison communities that were comparable to the PFAS Management Areas and within the same State or Territory: Alice Springs in NT, Dalby in Qld, and Kiama and Shellharbour in NSW. We identified the comparison communities based on area-level (postcode) attributes including sociodemographic characteristics (Socio-Economic Indexes for Areas) and remoteness (Accessibility and Remoteness Index of Australia), according to the Australian Bureau of Statistics 2016 Census data. Further, we considered the proportion of Aboriginal and Torres Strait Islander persons in the community. Based on an expected participation rate of 2%, we required a minimum population of 10,000 residents in each comparison community. The final selection of the comparison communities was based on access to pathology services for blood collection, to align with the data collection methods used for the exposed population.

On behalf of the ANU study team, Services Australia (the Australian Government agency responsible for Medicare) randomly sampled individuals from the comparison populations from the Australian Government Medicare Enrolment File, based on residents of the postcodes 0870 (Alice Springs), 4405 (Dalby), and 2529 and 2533 (Kiama and Shellharbour). We recruited comparison participants for the survey from 10 August to 5 October, 2020. Services Australia sent 10,000 randomly selected adult residents (≥16 years old) from each comparison community an invitation to participate in the PFAS Health Study. Residents were contacted using a tiered approach over eight weeks, which included a reminder letter sent by Services Australia two weeks after the initial invitation. We provided information on the purpose of the study and instructed residents to register for the study online or via telephone. At the time of recruitment for the study, we also invited participants to provide a blood sample for the Blood Serum Study.[65]

A map of the exposed and comparison communities is shown in Figure 1.

Figure 1. Map of the PFAS Management Areas in Katherine, Oakey, and Williamtown and the corresponding comparison communities for the PFAS Health Study Cross-sectional Survey and Blood Serum Study.



Population data sourced from Australian Census QuickStats, 2016.[68]

Data collection

Cross-sectional Survey

The ANU Social Research Centre was contracted to provide advice on the design and formatting of the questionnaire, to host the online survey on a secure platform, to receive and scan completed paper surveys, and to provide datasets at the end of the collection period.

We recruited participants from the exposed communities from 5 August to 28 October, 2019. For children under the age of 16 years, a parent or guardian completed the survey on behalf of the child. The survey was pilot tested on 100 randomly-selected adult and child Blood Serum Study participants from the exposed communities. The survey instrument was refined before invitations were issued to the remainder of the participants. Participants from the exposed communities were sent an information pack containing a participant information sheet, consent form for the participant to keep and individual login details for the survey. The paper survey, participant information sheets, and consent forms are available on the PFAS Health Study website[3]. We sent a reminder to study participants two weeks after the invitation and included a paper copy of the survey and reply-paid envelope to return the completed survey. Participants who were invited to complete the mini-survey were sent an information pack containing a participant information sheet, consent form for the participant to keep, and individual login details. No reminders were sent for the mini-survey, and completions were accepted until 15 January 2021.

[3] https://rsph.anu.edu.au/research/projects/pfas-health-study

During recruitment in the comparison communities, we invited participants to complete an online or paper survey, and sent an information pack containing a participant information sheet, consent form for the participant to keep, and either individual login details for the survey or a paper copy and reply-paid envelope to return the survey. We sent participants two reminders to participate in the study. First letters were sent from 10 August 2020 and we accepted completed surveys until 15 January 2021.

Measurement of PFAS and biochemical markers in blood serum

Measurement of PFAS and biochemical markers in blood serum is described in detail in the Blood Serum Study report.[65]

Data analysis

Outcomes

The study included four groups of outcomes: 1) self-reported health outcomes; 2) psychological distress outcomes; 3) health concerns and health-related behaviours; and 4) experiences and perceptions of the PFAS blood testing.

Self-reported health outcomes

Participants were asked whether they had ever been diagnosed with any of 32 health conditions, including 10 cancer outcomes, four cardiovascular outcomes, three liver outcomes, two kidney outcomes, six autoimmune outcomes, four endocrine outcomes, and three neurological outcomes (see Box 1). Participants were also asked whether they had ever experienced problems with their fertility and, for female participants, the age that they started menopause, to give a total of 34 self-reported health outcomes. We defined early-onset menopause as commencing at 45 years of age or earlier, but not induced by a medical treatment or procedure.

We analysed health conditions separately and as combined outcomes, to allow analysis of rare health outcomes. Combined outcomes included 'any cancer', 'any cardiovascular disease' (excluding hypercholesterolaemia), 'any kidney disease', 'any liver disease', and 'any autoimmune disease' (Box 1). The endocrine and reproductive outcomes were only analysed separately. We excluded from analysis separate and combined health outcomes that had fewer than 10 cases in all exposed communities. There were insufficient cases to analyse type I diabetes, motor neurone disease, Parkinson's disease, dementia, or 'any neurological outcome' combined. Self-reported health outcomes were analysed as binary variables (e.g., ever diagnosed or never diagnosed with a health condition).

Psychological distress outcomes

We assessed psychological distress using four validated screening scales that measure general psychological distress, somatisation (i.e., psychological distress that manifests as physical symptoms), and anxiety levels. Somatisation was assessed using the Patient Health Questionnaire-15 (PHQ-15),[69] which asked participants about the severity of 15 symptoms that they may have experienced in the four weeks before survey completion. Symptom severity was assessed on a 3-point scale (0 = 'not bothered at all', 1 = 'bothered a little', 2 = 'bothered a lot'). We excluded one item that applied only to females ('menstrual cramps or other problems with your periods'), and summed the responses for the remaining 14 items to generate a score ranging from 0 to 28.

General psychological distress was measured using the Kessler-6 scale (K6)[70] and Distress Questionnaire-5 (DQ5),[71] which assessed the level of distress participants had experienced in the 30 days before survey completion. Responses to the six questions in the K6 were given on a five-point scale (1 = 'never', 2 = 'rarely', 3 = 'sometimes', 4 = 'often', 5 = 'always') and were summed to generate a score ranging from 6 to 30. Similarly, responses to the five questions in the DQ5 were

given on a five-point scale (1 = 'none of the time', 2 = 'a little of the time', 3 = 'some of the time', 4 = 'most of the time', 5 = 'all of the time') and were summed to generate a score ranging from 5 to 25.

Box 1. Self-reported health outcomes.

Cancer outcomes
- Bone cancer
- Brain cancer
- Breast cancer
- Kidney cancer
- Leukaemia
- Liver cancer
- Ovarian cancer
- Prostate cancer
- Testicular cancer
- Thyroid cancer

Liver outcomes
- Hepatitis (non-infectious)
- Fatty liver disease
- Cirrhosis of the liver

Kidney outcomes
- Chronic kidney disease
- Gout

Reproductive outcomes
- *Problems with fertility*
- *Early onset menopause*

Cardiovascular outcomes
- Heart attack
- High blood pressure
- Stroke
- *Hypercholesterolaemia*

Autoimmune outcomes
- Lupus
- Ulcerative colitis
- Crohn's disease
- Multiple sclerosis
- Rheumatoid arthritis
- Asthma

Endocrine outcomes
- *Type I diabetes*
- *Type II diabetes*
- *Hypothyroidism*
- *Hyperthyroidism*

Neurological outcomes
- *Dementia*
- *Parkinson's disease*
- *Motor neurone disease*

Outcomes in *italics* were not analysed as combined outcomes. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Self-reported symptoms of anxiety experienced by participants in the two weeks before survey completion were measured using the Generalised Anxiety Disorder (GAD-7) scale,[72] which consisted of seven items and a four-point response scale (0 = 'not at all', 1 = 'several days', 2 = 'more than half the days', 3 = 'nearly every day'). The items were summed to give a score ranging from 0 to 21.

We analysed psychological outcomes both as continuous variables and as binary variables. We defined the binary psychological outcomes based on validated cut-off scores that aim to identify individuals who meet clinical criteria for psychological distress and anxiety. Specifically, participants with a score of 10 or more on the PHQ-15, a score of 13 or more on the K6, a score of 14 or more in the DQ5, or a score of 10 or more on the GAD-7 scale were considered to demonstrate self-reported symptoms that may be of clinical significance (i.e., symptoms that are likely to interfere with everyday functioning),[69,71-73] referred to as 'clinically-significant' scores throughout the report.

Health concerns and health-related behaviours in exposed communities

Participants were asked to rate their concerns about issues related to residing or working in a PFAS Management Area on a five-point scale ('unconcerned', 'slightly concerned', 'moderately concerned', 'very concerned', 'extremely concerned'). Concerns included physical health, mental health, stigma, uncertainty about the future, finances, and work disruption. Participants were also separately asked whether they were concerned about their own health, their partner's health, and their children's health.

Participants were asked about changes in their health-related behaviours in relation to residing or working in a PFAS Management Area, with responses of 'yes', 'yes, but not attributed to PFAS', or 'no'. Behaviours included the commencement of, or increase in, smoking and alcohol consumption, use of prescription medication for sleep, and reduced physical activity.

Participants were asked whether they sought assistance from a health professional in relation to residing or working in a PFAS Management Area to manage physical or mental health. Participants were further asked whether they sought assistance from specific health professionals, including general practitioners (GPs), specialists, psychologists, psychiatrists, counsellors, and telephone counselling services. Participants who reported seeking assistance were asked whether the assistance they received had been helpful.

PFAS blood test experiences and perceptions in exposed communities

Participants in exposed communities were asked whether they found the PFAS blood testing through the VBTP helpful and were asked to state the reasons for their response in free text.

Exposures

We considered the following PFAS exposure variables in our analyses of self-reported health and psychological distress:

1. Community membership, defined as residence or work in the PFAS Management Areas in Katherine, Oakey, and Williamtown, versus the corresponding comparison communities in Alice Springs, Dalby, and Kiama and Shellharbour, and
2. PFAS concentrations measured in blood serum in the Blood Serum Study.

We used blood serum concentrations of PFOS, PFOA, and PFHxS in our analyses, which were detected in blood serum in >80% of participants. We did not analyse relationships between health outcomes and six PFAS that were detected in blood serum with lower frequency (i.e., perfluorodecanoic acid (PFDA), perfluorononanoic acid (PFNA), perfluorohexanoic acid (PFHxA), perfluoroheptanoic acid (PFHpA), perfluorobutane sulfonic acid (PFBS), and 6:2 fluorotelomer sulfonic acid (6:2 FTS)). In the main analysis, we replaced PFAS values below the limit of quantification with the limit divided by the square root of two, following convention.[74] To address the potential bias induced by this single imputation, in a sensitivity analysis we treated values below the limit of quantification as censored values, which we imputed using multiple imputation by chained equations.[74]

We log-transformed (base 2) blood serum concentrations of PFAS to express effects per doubling in PFAS serum concentrations. We used this scale so that effect sizes were comparable across the communities and for ease of interpretation. Interpretation of effects is explained below in Box 2.

Covariates

We considered the following sociodemographic and biochemical factors as potential confounders in our various analyses: age; sex; BMI (weight in kilograms divided by squared height in metres categorised into underweight (<18.5 kg/m^2), normal (18.5 to <25 kg/m^2), overweight (25 to <30 kg/m^2), and obese (≥30 kg/m^2)); highest level of education (combined into three categories: bachelor degree level and higher, certificate or diploma, and high school or lower); gross household annual income (five categories: ≤$25,999, $26,000–$64,999, $65,000–$129,999, $130,000–

$233,999, ≥$234,000); marital status (combined into two categories: married or de facto, and other); smoking status (combined into two categories: never and ever), alcohol consumption (categorised according to NHMRC guidelines:[75] none, within guideline (≤10 standard drinks per week), exceeds guideline (>10 standard drinks per week)); physical activity (duration and intensity

Box 2. Guide to interpretation of results

The prevalence of a health outcome refers to the percentage of participants with a particular outcome in the study. In the Cross-sectional Survey, we report prevalence ratios (denoted as PR), which tell us how much more or less common a health outcome is in one group of participants relative to another group of participants. We estimate two main sets of prevalence ratios: to compare the prevalence of health outcomes in exposed and comparison communities (Model 1), and, separately, to assess whether the prevalence of a health outcome changes with increasing PFAS blood serum concentrations (Model 2).

Alongside each estimate of a prevalence ratio, we report an accompanying 95% confidence interval (denoted as 95% CI). The CI tells us how the prevalence ratio estimate may vary if we were to repeat this study many times. Wide confidence intervals indicate less certainty or 'confidence' in the results, while narrow intervals indicate more certainty or 'confidence' in the results. We therefore give a range of estimates for the prevalence ratio that are compatible with our data and modelling assumptions (e.g., PR = 1.88, 95% CI 1.30 to 2.73).

If the prevalence ratio and its CI are greater than 1.00, our results suggest that the prevalence of a health outcome is likely to be higher in the exposed community than in the comparison community (Model 1), or that it is higher with increasing PFAS concentrations (Model 2). Conversely, if the prevalence ratio and its CI are below 1.00, our data point to the conclusion that the prevalence is lower in the exposed community or that it is lower with increasing PFAS concentrations.

The further away that the prevalence ratio is from 1.00, in either direction, the stronger the association. For example, in Model 1, a prevalence ratio of 0.50 suggests that the prevalence of a health outcome in an exposed community is half the prevalence in the comparison community (referred to as an 'inverse' association), whereas a prevalence ratio of 2.00 suggests that the prevalence of a health outcome in an exposed community is twice the prevalence in the comparison community (referred to as a 'positive' association).

If the CI includes 1.00, the data are compatible with 'no association' (i.e., no difference in prevalence) along with other possibilities:

1. If the upper and lower limits of the CI are close to 1.00 (e.g., 0.96 to 1.04), our data point to the conclusion that there is no meaningful difference in prevalence between the exposed and comparison communities (Model 1), or with increasing PFAS concentrations (Model 2).
2. If one of the CI limits is close to 1.00 (e.g. 0.95 to 3.90), the prevalence is likely different, but too imprecisely estimated to confidently conclude that there is an association ('uncertain').
3. If the CI is wide and neither of its limits are close to 1.00 (e.g. 0.50 to 3.90), we are unable to conclude whether or not the prevalence is different, and the difference could range from anywhere between much lower to much higher.

When an absolute difference measure is used rather than a ratio (e.g., difference in means), the reference point of no difference is zero instead of 1.00. That is, if the difference estimate and its CI are greater than zero, the data are most compatible with the conclusion that the mean is higher in the exposed community than in the comparison community; conversely, if the difference estimate and its CI are below zero, the data are most compatible with the conclusion that the mean is lower in the exposed community.

These interpretations need to be considered in the context of our modelling assumptions and possible biases in our data collection and analysis, as outlined in the Discussion sections 'Strengths and limitations'.

of exercise were converted into metabolic equivalent minutes and categorised into four categories according to the Department of Health physical activity guidelines:[76,77] nil/sedentary, low (i.e., below 150 minutes of moderate intensity or 75 minutes of high intensity physical activity per week), moderate (i.e., 150 to <300 minutes of moderate intensity or 75 to <150 minutes of high intensity physical activity per week), and high (i.e., ≥300 minutes of moderate intensity or ≥150 minutes of high intensity physical activity per week)); the eGFR (calculated using the CKD-EPI formula based on age, sex, and serum creatinine concentrations);[78] length of time living in a PFAS Management Area (decades, continuous variable); bore water use on property (never or ever), occupational AFFF exposure (yes or no, defined in the Blood Serum Study report),[65] and current (versus former) residence or work in a PFAS Management Area. Categories were determined based on sample size and clinical relevance.

Statistical analysis

Self-reported health outcomes

We calculated the crude prevalence of self-reported health outcomes in exposed and comparison communities. We then estimated adjusted prevalence ratios of self-reported health outcomes: (1) between participants in exposed versus comparison communities, and (2) per doubling in PFAS serum concentrations, to quantify the association between PFAS and these health outcomes (summarised in Table 1). We estimated prevalence ratios separately for each exposed community and comparison community pair.

We used modified Poisson regression models with a log link and robust error variance. Models were estimated via generalised estimating equations using an exchangeable correlation structure, to account for clustering of participants within households. When convergence could not be achieved using an exchangeable correlation structure, we used an independence correlation structure and cluster-robust standard errors. Linearity of relationships between self-reported health outcomes and continuous covariates (including PFAS serum concentrations) were assessed using univariable generalised additive models. Age was subsequently modelled using a restricted cubic spline with three knots. We modelled gross household annual income as an ordinal variable using category midpoints in the middle categories, and upper and lower limits in the lowest and highest categories, respectively. Analyses were conducted in Stata/SE v16.1 (StataCorp LLC, College Station, TX).

The models of PFAS serum concentrations included an interaction term between PFAS concentration and community membership so that PFAS effects were estimated separately for exposed and comparison communities. We did this as exposed and comparison communities were exposed to different mixtures of PFAS and were sampled several years apart. We report the effect estimates for each exposed community. Effect estimates for comparison communities are presented in Table A1-1 and not discussed further.

We adjusted for potential confounding variables (i.e., variables thought to be associated with both the exposure and outcome) as follows. In the primary analysis, we adjusted for sociodemographic variables: sex, age, education level, and gross household annual income. In a sensitivity analysis for the models of self-reported health outcomes and PFAS serum concentrations, we additionally adjusted for the eGFR (a measure of kidney function) and variables that may affect kidney function, including smoking status and alcohol consumption, on the basis that impaired kidney function may affect the renal excretion of PFAS and thus PFAS serum concentrations.[79] We did not adjust for BMI because it may be on the causal pathway between PFAS exposure and health outcomes, or physical activity.[80]

In the models of self-reported health outcomes and PFAS serum concentrations, we conducted a series of additional sensitivity analyses; we (1) excluded exposed participants who were currently residing in comparison communities; (2) restricted age to 25 years and over; (3) excluded exposed participants who had not lived in the exposed communities in the previous 5, 10, or 15 years, and excluded past workers, because their PFAS serum concentrations at the time of blood collection

may be least reflective of their long-term PFAS exposure levels; (4) treated PFAS concentrations below the limit of quantification as censored values that we imputed using multiple imputation by chained equations; and, (5) assessed the impact of missing values in confounder variables using multiple imputation by chained equations.

Table 1. Summary of analyses of self-reported health and psychological distress outcomes.

Exposure variables	Purpose
Models of self-reported health outcomes	
Exposed versus comparison community membership	To identify health outcomes that are more common in the exposed communities than the comparison communities.
PFAS concentrations in blood serum	To assess whether PFAS serum concentrations are associated with self-reported health.
Models of self-reported psychological distress outcomes	
Exposed versus comparison community membership	To assess whether the levels of psychological distress were higher in the exposed communities than the comparison communities.
PFAS concentrations in blood serum	To assess whether PFAS serum concentrations are associated with psychological distress in the exposed communities.
Factors that may affect the perceived risk of PFAS exposure, including occupational exposure to AFFF, bore water use, current or former residence or work in a PFAS Management Area, and length of time residing in a PFAS Management Area.	To identify risk factors for psychological distress in the exposed communities.
Concerns regarding health and mental health	To identify risk factors for psychological distress in the exposed communities.

Psychological distress outcomes

We conducted four analyses, summarised in Table 1. First, to compare psychological distress scores in exposed and comparison communities, we estimated differences in mean scores and prevalence ratios of high or 'clinically-significant' scores between participants in the exposed and comparison communities. Second, to assess relationships between psychological distress and PFAS exposure in the exposed communities, we estimated differences in mean scores and prevalence ratios of clinically-significant scores per doubling in PFAS serum concentrations. Third, to identify factors contributing to psychological distress in the exposed communities, we estimated relationships between psychological distress and factors that may affect the perceived risk of PFAS exposure, including length of time living in a PFAS Management Area, bore water use, occupational AFFF exposure, and current (versus former) residence or work in a PFAS Management Area. Fourth, we estimated relationships between psychological distress and level of concern about health and mental health.

We used linear regression models to estimate differences in mean psychological distress scores and modified Poisson regression models with log link and robust error variance to estimate

prevalence ratios of clinically-significant psychological distress scores. Models were estimated as specified in the previous section on the models of self-reported health outcomes.

We adjusted for potential confounding variables as follows. In models comparing psychological distress scores between exposed and comparison communities, we adjusted for sociodemographic variables: sex, age, education level, and gross household annual income. In models of psychological distress and PFAS serum concentrations in the exposed communities, we adjusted for sex, age, education level, gross household annual income, and factors that may affect both PFAS serum concentrations and the perceived risk of PFAS exposure: length of time living in a PFAS Management Area (per decade), bore water use (ever versus never), occupational AFFF exposure (yes versus no), and current (versus former) residence or work in a PFAS Management Area. To estimate effects for factors that may affect the perceived risk of PFAS exposure, we separately estimated a model without PFAS serum concentrations, as PFAS may be on the causal pathway between these factors and psychological distress. In models of psychological distress and health concerns, we adjusted for sex, age, education level, and gross household annual income. We estimated effects separately for each community (or pair of exposed and comparison communities), except in the model of psychological distress and health concerns, which are reported in aggregate for the three exposed communities as they were not expected to vary substantially across the three communities.

We conducted a series of sensitivity analyses in models comparing psychological distress scores between exposed and comparison communities, and in models of psychological distress and PFAS serum concentrations in exposed communities. We: (1) excluded exposed participants who were currently residing in comparison communities; and, (2) additionally adjusted for marital status (which may be a potential risk factor or protective factor for stress related to chronic environmental contamination).[81,82] In models comparing psychological distress scores between exposed and comparison communities, we also assessed the impact of missing values in confounder variables using multiple imputation by chained equations. In models of psychological distress and PFAS serum concentrations in the exposed communities, in addition to the above two numbered sensitivity analyses, we: (3) additionally adjusted for smoking status and alcohol consumption; and, (4) treated PFAS concentrations below the limit of quantification as censored values that we imputed using multiple imputation by chained equations.

Health concerns and health-related behaviours

We calculated proportions in each category of health concerns and health-related behaviours for all exposed communities combined.

PFAS blood test experiences and perceptions

We used thematic analysis to categorise the responses to the open-ended questions about the usefulness of the blood testing for PFAS through the VBTP. The categories reflected the perceptions and experiences of the participants. Participant responses were reviewed independently and in duplicate by the study team. We used an inductive approach to categorise the responses.[83] Categories were not mutually exclusive, so the sum of the responses across categories does not necessarily equal the total number of responses.

Ethical considerations

The design and methods of the Cross-sectional Survey were approved by the Northern Territory Department of Health and the Menzies School of Health Research Human Research Ethics Committee (protocol 2018-3130) and the ANU Human Research Ethics Committee (protocol 2016/707) in an initial ethics submission in 2016 and a series of amendments from 2017–2020. The ethics applications addressed the inclusion of children (<16 years old) and Aboriginal and Torres Strait Islander persons in the study.

Deviations from the study protocol

During the course of the Cross-sectional Survey there were some changes from the original protocol published in 2018.[84] These changes included:

- **Inclusion of children**: we proposed to recruit children in the comparison population; however, this was not feasible due to the small number of child participants in the PFAS Management Areas and additional barriers to recruitment.
- **Classification of residents**: we proposed classifying participants in exposed communities as either current residents or other participants. Instead, we classified participants as having ever lived or worked in a PFAS Management Area.
- **Changes to the questionnaire**: due to delays in recruiting participants in comparison communities, an additional three questions were included in the questionnaire for the comparison participants that measured the impact of the COVID-19 pandemic on the lives of these participants.

Results

Participation in the Cross-sectional Survey

Recruitment and study participation for the Cross-sectional Survey and Blood Serum Study are shown in Figure 2. In the exposed communities, a total of 917 people (881 adults, 36 children) completed the survey, including 32% (813/2,521) of the Blood Serum Study participants who took part in the VBTP for PFAS in 2019, a further 28 non-VBTP participants recruited for the survey in 2019, and 9% (76/877) of the recruited comparison participants who were re-classified as exposed, based on the residential and work history reported in the survey. Of the 881 adult participants from the exposed communities, 80% (705/881) were current or former residents and the remainder were workers only. There were 388 participants in Katherine, 224 in Oakey, and 424 in Williamtown, which includes 144 participants who resided or worked in multiple communities.

In the comparison communities, we recruited 1,115 adults from the 30,000 residents randomly sampled from the Medicare Enrolment File in Alice Springs, Dalby, or Kiama and Shellharbour. A total of 877 of these adults (2.9% of 30,000) completed the survey, with 801 classified as comparison participants after re-classifying as exposed the 76 participants who had previously lived or worked in one or more of the exposed communities. The sample of 801 adult comparison participants included 204 participants in Alice Springs, 166 participants in Dalby, and 431 participants in Kiama and Shellharbour.

Participant characteristics

There were more male than female participants in Oakey and Williamtown (70% and 66% male, respectively), whereas there were fewer male than female participants in the comparison communities of Alice Springs, Dalby, and Kiama and Shellharbour (ranging from 38% to 43% male) (Table 2). Participants in Katherine were younger than participants in Alice Springs, more likely to be overweight or obese, sedentary, born in Australia, and less likely to be educated at Bachelor degree level or higher. Similarly, participants in Williamtown were younger than participants in Kiama and Shellharbour, more likely to be obese and sedentary, and less likely to be educated at Bachelor degree level or higher. Participants in Oakey were also younger than participants in Dalby, and were more likely to be overweight and in higher household income brackets.

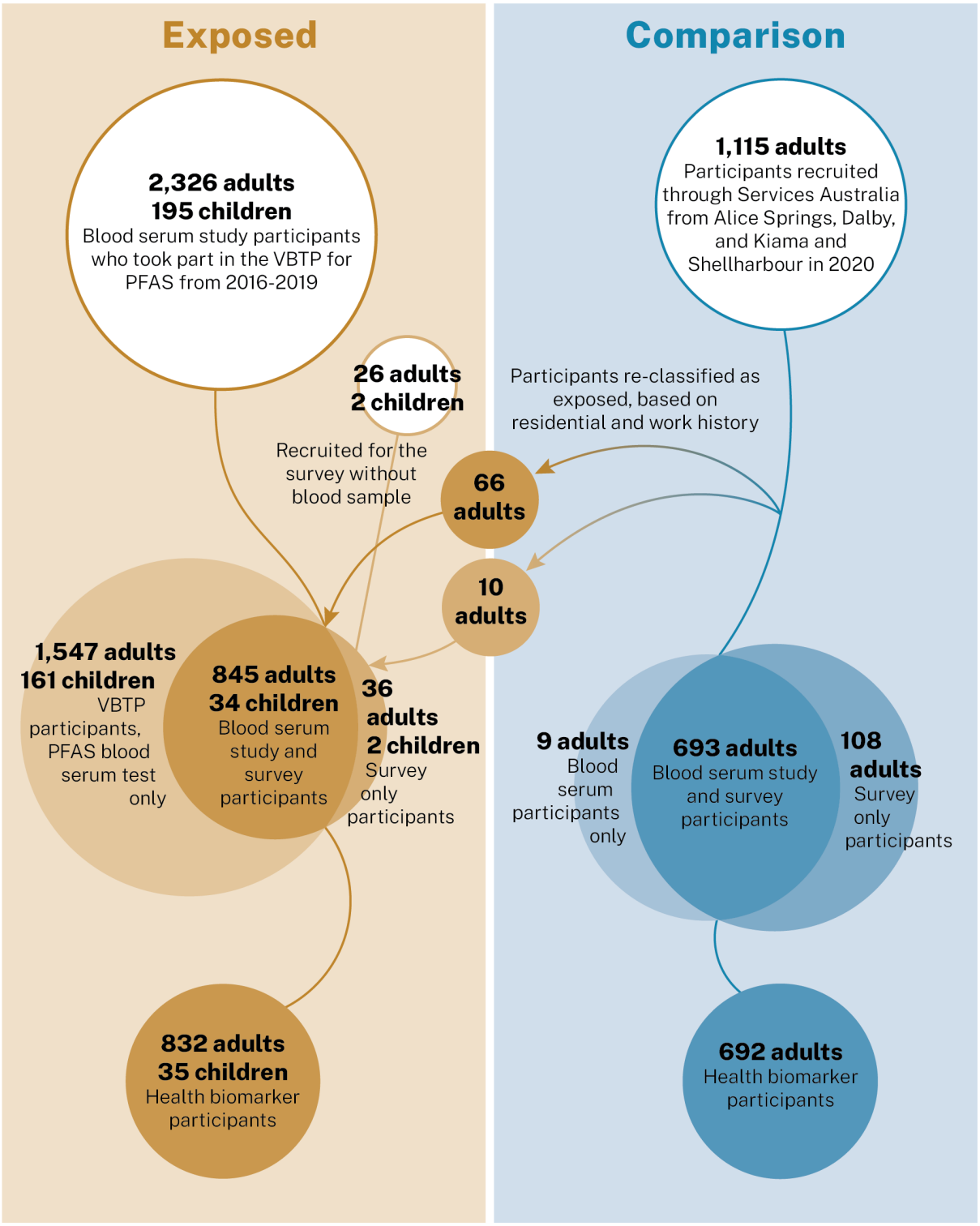
Compared to the general populations of their communities, study participants in Oakey and Williamtown were more likely to be male (51% and 56% male, respectively, according to the Australian Census 2016).[68] Across all exposed communities, study participants were more likely to be older (54%, 42%, and 34% of the general populations of Katherine, Oakey, and Williamtown, respectively, were 45 years or older),[68] born in Australia (66%, 77%, and 71%, respectively),[68] and more highly educated (14%, 5%, and 9%, respectively, were educated at Bachelor degree level or higher)[68] than the general population of their communities (Table 2).

Self-reported health

Crude prevalence of self-reported health outcomes

The crude (unadjusted) prevalence of self-reported health outcomes is presented in Table 3. The most common health outcomes reported in the exposed communities were high blood pressure (Katherine 23.2%; Oakey 27.1%; Williamtown 28.5%), hypercholesterolaemia (Katherine 19.8%; Oakey 26.7%; Williamtown 28.0%), asthma (Katherine 14.9%; Oakey 15.7%; Williamtown 15.5%), and problems with fertility (Katherine 10.0%; Oakey 13.0%; Williamtown 12.5%). Many of the health outcomes were rare with few reported cases. Neurological conditions (including dementia, Parkinson's disease, and motor neurone disease) and cancer (in particular bone cancer, brain cancer, and leukaemia) were the least common health outcomes.

Figure 2. Blood Serum Study and Cross-sectional Survey participation in PFAS Management Areas and comparison communities, 2016–2020.



In the exposed communities, 879 people (845 adults, 34 children) participated in the Blood Serum Study and the Cross-sectional Survey, including 813 VBTP participants and 66 comparison participants who were re-classified as exposed based on residential and work history. A further 38 people (36 adults, 2 children) participated in the Cross-sectional Survey only, including 28 people who were recruited for the survey without a blood sample and 10 comparison participants who were re-classified as exposed. This gave 917 survey participants (879+38) in the exposed communities, including 881 adults. In the comparison communities, 801 adults (693+108) participated in the Cross-sectional Survey.

Table 2. Sociodemographic characteristics of adult survey participants from PFAS Management Areas, 2019–2020, and comparison communities, 2020.

	Katherine and Alice Springs (NT)		Oakey and Dalby (Qld)		Williamtown and Kiama and Shellharbour (NSW)	
Characteristic	Exposed N (%)	Comparison N (%)	Exposed N (%)	Comparison N (%)	Exposed N (%)	Comparison N (%)
Total sample†	388	204	224	166	424	431
Sex						
Male	199 (51%)	78 (38%)	156 (70%)	72 (43%)	278 (66%)	186 (43%)
Female	189 (49%)	126 (62%)	68 (30%)	94 (57%)	146 (34%)	245 (57%)
Other	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Missing	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Age (years)						
16 to 30	30 (8%)	16 (8%)	11 (5%)	7 (4%)	25 (6%)	21 (5%)
31 to 45	104 (27%)	34 (17%)	40 (18%)	33 (20%)	88 (21%)	48 (11%)
46 to 60	130 (34%)	71 (35%)	98 (44%)	50 (30%)	125 (29%)	99 (23%)
≥61	124 (32%)	83 (41%)	75 (33%)	76 (46%)	186 (44%)	263 (61%)
Missing	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Body mass index (kg/m^2)						
Underweight (<18.5)	2 (1%)	5 (2%)	2 (1%)	1 (1%)	0 (0%)	3 (1%)
Normal (18.5 to <25)	98 (25%)	80 (39%)	45 (20%)	50 (30%)	84 (20%)	159 (37%)
Overweight (25 to <30)	121 (31%)	59 (29%)	85 (38%)	50 (30%)	157 (37%)	173 (40%)
Obese (≥30)	100 (26%)	49 (24%)	70 (31%)	55 (33%)	110 (26%)	84 (19%)
Missing	67 (17%)	11 (5%)	22 (10%)	10 (6%)	73 (17%)	12 (3%)
Physical activity (Department of Health categories)						
Nil/sedentary	76 (20%)	22 (11%)	32 (14%)	23 (14%)	86 (20%)	48 (11%)
Low	81 (21%)	41 (20%)	35 (16%)	38 (23%)	81 (19%)	66 (15%)
Moderate	72 (19%)	42 (21%)	44 (20%)	27 (16%)	69 (16%)	91 (21%)
High	144 (37%)	88 (43%)	98 (44%)	57 (34%)	167 (39%)	201 (47%)
Missing	15 (4%)	11 (5%)	15 (7%)	21 (13%)	21 (5%)	25 (6%)
Smoking status						

	Katherine and Alice Springs (NT)		Oakey and Dalby (Qld)		Williamtown and Kiama and Shellharbour (NSW)	
Characteristic	**Exposed N (%)**	**Comparison N (%)**	**Exposed N (%)**	**Comparison N (%)**	**Exposed N (%)**	**Comparison N (%)**
Never	232 (60%)	127 (62%)	135 (60%)	118 (71%)	257 (61%)	298 (69%)
Past	106 (27%)	66 (32%)	69 (31%)	39 (23%)	124 (29%)	118 (27%)
Current	34 (9%)	10 (5%)	14 (6%)	9 (5%)	29 (7%)	14 (3%)
Missing	16 (4%)	1 (0%)	6 (3%)	0 (0%)	14 (3%)	1 (0%)
Alcohol consumption (NHMRC 2020 guidelines)						
No alcohol	101 (26%)	53 (26%)	61 (27%)	67 (40%)	103 (24%)	105 (24%)
Within guideline (≤10 standard drinks per week)	197 (51%)	108 (53%)	102 (46%)	71 (43%)	228 (54%)	248 (58%)
Exceeds guideline (>10 standard drinks per week)	51 (13%)	36 (18%)	39 (17%)	23 (14%)	65 (15%)	63 (15%)
Missing	39 (10%)	7 (3%)	22 (10%)	5 (3%)	28 (7%)	15 (3%)
Marital status						
Married or de facto	290 (75%)	130 (64%)	161 (72%)	127 (77%)	339 (80%)	334 (77%)
Other	82 (21%)	74 (36%)	56 (25%)	39 (23%)	73 (17%)	96 (22%)
Missing	16 (4%)	0 (0%)	7 (3%)	0 (0%)	12 (3%)	1 (0%)
Country of birth						
Australia	313 (81%)	141 (69%)	195 (87%)	142 (86%)	368 (87%)	346 (80%)
Other	64 (16%)	63 (31%)	22 (10%)	24 (14%)	45 (11%)	85 (20%)
Missing	11 (3%)	0 (0%)	7 (3%)	0 (0%)	11 (3%)	0 (0%)
Aboriginal and Torres Strait Islander status						
No	357 (92%)	199 (98%)	214 (96%)	164 (99%)	402 (95%)	426 (99%)
Yes	18 (5%)	5 (2%)	4 (2%)	2 (1%)	9 (2%)	4 (1%)
Missing	13 (3%)	0 (0%)	6 (3%)	0 (0%)	13 (3%)	1 (0%)
Level of education						
Bachelor degree or higher	142 (37%)	109 (53%)	73 (33%)	50 (30%)	126 (30%)	188 (44%)

	Katherine and Alice Springs (NT)		Oakey and Dalby (Qld)		Williamtown and Kiama and Shellharbour (NSW)	
Characteristic	Exposed N (%)	Comparison N (%)	Exposed N (%)	Comparison N (%)	Exposed N (%)	Comparison N (%)
Certificate or diploma	181 (47%)	72 (35%)	115 (51%)	66 (40%)	222 (52%)	181 (42%)
High school certificate or lower	44 (11%)	20 (10%)	23 (10%)	43 (26%)	56 (13%)	52 (12%)
Missing	21 (5%)	3 (1%)	13 (6%)	7 (4%)	20 (5%)	10 (2%)
Gross household annual income						
≤$25,999	22 (6%)	15 (7%)	16 (7%)	32 (19%)	33 (8%)	45 (10%)
$26,000 to $64,999	43 (11%)	21 (10%)	33 (15%)	41 (25%)	62 (15%)	108 (25%)
$65,000 to $129,999	149 (38%)	83 (41%)	82 (37%)	46 (28%)	142 (33%)	124 (29%)
$130,000 to $233,999	79 (20%)	58 (28%)	41 (18%)	23 (14%)	62 (15%)	64 (15%)
≥$234,000	10 (3%)	7 (3%)	5 (2%)	2 (1%)	9 (2%)	15 (3%)
Missing	85 (22%)	20 (10%)	47 (21%)	22 (13%)	116 (27%)	75 (17%)

N: sample size.

† In exposed communities, the total sample was defined as ever living or working in the PFAS Management Area, including participants who have lived or worked across multiple PFAS Management Areas.

Table 3. Crude prevalence of self-reported health outcomes in participants from PFAS Management Areas, 2019–2020, and comparison communities, 2020.

	Katherine and Alice Springs (NT)		Oakey and Dalby (Qld)		Williamtown and Kiama and Shellharbour (NSW)	
	Exposed % (cases/N)	Comparison % (cases/N)	Exposed % (cases/N)	Comparison % (cases/N)	Exposed % (cases/N)	Comparison % (cases/N)
Bone cancer	Low (≤5/377)	Low (≤5/204)	Low (≤5/218)	Low (≤5/163)	Low (≤5/411)	Low (≤5/424)
Brain cancer	Low (≤5/377)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/411)	Low (≤5/423)
Breast cancer	4.0% (15/377)	Low (≤5/204)	Low (≤5/218)	4.3% (7/163)	2.4% (10/412)	3.3% (14/423)
Kidney cancer	Low (≤5/377)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/411)	Low (≤5/423)
Leukaemia	Low (≤5/377)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/412)	Low (≤5/423)

	Katherine and Alice Springs (NT)		Oakey and Dalby (Qld)		Williamtown and Kiama and Shellharbour (NSW)	
	Exposed % (cases/N)	Comparison % (cases/N)	Exposed % (cases/N)	Comparison % (cases/N)	Exposed % (cases/N)	Comparison % (cases/N)
Liver cancer	Low (≤5/377)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/411)	Low (≤5/423)
Ovarian cancer	Low (≤5/178)	Low (≤5/126)	Low (≤5/67)	Low (≤5/93)	Low (≤5/139)	Low (≤5/240)
Prostate cancer	Low (≤5/200)	Low (≤5/78)	4.6% (7/152)	11.3% (8/71)	4.8% (13/272)	8.1% (15/185)
Testicular cancer	Low (≤5/200)	Low (≤5/78)	Low (≤5/152)	Low (≤5/70)	Low (≤5/273)	Low (≤5/183)
Thyroid cancer	Low (≤5/377)	Low (≤5/204)	Low (≤5/216)	Low (≤5/163)	Low (≤5/411)	1.9% (8/423)
Heart attack	2.4% (9/379)	3.9% (8/203)	8.6% (19/220)	5.5% (9/163)	6.8% (28/412)	5.6% (24/425)
High blood pressure	23.2% (88/379)	23.5% (48/204)	27.1% (60/221)	24.1% (39/162)	28.5% (119/418)	30.4% (130/427)
Hypercholesterolaemia	19.8% (75/379)	17.6% (36/204)	26.7% (58/217)	19.8% (32/162)	28.0% (116/415)	28.1% (120/427)
Stroke	Low (≤5/375)	Low (≤5/204)	Low (≤5/218)	Low (≤5/163)	2.2% (9/412)	2.1% (9/423)
Lupus	Low (≤5/375)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/413)	Low (≤5/424)
Ulcerative colitis	Low (≤5/375)	Low (≤5/204)	3.2% (7/217)	Low (≤5/163)	1.5% (6/413)	1.7% (7/424)
Crohn's disease	Low (≤5/375)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/413)	Low (≤5/424)
Multiple sclerosis	Low (≤5/375)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/413)	Low (≤5/423)
Rheumatoid arthritis	4.3% (16/375)	2.9% (6/204)	5.1% (11/217)	Low (≤5/163)	7.5% (31/413)	4.2% (18/424)
Asthma	14.9% (56/376)	15.8% (32/203)	15.7% (34/217)	19.6% (32/163)	15.5% (64/412)	13.9% (59/425)
Type I diabetes	Low (≤5/375)	Low (≤5/203)	Low (≤5/214)	Low (≤5/161)	Low (≤5/408)	Low (≤5/420)
Type II diabetes	4.8% (18/377)	7.4% (15/204)	9.7% (21/217)	4.3% (7/163)	8.9% (37/416)	6.4% (27/424)

	Katherine and Alice Springs (NT)		Oakey and Dalby (Qld)		Williamtown and Kiama and Shellharbour (NSW)	
	Exposed % (cases/N)	Comparison % (cases/N)	Exposed % (cases/N)	Comparison % (cases/N)	Exposed % (cases/N)	Comparison % (cases/N)
Hepatitis (non-infectious)	2.7% (10/376)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	1.5% (6/413)	2.6% (11/426)
Fatty liver disease	6.6% (25/376)	2.9% (6/204)	5.5% (12/218)	3.7% (6/163)	5.8% (24/414)	4.0% (17/424)
Cirrhosis of the liver	Low (≤5/376)	Low (≤5/203)	Low (≤5/217)	Low (≤5/163)	Low (≤5/413)	Low (≤5/423)
Dementia	Low (≤5/376)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/413)	Low (≤5/424)
Parkinson's disease	Low (≤5/376)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/413)	Low (≤5/424)
Motor neurone disease	Low (≤5/376)	Low (≤5/204)	Low (≤5/217)	Low (≤5/163)	Low (≤5/413)	Low (≤5/424)
Hypothyroidism	4.5% (17/379)	10.8% (22/204)	5.0% (11/218)	9.8% (16/163)	3.9% (16/410)	6.6% (28/424)
Hyperthyroidism	3.8% (14/373)	Low (≤5/201)	4.6% (10/217)	Low (≤5/163)	3.9% (16/412)	3.8% (16/423)
Chronic kidney disease	Low (≤5/377)	Low (≤5/204)	4.1% (9/217)	Low (≤5/163)	Low (≤5/414)	1.4% (6/423)
Gout	6.9% (26/376)	3.9% (8/203)	9.2% (20/217)	5.5% (9/164)	9.5% (39/411)	6.4% (27/423)
Problems with fertility	10.0% (32/320)	11.1% (18/162)	13.0% (23/177)	16.8% (24/143)	12.5% (43/345)	8.0% (31/387)
Early onset menopause	9.0% (17/189)	6.3% (8/126)	Low (≤5/68)	7.4% (7/94)	8.2% (12/146)	11.4% (28/245)

N: sample size.

Prevalence was replaced with 'Low' when there were five or fewer participants who reported a health outcome.

Self-reported health outcomes in exposed versus comparison communities

In Katherine compared to Alice Springs, we observed higher prevalence of self-reported history of breast cancer (PR = 3.09, 95% CI 1.17 to 8.17), any cancer (PR = 3.78, 95% CI 1.66 to 8.60), fatty liver disease (PR = 2.42, 95% CI 0.97 to 6.02), and any liver disease (PR = 2.39, 95% CI 1.06 to 5.40), after adjusting for sociodemographic characteristics (Table 4). In Williamtown compared to Kiama and Shellharbour, we observed higher prevalence of self-reported history of rheumatoid arthritis (PR = 2.25, 95% CI 1.04 to 4.90), hypercholesterolaemia (PR = 1.25, 95% CI 0.98 to 1.59), type II diabetes (PR = 1.77, 95% CI 1.01 to 3.09), and problems with fertility (PR = 2.15, 95% CI 1.30 to 3.54). For the remaining self-reported health outcomes in Katherine and Williamtown, the evidence for

Table 4. Adjusted prevalence ratios of self-reported health outcomes between participants in PFAS Management Areas, 2019–2020, and comparison communities, 2020.

	Katherine vs. Alice Springs (NT)		Oakey vs. Dalby (Qld)		Williamtown vs. Kiama and Shellharbour (NSW)	
	Exposed N (cases); comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); comparison N (cases)	Adjusted PR (95% CI)†
Breast cancer	290 (13); 184 (4)	3.09 (1.17,8.17)	170 (2); 139 (4)	NC	299 (8); 348 (12)	1.63 (0.63,4.21)
Prostate cancer	155 (4); 70 (1)	NC	118 (4); 63 (8)	0.65 (0.19,2.30)	199 (11); 156 (13)	1.33 (0.62,2.84)
Any cancer^	290 (22); 184 (6)	3.78 (1.66,8.60)	172 (11); 140 (13)	0.96 (0.45,2.04)	302 (24); 350 (37)	1.03 (0.59,1.78)
Heart attack	292 (4); 183 (5)	NC	172 (14);139 (6)	1.78 (0.77,4.15)	301 (19); 350 (19)	1.51 (0.84,2.71)
High blood pressure	291 (64); 184 (43)	1.21 (0.88,1.67)	172 (42); 138 (30)	1.34 (0.89,2.02)	302 (81); 351 (103)	1.16 (0.92,1.47)
Any cardiovascular disease^	292 (65); 184 (46)	1.15 (0.84,1.57)	173 (43); 139 (34)	1.18 (0.80,1.74)	302 (84); 351 (110)	1.13 (0.91,1.41)
Hypercholesterolaemia	292 (52); 184 (32)	1.25 (0.85,1.86)	170 (38); 139 (24)	1.38 (0.85,2.23)	301 (79); 350 (97)	1.25 (0.98,1.59)
Fatty liver disease	291 (19); 184 (5)	2.42 (0.97,6.02)	172 (9); 139 (5)	1.60 (0.61,4.25)	301 (14); 349 (14)	0.91 (0.46,1.78)
Any liver disease^	292 (26); 184 (7)	2.39 (1.06,5.40)	172 (10); 139 (9)	0.96 (0.41,2.25)	301 (17); 351 (20)	0.91 (0.49,1.69)
Gout	291 (21); 183 (7)	1.21 (0.52,2.78)	171 (15); 139 (7)	1.73 (0.69,4.33)	300 (33); 350 (22)	1.58 (0.92,2.72)
Any kidney disease^	292 (24); 184 (9)	1.37 (0.64,2.93)	172 (19); 139 (9)	2.02 (0.84,4.88)	302 (35); 350 (25)	1.54 (0.91,2.60)
Asthma	292 (44); 183 (30)	0.97 (0.64,1.46)	172 (27); 139 (26)	0.95 (0.56,1.62)	301 (45); 349 (48)	1.05 (0.72,1.55)
Rheumatoid arthritis	291 (11); 184 (6)	1.21 (0.45,3.25)	172 (10); 139 (3)	2.69 (0.87,8.36)	301 (17); 348 (11)	2.25 (1.04,4.90)
Any autoimmune disease^	292 (61); 184 (34)	1.13 (0.79,1.63)	172 (39); 139 (29)	1.22 (0.78,1.92)	301 (68); 349 (61)	1.35 (0.97,1.87)
Type II diabetes	292 (15); 184 (15)	0.69 (0.34,1.40)	171 (14);139 (5)	2.49 (0.85,7.28)	302 (25); 348 (22)	1.77 (1.01,3.09)
Hypothyroidism	293 (10); 184 (21)	0.46 (0.21,1.02)	173 (8); 139 (13)	0.69 (0.28,1.68)	299 (11); 349 (20)	1.13 (0.55,2.34)
Hyperthyroidism	291 (10); 181 (1)	6.65 (0.87,50.80)	172 (6); 139 (4)	2.09 (0.53,8.17)	300 (12); 349 (12)	1.64 (0.74,3.63)
Problems with fertility	248 (30); 147 (18)	1.02 (0.61,1.72)	140 (17); 119 (18)	1.04 (0.60,1.79)	257 (38); 316 (25)	2.15 (1.30,3.54)
Early onset menopause	142 (10); 114 (8)	0.97 (0.41,2.30)	53 (0);77 (7)	NC	104 (9); 196 (21)	0.95 (0.45,1.98)

N: sample size; NC: convergence not achieved; PR: prevalence ratio; CI: confidence interval. Sample sizes differ to those in Table 3 because of missing values in confounders.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

dissimilar prevalence between the communities was limited. Some prevalence ratios were of modest size, but were not estimated with sufficient precision to indicate the direction of the association with certainty (e.g., in Williamtown compared to Kiama and Shellharbour: gout PR = 1.58, 95% CI 0.92 to 2.72, and any kidney disease PR = 1.54, 95% CI 0.91 to 2.60). This was also largely the case when comparing prevalence of self-reported health conditions between Oakey and Dalby (e.g., gout PR = 1.73, 95% CI 0.69 to 4.33, and any kidney disease PR = 2.02, 95% CI 0.84 to 4.88). The limitations of this analysis are described in the Discussion section.

Self-reported health outcomes and PFAS serum concentrations

Cancer

In most cases, we observed lower prevalence of self-reported cancer per doubling in PFAS serum concentrations, after adjusting for potential confounders (Table 5, Figure 3, Figure 4, and Figure 5). However, the prevalence ratios were either not consistent in direction across exposed communities or were not estimated with sufficient precision to demonstrate the existence or direction of the associations with certainty (e.g., breast cancer and PFOS: Katherine PR = 0.71, 95% CI 0.52 to 0.97; Williamtown PR = 1.15, 95% CI 0.72 to 1.83). The findings from the analyses of self-reported cancer were not appreciably changed in sensitivity analyses (Table A1-2 to Table A1-10, Appendix 1).

Cardiovascular outcomes

We observed higher prevalence of self-reported hypercholesterolaemia per doubling in PFOA serum concentrations (Table 5 and Figure 4). The evidence was strongest in Katherine (PR = 1.36, 95% CI 1.08 to 1.71), but considerably weaker in Oakey (PR = 1.06, 95% CI 0.81 to 1.39) and Williamtown (PR = 1.13, 95% CI 0.92 to 1.38). In contrast with the observations for PFOA, there was no meaningful difference in prevalence of hypercholesterolaemia per doubling in PFOS and PFHxS serum concentrations in exposed communities (e.g., PFHxS: Katherine, PR = 1.04, 95% CI 0.93 to 1.16; Oakey, PR = 0.97, 95% CI 0.83 to 1.14; Williamtown, PR = 1.06, 95% CI 0.94 to 1.19) (Table 5, Figure 3, and Figure 5). This was also the case for the prevalence of self-reported heart attacks, high blood pressure, and any cardiovascular disease (e.g., any cardiovascular disease and PFOS: Katherine, PR = 0.96, 95% CI 0.84 to 1.10; Oakey, PR = 0.82, 95% CI 0.67 to 0.99; Williamtown, PR = 1.06, 95% CI 0.93 to 1.21). The findings from the analyses of self-reported cardiovascular outcomes were not appreciably changed in sensitivity analyses (Table A1-2 to Table A1-10, Appendix 1).

Liver disease

We had no clear evidence of associations between self-reported liver disease and PFAS serum concentrations (Table 5, Figure 3, Figure 4, and Figure 5). While some prevalence ratio estimates were in a consistent positive direction (defined in Box 2) across exposed communities, they were mostly small in magnitude and uninformative with regard to the presence or absence of associations (e.g., any liver disease and PFOA: Katherine, PR = 1.15, 95% CI 0.85 to 1.56; Oakey, PR = 1.31, 95% CI 0.77 to 2.24; Williamtown, PR = 1.07, 95% CI 0.76 to 1.50). The findings from the analyses of self-reported liver disease were not markedly changed in sensitivity analyses (Table A1-2 to Table A1-10, Appendix 1).

Kidney disease

We observed higher prevalence of self-reported gout and any kidney disease per doubling in PFOA serum concentrations in Katherine (e.g., gout PR = 1.61, 95% CI 1.09 to 2.37) and Williamtown (e.g., gout PR = 1.54, 95% CI 1.06 to 2.24), but not in Oakey (e.g., gout PR = 0.86, 95% CI 0.52 to 1.41) (Table 5 and Figure 4). In contrast, lower prevalence of gout and any kidney disease per doubling in PFOS and PFHxS serum concentrations was most compatible with our data, given our assumed models, in all exposed communities (e.g., PFOS and any kidney disease, Katherine PR = 0.93, 95% CI 0.78 to 1.11; Oakey PR = 0.74, 95% CI 0.59 to 0.92, Williamtown PR = 0.74, 95% CI 0.62 to 0.89) (Table 5,

Figure 3, and Figure 5). The findings from the analyses of self-reported kidney outcomes were not markedly changed in sensitivity analyses (Table A1-2 to Table A1-10, Appendix 1).

Autoimmune outcomes

For autoimmune outcomes, we had limited evidence of associations with PFAS serum concentrations (Table 5, Figure 3, Figure 4, and Figure 5). Lower prevalence of self-reported rheumatoid arthritis and any autoimmune disease per doubling in PFAS serum concentrations in all exposed communities was most compatible with our data, given our assumed models. However, the prevalence ratios were not estimated with sufficient precision to determine the direction of the associations with certainty (e.g., rheumatoid arthritis and PFOS: Katherine, PR = 0.78, 95% CI 0.53 to 1.15; Williamtown, PR = 0.72, 95% CI 0.50 to 1.05). For asthma, there was no meaningful difference in prevalence per doubling in PFAS serum concentrations (e.g., PFOS: Katherine, PR = 1.00, 95% CI 0.84 to 1.19; Oakey, PR = 1.02, 95% CI 0.80 to 1.31; Williamtown, PR = 1.17, 95% CI 0.96 to 1.42). The findings from the analyses of self-reported autoimmune outcomes were not appreciably changed in sensitivity analyses (Table A1-2 to Table A1-10, Appendix 1).

Endocrine outcomes

We had no clear evidence of associations between self-reported type II diabetes and PFAS serum concentrations (e.g., PFOS: Katherine, PR = 0.85, 95% CI 0.64 to 1.15; Oakey, PR = 0.73, 95% CI 0.52 to 1.01; Williamtown, PR = 0.92, 95% CI 0.68 to 1.24) (Table 5, Figure 3, Figure 4, and Figure 5).

In models of hypothyroidism, hyperthyroidism, and PFAS, prevalence ratios were not consistent in direction across exposed communities and were imprecisely estimated. For example, based on only 8–10 hypothyroidism cases, higher prevalence of self-reported hypothyroidism per doubling in PFOA concentrations, lack of association, and inverse associations were all compatible with our data under our assumed models (Katherine, PR = 2.12, 95% CI 1.30 to 3.47; Oakey, PR = 1.00, 95% CI 0.49 to 2.06; Williamtown, PR = 1.18, 95% CI 0.71 to 1.97). The findings from the analyses of self-reported endocrine outcomes were not markedly changed in sensitivity analyses (Table A1-2 to Table A1-10, Appendix 1); however, the larger prevalence ratios were attenuated when we imputed missing data (Table A1-10, Appendix 1) and prevalence ratios were estimated with greater uncertainty in some sensitivity analyses that restricted the sample to fewer hypothyroidism and hyperthyroidism cases (e.g., Table A1-3, Appendix 1).

Reproductive outcomes

We had no clear evidence of associations between self-reported problems with fertility and PFAS serum concentrations: estimated prevalence ratios were small, not consistent in direction across exposed communities, and were uninformative with regard to the absence or presence of associations (e.g., PFOS: Katherine, PR = 0.90, 95% CI 0.64 to 1.27; Oakey, PR = 1.10, 95% CI 0.81 to 1.50; Williamtown, PR = 0.90, 95% CI 0.72 to 1.14) (Table 5, Figure 3, Figure 4, and Figure 5). This was also the case for early onset menopause (e.g., PFOA: Katherine PR = 0.63, 95% CI 0.40 to 1.01; Williamtown PR = 0.99, 95% CI 0.53 to 1.82). The findings from the analyses of self-reported reproductive outcomes were not markedly changed in sensitivity analyses (Table A1-2 to Table A1-10, Appendix 1).

Psychological distress

Summary statistics of psychological distress outcomes

The crude prevalence of clinically-significant scores on self-reported measures of psychological distress, somatisation, and anxiety were substantially higher in the exposed communities than in the comparison communities (e.g., PHQ-15 score of 10 or higher, exposed 24% to 28%, comparison 8% to 18%) (Table 6). Mean self-reported psychological distress, somatisation, and anxiety scores were also higher in the exposed communities (e.g., PHQ-15, mean scores ranged 6.7 to 7.6) than in the comparison communities (e.g., PHQ-15, mean scores ranged 4.4 to 5.2) (Table 7).

Table 5. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	278 (13)	0.71 (0.52,0.97)	158 (2)	NC	291 (7)	1.15 (0.72,1.83)
PFOA	278 (13)	0.65 (0.35,1.21)	158 (2)	NC	291 (7)	0.73 (0.30,1.77)
PFHxS	278 (13)	0.77 (0.62,0.94)	158 (2)	NC	291 (7)	1.19 (0.90,1.57)
Prostate cancer						
PFOS	151 (4)	NC	113 (3)	0.58 (0.36,0.94)	196 (10)	0.79 (0.46,1.34)
PFOA	151 (4)	NC	113 (3)	0.55 (0.30,1.02)	196 (10)	0.81 (0.32,2.03)
PFHxS	151 (4)	NC	113 (3)	0.55 (0.39,0.77)	196 (10)	0.72 (0.43,1.20)
Any cancer^						
PFOS	278 (22)	0.82 (0.63,1.05)	160 (9)	0.63 (0.44,0.91)	294 (22)	1.00 (0.75,1.34)
PFOA	278 (22)	0.74 (0.49,1.12)	160 (9)	0.74 (0.47,1.19)	294 (22)	0.96 (0.59,1.57)
PFHxS	278 (22)	0.87 (0.73,1.03)	160 (9)	0.67 (0.51,0.88)	294 (22)	0.87 (0.70,1.09)
Heart attack						
PFOS	280 (4)	NC	160 (14)	0.76 (0.55,1.06)	293 (19)	0.86 (0.65,1.15)
PFOA	280 (4)	NC	160 (14)	1.09 (0.76,1.57)	293 (19)	0.83 (0.54,1.28)
PFHxS	280 (4)	NC	160 (14)	0.93 (0.78,1.12)	293 (19)	0.90 (0.73,1.11)
High blood pressure						
PFOS	279 (61)	0.97 (0.85,1.11)	161 (40)	0.81 (0.66,0.99)	294 (80)	1.07 (0.93,1.22)
PFOA	279 (61)	0.90 (0.74,1.11)	161 (40)	0.87 (0.69,1.09)	294 (80)	0.95 (0.78,1.15)
PFHxS	279 (61)	0.93 (0.84,1.02)	161 (40)	0.94 (0.81,1.10)	294 (80)	1.02 (0.91,1.14)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	280 (62)	0.96 (0.84,1.10)	161 (41)	0.82 (0.67,0.99)	294 (83)	1.06 (0.93,1.21)
PFOA	280 (62)	0.90 (0.74,1.10)	161 (41)	0.87 (0.70,1.09)	294 (83)	0.95 (0.78,1.15)
PFHxS	280 (62)	0.92 (0.84,1.01)	161 (41)	0.95 (0.81,1.10)	294 (83)	1.02 (0.91,1.14)
Hypercholesterolaemia						
PFOS	280 (51)	1.09 (0.95,1.24)	159 (36)	1.00 (0.82,1.22)	293 (76)	1.04 (0.91,1.18)
PFOA	280 (51)	1.36 (1.08,1.71)	159 (36)	1.06 (0.81,1.39)	293 (76)	1.13 (0.92,1.38)
PFHxS	280 (51)	1.04 (0.93,1.16)	159 (36)	0.97 (0.83,1.14)	293 (76)	1.06 (0.94,1.19)
Fatty liver disease						
PFOS	279 (18)	1.04 (0.78,1.40)	160 (8)	1.13 (0.79,1.62)	293 (14)	0.88 (0.53,1.45)
PFOA	279 (18)	1.15 (0.80,1.65)	160 (8)	1.26 (0.74,2.16)	293 (14)	1.14 (0.78,1.69)
PFHxS	279 (18)	1.02 (0.79,1.30)	160 (8)	1.29 (0.98,1.69)	293 (14)	0.90 (0.68,1.21)
Any liver disease^						
PFOS	280 (24)	1.05 (0.82,1.34)	160 (9)	0.97 (0.70,1.36)	293 (17)	0.92 (0.60,1.42)
PFOA	280 (24)	1.15 (0.85,1.56)	160 (9)	1.31 (0.77,2.24)	293 (17)	1.07 (0.76,1.50)
PFHxS	280 (24)	1.08 (0.89,1.32)	160 (9)	1.20 (0.94,1.53)	293 (17)	0.90 (0.70,1.16)
Gout						
PFOS	279 (20)	0.90 (0.74,1.09)	159 (13)	0.76 (0.56,1.03)	292 (33)	0.74 (0.62,0.90)
PFOA	279 (20)	1.61 (1.09,2.37)	159 (13)	0.86 (0.52,1.41)	292 (33)	1.54 (1.06,2.24)
PFHxS	279 (20)	0.96 (0.82,1.13)	159 (13)	0.83 (0.63,1.09)	292 (33)	0.84 (0.70,1.01)
Any kidney disease^						
PFOS	280 (23)	0.93 (0.78,1.11)	160 (17)	0.74 (0.59,0.92)	294 (35)	0.74 (0.62,0.89)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA	280 (23)	1.36 (0.84,2.22)	160 (17)	0.89 (0.58,1.38)	294 (35)	1.48 (1.04,2.12)
PFHxS	280 (23)	0.95 (0.82,1.09)	160 (17)	0.88 (0.68,1.15)	294 (35)	0.82 (0.68,0.98)
Asthma						
PFOS	280 (44)	1.00 (0.84,1.19)	160 (25)	1.02 (0.80,1.31)	293 (43)	1.17 (0.96,1.42)
PFOA	280 (44)	0.85 (0.67,1.07)	160 (25)	0.78 (0.58,1.05)	293 (43)	0.93 (0.65,1.32)
PFHxS	280 (44)	1.03 (0.90,1.18)	160 (25)	1.00 (0.80,1.27)	293 (43)	1.05 (0.87,1.27)
Rheumatoid arthritis						
PFOS	279 (11)	0.78 (0.53,1.15)	160 (10)	NC	293 (17)	0.72 (0.50,1.05)
PFOA	279 (11)	0.57 (0.36,0.90)	160 (10)	NC	293 (17)	0.66 (0.34,1.26)
PFHxS	279 (11)	0.94 (0.75,1.17)	160 (10)	NC	293 (17)	0.99 (0.74,1.31)
Any autoimmune disease^						
PFOS	280 (61)	0.91 (0.78,1.06)	160 (36)	0.97 (0.80,1.19)	293 (66)	0.94 (0.78,1.13)
PFOA	280 (61)	0.82 (0.68,0.99)	160 (36)	0.85 (0.67,1.07)	293 (66)	0.81 (0.62,1.05)
PFHxS	280 (61)	0.95 (0.85,1.06)	160 (36)	0.97 (0.82,1.15)	293 (66)	1.01 (0.88,1.17)
Type II diabetes						
PFOS	280 (14)	0.85 (0.64,1.15)	159 (13)	0.73 (0.52,1.01)	294 (24)	0.92 (0.68,1.24)
PFOA	280 (14)	0.76 (0.51,1.13)	159 (13)	0.80 (0.49,1.29)	294 (24)	0.74 (0.50,1.09)
PFHxS	280 (14)	0.96 (0.77,1.20)	159 (13)	0.80 (0.61,1.06)	294 (24)	0.99 (0.80,1.23)
Hypothyroidism						
PFOS	281 (9)	0.83 (0.63,1.10)	161 (8)	0.76 (0.50,1.16)	291 (10)	0.75 (0.53,1.07)
PFOA	281 (9)	2.12 (1.30,3.47)	161 (8)	1.00 (0.49,2.06)	291 (10)	1.18 (0.71,1.97)
PFHxS	281 (9)	0.75 (0.58,0.97)	161 (8)	0.92 (0.55,1.53)	291 (10)	1.08 (0.83,1.39)

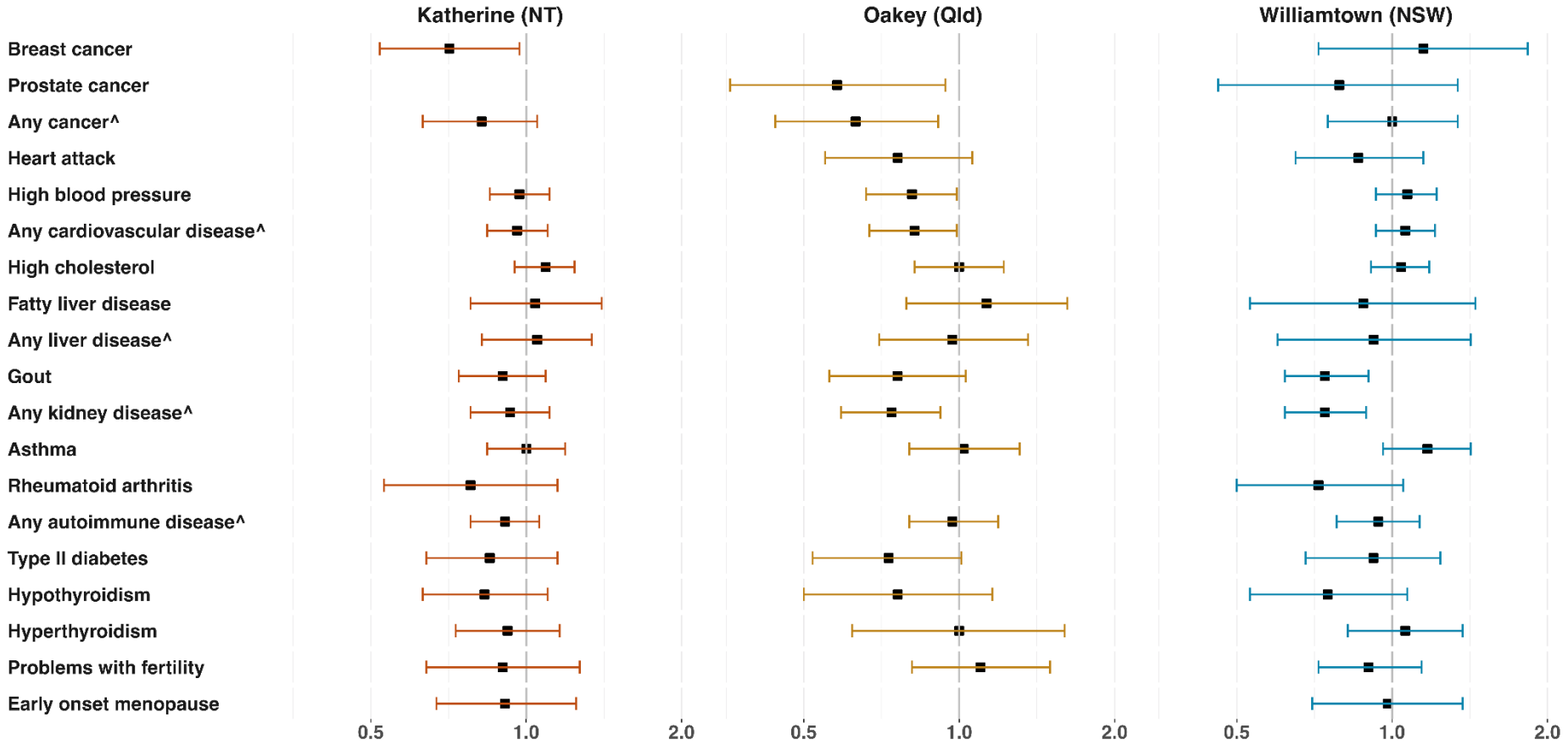
	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	279 (10)	0.92 (0.73,1.16)	160 (5)	1.00 (0.62,1.60)	292 (12)	1.06 (0.82,1.37)
PFOA	279 (10)	0.79 (0.51,1.24)	160 (5)	1.00 (0.48,2.06)	292 (12)	0.93 (0.55,1.58)
PFHxS	279 (10)	0.95 (0.77,1.16)	160 (5)	1.05 (0.61,1.80)	292 (12)	0.95 (0.71,1.27)
Problems with fertility						
PFOS	236 (28)	0.90 (0.64,1.27)	130 (16)	1.10 (0.81,1.50)	251 (37)	0.90 (0.72,1.14)
PFOA	236 (28)	0.80 (0.54,1.19)	130 (16)	0.87 (0.62,1.20)	251 (37)	0.92 (0.68,1.26)
PFHxS	236 (28)	0.91 (0.73,1.12)	130 (16)	1.22 (0.93,1.61)	251 (37)	0.82 (0.67,1.00)
Early onset menopause						
PFOS	134 (10)	0.91 (0.67,1.25)	46 (0)	NC	99 (9)	0.98 (0.70,1.37)
PFOA	134 (10)	0.63 (0.40,1.01)	46 (0)	NC	99 (9)	0.99 (0.53,1.82)
PFHxS	134 (10)	1.00 (0.80,1.25)	46 (0)	NC	99 (9)	1.00 (0.74,1.35)

N: sample size; NC: convergence not achieved; PR: prevalence ratio; CI: confidence interval. Sample sizes differ to those in Table 3 because of missing values in confounders.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

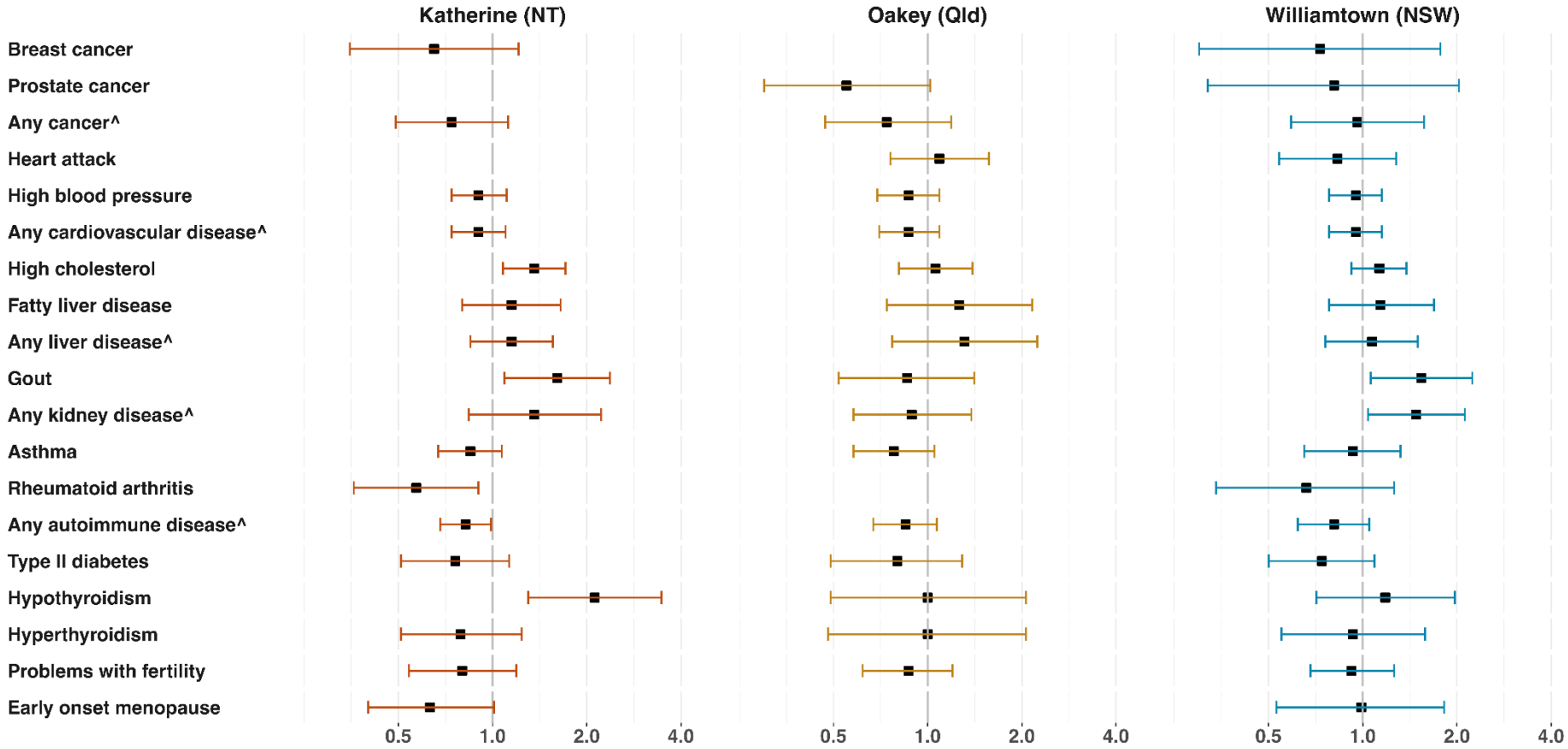
Figure 3. Forest plot showing adjusted prevalence ratios of self-reported health outcomes per doubling in PFOS serum concentrations in participants from PFAS Management Areas, 2016–2020.



Prevalence ratios were adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots. Some associations are missing due to non-convergence. A base-2 log scale is used for the *x*-axis. See Table 5 for sample sizes, number of cases, and prevalence ratios.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

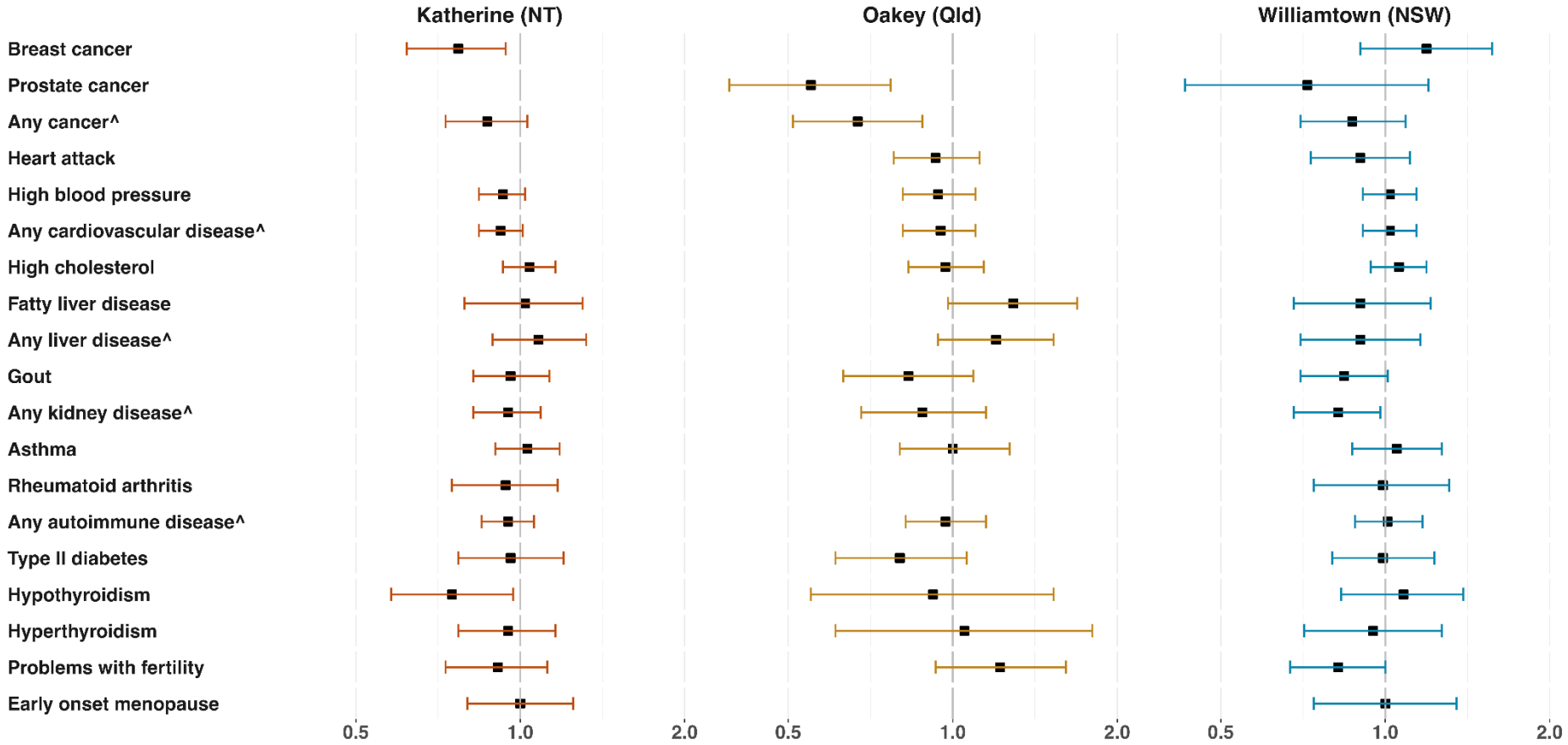
Figure 4. Forest plot showing adjusted prevalence ratios of self-reported health outcomes per doubling in PFOA serum concentrations in participants from PFAS Management Areas, 2016–2020.



Prevalence ratios were adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots. Some associations are missing due to non-convergence. A base-2 log scale is used for the *x*-axis. See Table 5 for sample sizes, number of cases, and prevalence ratios.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Figure 5. Forest plot showing adjusted prevalence ratios of self-reported health outcomes per doubling in PFHxS serum concentrations in participants from PFAS Management Areas, 2016–2020.



Prevalence ratios were adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots. Some associations are missing due to non-convergence. A base-2 log scale is used for the *x*-axis. See Table 5 for sample sizes, number of cases, and prevalence ratios.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table 6. Crude prevalence of clinically-significant self-reported psychological distress scores in participants from PFAS Management Areas, 2019–2020, and comparison communities, 2020.

	Katherine and Alice Springs (NT)		Oakey and Dalby (Qld)		Williamtown and Kiama and Shellharbour (NSW)	
	Exposed % (cases/N)	Comparison % (cases/N)	Exposed % (cases/N)	Comparison % (cases/N)	Exposed % (cases/N)	Comparison % (cases/N)
PHQ-15 score ≥10	24% (84/356)	8% (16/198)	28% (58/205)	18% (27/154)	27% (105/396)	12% (50/415)
K6 score ≥13	21% (77/372)	12% (25/203)	26% (56/216)	13% (22/164)	22% (92/413)	12% (49/425)
DQ5 score ≥14	20% (75/378)	14% (29/203)	28% (60/217)	8% (13/164)	21% (85/411)	12% (52/423)
GAD-7 score ≥10	10% (36/374)	4% (8/203)	18% (38/217)	5% (8/162)	11% (47/412)	6% (26/426)

N: sample size; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15.

Table 7. Summary statistics of self-reported psychological distress scores in participants from PFAS Management Areas, 2019–2020, and comparison communities, 2020.

	Exposed					Comparison				
	N	Mean	p25	Median	p75	N	Mean	p25	Median	p75
Katherine and Alice Springs, NT										
PHQ-15 score	356	6.7	3	6	9	198	4.4	2	4	7
K6 score	372	9.8	6	8	12	203	8.7	6	8	10
DQ5 score	378	9.9	6	9	12	203	8.7	6	8	10
GAD-7 score	374	3.6	0	2	5	203	2.4	0	1	4
Oakey and Dalby, Qld										
PHQ-15 score	205	7.6	4	7	10	154	5.2	2	4	8
K6 score	216	10.8	7	9	13	164	8.7	6	7	9.5
DQ5 score	217	10.6	7	10	14	164	8.5	5	7	11
GAD-7 score	217	4.6	0	3	7	162	2.3	0	1	3
Williamtown and Kiama and Shellharbour, NSW										
PHQ-15 score	396	6.9	3	6	10	415	4.9	2	4	7
K6 score	413	10.0	6	8	12	425	8.5	6	7	10
DQ5 score	411	10.0	6	9	13	423	8.5	5	7	10
GAD-7 score	412	3.8	0	2	6	426	2.5	0	1	4

N: sample size; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; p25: 25th percentile; p75: 75th percentile.

Psychological distress in the exposed versus comparison communities

We observed substantially higher prevalence of clinically-significant scores on self-reported measures of psychological distress, somatisation, and anxiety in exposed communities compared

to their corresponding comparison communities, after adjusting for sociodemographic characteristics (Table 8). For example, we estimated that the prevalence of self-reported somatisation scores of clinical significance were 3.65 times as high in Katherine than in Alice Springs (95% CI 2.04 to 6.56), 1.82 times as high in Oakey than in Dalby (95% CI 1.16 to 2.85), and 2.26 times as high in Williamtown than in Kiama and Shellharbour (95% CI 1.56 to 3.28). In a few cases, prevalence ratios were not estimated with sufficient precision to give certainty as to the direction of the association; however, the direction of the associations were consistently positive (defined in Box 2) across communities for all psychological distress measures (e.g., GAD-7 score of 10 or higher: Katherine compared to Alice Springs PR = 2.82, 95% CI 1.16 to 6.89; Oakey compared to Dalby PR = 3.30, 95% CI 1.25 to 8.67; Williamtown compared to Kiama and Shellharbour PR = 1.22, 95% CI 0.66 to 2.25).

When comparing mean self-reported psychological distress, somatisation, and anxiety scores between exposed and comparison communities, we observed higher mean scores in exposed communities for all psychological distress measures (Table A2-1, Appendix 2). Effect sizes did not change materially in sensitivity analyses (Table A2-2 to Table A2-7, Appendix 2).

Table 8. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores in participants from PFAS Management Areas, 2019–2020, versus comparison communities, 2020.

	Katherine vs. Alice Springs (NT)		Oakey vs. Dalby (Qld)		Williamtown vs. Kiama and Shellharbour (NSW)	
	Exposed N (cases); comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); comparison N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10	281 (71); 180 (12)	3.65 (2.04,6.56)	164 (43); 133 (22)	1.82 (1.16,2.85)	293 (72); 345 (39)	2.26 (1.56,3.28)
K6 score ≥ 13	291 (61); 184 (21)	1.69 (1.09,2.62)	171 (37); 140 (19)	1.94 (1.16,3.24)	300 (62); 349 (40)	1.58 (1.06,2.35)
DQ5 score ≥ 14	293 (56); 184 (24)	1.31 (0.86,2.00)	172 (38); 140 (11)	3.62 (1.95,6.75)	300 (54); 347 (43)	1.26 (0.85,1.87)
GAD-7 score ≥ 10	291 (28); 184 (5)	2.82 (1.16,6.89)	172 (21); 138 (6)	3.30 (1.25,8.67)	301 (28); 349 (21)	1.22 (0.66,2.25)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval. Sample sizes differ to those in Table 6 because of missing values in confounders.

† Adjusted for age, sex, level of education, and gross household annual income.

Psychological distress and PFAS serum concentrations

We had very limited evidence of positive associations between self-reported psychological distress, somatisation, and anxiety levels and PFAS exposure. Per doubling in PFAS serum concentrations, slightly lower mean scores and lower prevalence of clinically-significant scores on self-reported measures of psychological distress were most compatible with our data and models, after adjusting for confounders, in almost all cases (Table 9 and Table A2-8, Appendix 2). However, in most cases, we were not able to determine the existence or direction of the associations with sufficient certainty. For example, prevalence ratio estimates ranged between 0.71 and 1.07 for self-reported psychological distress per doubling in PFAS serum concentrations, with inconsistent direction across exposed communities (e.g., DQ5 score of 14 or higher and PFOA: Katherine, PR = 0.75, 95% CI 0.59 to 0.97; Oakey, PR = 0.83, 95% CI 0.58 to 1.18; Williamtown, PR = 1.06, 95% CI 0.76 to 1.48). There were no appreciable changes to our findings in sensitivity analyses (Table A2-9 to Table A2-16, Appendix 2).

Table 9. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10						
PFOS	260 (65)	0.94 (0.81,1.09)	149 (33)	0.79 (0.58,1.07)	280 (70)	0.89 (0.75,1.06)
PFOA	260 (65)	0.83 (0.68,1.02)	149 (33)	0.81 (0.62,1.06)	280 (70)	0.92 (0.73,1.15)
PFHxS	260 (65)	0.98 (0.87,1.11)	149 (33)	0.97 (0.79,1.21)	280 (70)	0.98 (0.85,1.12)
K6 score ≥13						
PFOS	268 (54)	0.94 (0.78,1.13)	156 (31)	0.85 (0.64,1.13)	284 (59)	0.99 (0.83,1.17)#
PFOA	268 (54)	0.71 (0.57,0.89)	156 (31)	0.78 (0.58,1.04)	284 (59)	0.87 (0.66,1.15)#
PFHxS	268 (54)	0.95 (0.82,1.10)	156 (31)	0.90 (0.74,1.11)	284 (59)	0.90 (0.77,1.06)#
DQ5 score ≥14						
PFOS	270 (49)	0.95 (0.78,1.15)	157 (32)	0.74 (0.58,0.95)	284 (50)	1.05 (0.89,1.25)
PFOA	270 (49)	0.75 (0.59,0.97)	157 (32)	0.83 (0.58,1.18)	284 (50)	1.06 (0.76,1.48)
PFHxS	270 (49)	0.94 (0.81,1.09)	157 (32)	0.88 (0.72,1.07)	284 (50)	0.95 (0.83,1.10)
GAD-7 score ≥10						
PFOS	267 (25)	0.85 (0.65,1.10)	157 (16)	0.98 (0.63,1.53)	285 (26)	0.96 (0.72,1.30)#
PFOA	267 (25)	0.72 (0.47,1.11)	157 (16)	0.85 (0.55,1.32)	285 (26)	1.07 (0.61,1.88)
PFHxS	267 (25)	0.83 (0.68,1.01)	157 (16)	0.97 (0.66,1.43)	285 (26)	0.90 (0.70,1.17)#

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval. Sample sizes differ to those in Table 6 because of missing values in confounders.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), and occupational AFFF exposure (yes vs. no).

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Psychological distress and factors that may affect the perceived risk of PFAS exposure

In participants who reported occupational exposure to AFFF, compared to participants who did not report occupational exposure, we observed higher mean scores and higher prevalence of clinically-significant scores on self-reported measures of psychological distress, somatisation, and anxiety in all exposed communities (Table 10 and Table A2-17, Appendix 2). The evidence was strongest in Katherine and Williamtown; for example, we estimated a 3.39 and 6.28 times as high prevalence of clinically-significant self-reported anxiety scores among participants who were occupationally exposed to AFFF than among participants who were not occupationally exposed (GAD-7 score of 10 or higher: Katherine, PR = 3.39, 95% CI 1.75 to 6.60; Williamtown, PR = 6.28, 95% CI 2.89 to 13.65). The evidence was weakest in Oakey, where prevalence ratios were imprecisely estimated and uninformative (e.g., GAD-7 score of 10 or higher: PR = 1.30, 95% CI 0.51 to 3.32).

Table 10. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores for factors that may affect the perceived risk of PFAS exposure in participants from PFAS Management Areas, 2019–2020.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10						
Current (vs. former) residence or work	272 (69)	0.81 (0.49,1.33)	161 (41)	1.14 (0.62,2.08)	287 (72)	0.83 (0.51,1.35)
Per decade of residence	272 (69)	0.92 (0.71,1.19)	161 (41)	0.70 (0.53,0.94)	287 (72)	0.88 (0.72,1.07)
Occupational AFFF exposure	272 (69)	2.40 (1.45,3.97)	161 (41)	1.19 (0.69,2.07)	287 (72)	2.16 (1.36,3.44)
Bore water use	272 (69)	1.38 (0.87,2.17)	161 (41)	2.07 (1.26,3.42)	287 (72)	1.66 (1.00,2.74)
K6 score ≥13						
Current (vs. former) residence or work	280 (58)	0.72 (0.43,1.20)	168 (35)	0.98 (0.49,1.96)	292 (60)	0.53 (0.30,0.93)#
Per decade of residence	280 (58)	1.04 (0.81,1.32)	168 (35)	0.86 (0.64,1.16)	292 (60)	0.97 (0.81,1.17)#
Occupational AFFF exposure	280 (58)	1.84 (0.99,3.40)	168 (35)	1.57 (0.77,3.19)	292 (60)	1.95 (1.18,3.23)#
Bore water use	280 (58)	1.82 (1.13,2.95)	168 (35)	1.12 (0.59,2.12)	292 (60)	1.43 (0.82,2.50)#
DQ5 score ≥14						
Current (vs. former) residence or work	282 (53)	0.59 (0.35,1.00)	169 (35)	0.65 (0.28,1.49)	292 (51)	0.61 (0.32,1.16)
Per decade of residence	282 (53)	1.01 (0.79,1.29)	169 (35)	1.08 (0.80,1.44)	292 (51)	0.90 (0.72,1.13)
Occupational AFFF exposure	282 (53)	2.58 (1.39,4.81)	169 (35)	1.52 (0.76,3.07)	292 (51)	2.98 (1.73,5.11)
Bore water use	282 (53)	1.61 (0.94,2.76)	169 (35)	1.22 (0.63,2.37)	292 (51)	1.41 (0.69,2.87)
GAD-7 score ≥10						
Current (vs. former) residence or work	279 (27)	0.80 (0.38,1.67)	169 (19)	0.73 (0.27,1.96)	293 (27)	0.49 (0.21,1.17)
Per decade of residence	279 (27)	0.93 (0.58,1.48)	169 (19)	0.96 (0.65,1.42)	293 (27)	0.96 (0.72,1.30)
Occupational AFFF exposure	279 (27)	3.39 (1.75,6.60)	169 (19)	1.30 (0.51,3.32)	293 (27)	6.28 (2.89,13.65)
Bore water use	279 (27)	2.16 (0.99,4.68)	169 (19)	1.58 (0.71,3.54)	293 (27)	1.95 (0.72,5.28)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval. Sample sizes differ to those in Table 6 because of missing values.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), and occupational AFFF exposure (yes vs. no).

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Higher prevalence of clinically-significant scores on self-reported measures of psychological distress, somatisation, and anxiety with bore water use were most compatible with our data, under our assumed models, in all exposed communities (Table 10 and Table A2-17, Appendix 2). Prevalence ratio estimates ranged between 1.12 and 2.16; however, in some communities, prevalence ratios were imprecisely estimated and we were not able to determine the direction of the associations with certainty (e.g., GAD-7 score of 10 or higher: Katherine, PR = 2.16, 95% CI 0.99 to 4.68; Oakey, PR = 1.58, 95% CI 0.71 to 3.54; Williamtown, PR = 1.95, 95% CI 0.72 to 5.28).

In contrast, there was very limited evidence for higher prevalence of clinically-significant self-reported psychological distress scores in current residents and workers compared to former residents and workers (e.g., K6 score of 13 or higher: Katherine, PR = 0.72, 95% CI 0.43 to 1.20; Oakey, PR = 0.98, 95% CI 0.49 to 1.96; Williamtown, PR = 0.53, 95% CI 0.30 to 0.93) and per decade of residence in exposed communities (e.g., K6 score of 13 or higher: Katherine, PR = 1.04, 95% CI 0.81 to 1.32; Oakey, PR = 0.86, 95% CI 0.64 to 1.16; Williamtown, PR = 0.97, 95% CI 0.81 to 1.17).

Psychological distress and health concerns

We observed increasing prevalence of clinically-significant scores on self-reported measures of psychological distress, somatisation, and anxiety with increasing participant concerns about physical health and mental health (Table 11). For example, the prevalence of clinically-significant psychological distress scores for participants who were 'moderately', 'very', and 'extremely' concerned about their health was 2.28 (95% CI 1.05 to 4.99), 3.27 (95% CI 1.53 to 7.01), and 5.25 (95% CI 2.47 to 11.15) times as high, respectively, as for participants who were not concerned about their health (DQ5 score of 14 or higher). The number and proportion of participants with each level of health concern are presented below.

Health concerns and health-related behaviours

Between 85% (753/881) and 95% (835/881) of adult participants in the exposed communities responded to the survey questions on concerns related to living or working in a PFAS Management Area in Katherine, Oakey, and Williamtown. Of these participants, 84% (699/835) reported being at least 'slightly' concerned about their health, with 26% (220/835) of participants expressing moderate concerns and 32% (270/835) of participants reporting being 'very' or 'extremely' concerned (Figure 6). The survey provided an opportunity for participants to list specific health concerns in free text; a substantial number of participants (141) recorded that they were concerned about the possible link between PFAS and cancer.

Sixty-five percent (544/832) of participants expressed concern for their own health and over half of the participants also expressed concern regarding the health of their family members, including their partner (52%; 350/668) and children (58%; 358/621). Participants also separately listed that they were concerned about the health of their friends, neighbours, colleagues, and other members of the community (156 participants).

Of the 829 participants who responded to the question on mental health concerns, 54% (450/829) expressed that they were at least 'slightly' concerned, with 16% (129/829) of participants expressing moderate concerns about mental health and 19% (156/829) of participants reporting being 'very' or 'extremely' concerned (Figure 6). Forty-two percent (347/829) of participants also reported at least slight concerns regarding stigma and 68% (562/832) of participants expressed at least slight concerns regarding uncertainty about the future. Less than half of the participants who responded to the remaining questions expressed varying levels of concern regarding finances (48%; 399/825), time costs (37%; 296/810), and work disruption (32%; 240/753). Participants separately listed property devaluation as a concern (66 participants).

Across all three communities, 19% (158/830) of participants reported seeking professional assistance to manage physical or mental health symptoms in relation to living or working in a PFAS Management Area. Participants were most likely to report seeking assistance from a GP or medical

specialist (93%; 147/158), followed by assistance from a psychologist or psychiatrist (25%; 40/158). Smaller proportions of participants reported seeking assistance from a counsellor (16%; 25/158) or a telephone counselling service (4%; 7/158). Across all four sources of physical and mental health assistance that the survey investigated, 71% to 80% of participants who received assistance reported that it was helpful.

Few participants reported changes in health-related behaviours that they attributed to residing or working in a PFAS Management Area; only 2% (16/825) of participants reported having commenced or increased smoking, 3% (22/821) of participants reported commencing or increasing alcohol intake, 3% (25/822) of participants reported using prescription medication for sleep, and 4% (31/820) of participants reported reduced physical activity.

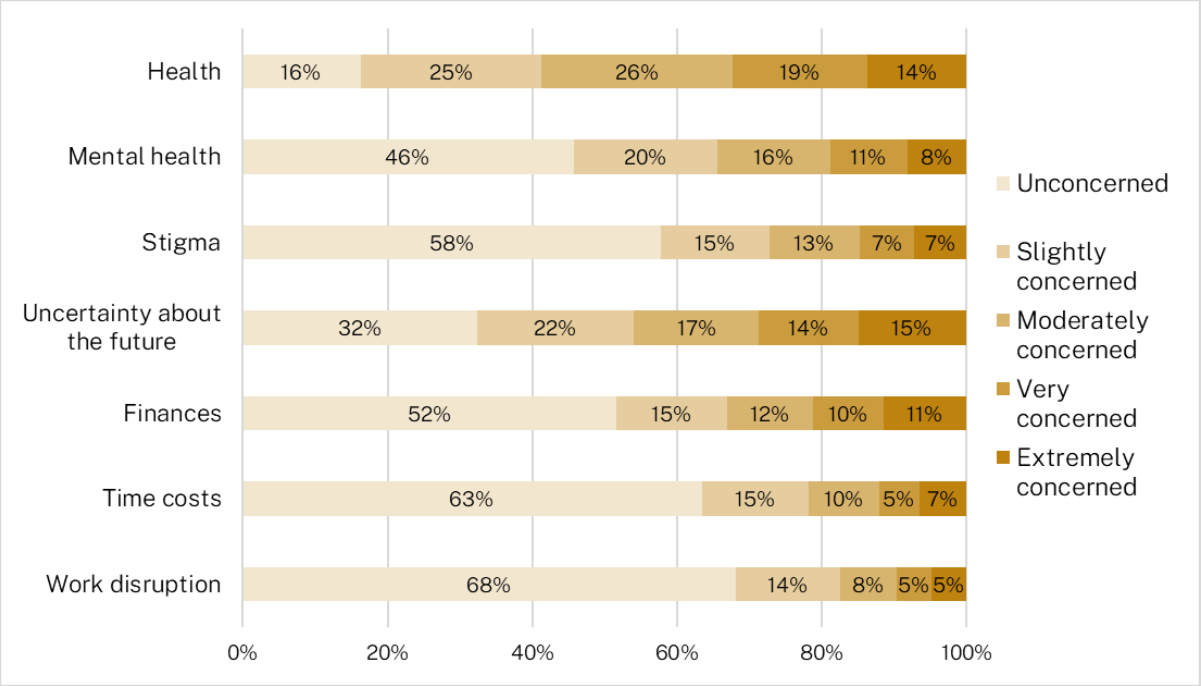
Table 11. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores for increasing levels of health concern, relative to no concern, in participants from PFAS Management Areas, 2019–2020.

	General health concerns		Mental health concerns	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10				
Unconcerned	100 (8)	Reference	287 (35)	Reference
Slightly concerned	169 (28)	2.10 (0.98,4.50)	131 (28)	1.68 (1.08,2.61)
Moderately concerned	155 (44)	3.53 (1.67,7.46)	89 (42)	3.85 (2.65,5.59)
Very concerned	107 (37)	4.44 (2.12,9.31)	55 (25)	3.60 (2.36,5.50)
Extremely concerned	77 (38)	6.09 (2.94,12.64)	42 (22)	4.11 (2.70,6.26)
K6 score ≥13				
Unconcerned	105 (5)	Reference	296 (17)	Reference
Slightly concerned	173 (20)	2.47 (0.93,6.59)	134 (24)	2.53 (1.42,4.51)
Moderately concerned	159 (33)	4.48 (1.73,11.58)	94 (36)	6.16 (3.70,10.26)
Very concerned	113 (35)	6.26 (2.41,16.24)	57 (30)	7.96 (4.67,13.55)
Extremely concerned	81 (40)	9.78 (3.82,25.03)	46 (23)	7.64 (4.41,13.24)
DQ5 score ≥14				
Unconcerned	104 (8)	Reference	296 (20)	Reference
Slightly concerned	174 (15)	1.12 (0.48,2.62)	135 (14)	1.25 (0.64,2.42)
Moderately concerned	160 (29)	2.28 (1.05,4.99)	95 (34)	4.98 (3.03,8.18)
Very concerned	113 (30)	3.27 (1.53,7.01)	57 (25)	5.82 (3.42,9.91)
Extremely concerned	81 (37)	5.25 (2.47,11.15)	46 (23)	6.35 (3.70,10.90)
GAD-7 score ≥10				
Unconcerned	106 (3)	Reference	298 (7)	Reference
Slightly concerned	174 (6)	1.53 (0.32,7.18)	135 (6)	1.30 (0.43,3.94)
Moderately concerned	160 (11)	2.90 (0.67,12.49)	94 (18)	7.48 (3.25,17.22)
Very concerned	114 (19)	6.71 (1.64,27.41)	58 (16)	10.31 (4.38,24.26)
Extremely concerned	79 (26)	12.02 (3.00,48.19)	45 (16)	12.36 (5.32,28.71)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval. Sample sizes differ to those in Table 6 because of missing values.

† Adjusted for age, sex, level of education, and gross household annual income.

Figure 6. Participant health and other concerns in relation to residing or working in a PFAS Management Area, 2019–2020.



Percentages are relative to the number of participants who responded to these questions (i.e., between 85% (753/881) and 96% (835/881) of all adult Cross-sectional Survey participants).

Experiences and perceptions of PFAS blood testing in the VBTP

In total, 92% (812/881) of adult participants from Katherine, Oakey, and Williamtown had their blood tested for PFAS through the VBTP. We asked 89% (724/812) of these adult participants whether they found the PFAS blood test results helpful. Overall, 42% (307/724) of participants reported that the blood test was helpful; however, 15% (109/724) reported that the blood test was not helpful, and 25% (182/724) were unsure. The remainder of participants had not received, or could not recall receiving, their blood test results at the time of the survey (16%; 119/724) or did not respond to the survey question (1%; 7/724). We observed distinct differences in the experiences and perceptions of participants who reported that the blood test was helpful and those who reported the blood test was not helpful or who were unsure. A summary of the experiences and perceptions of the VBTP for exposed participants from Katherine, Oakey, and Williamtown is shown in Figure 7.

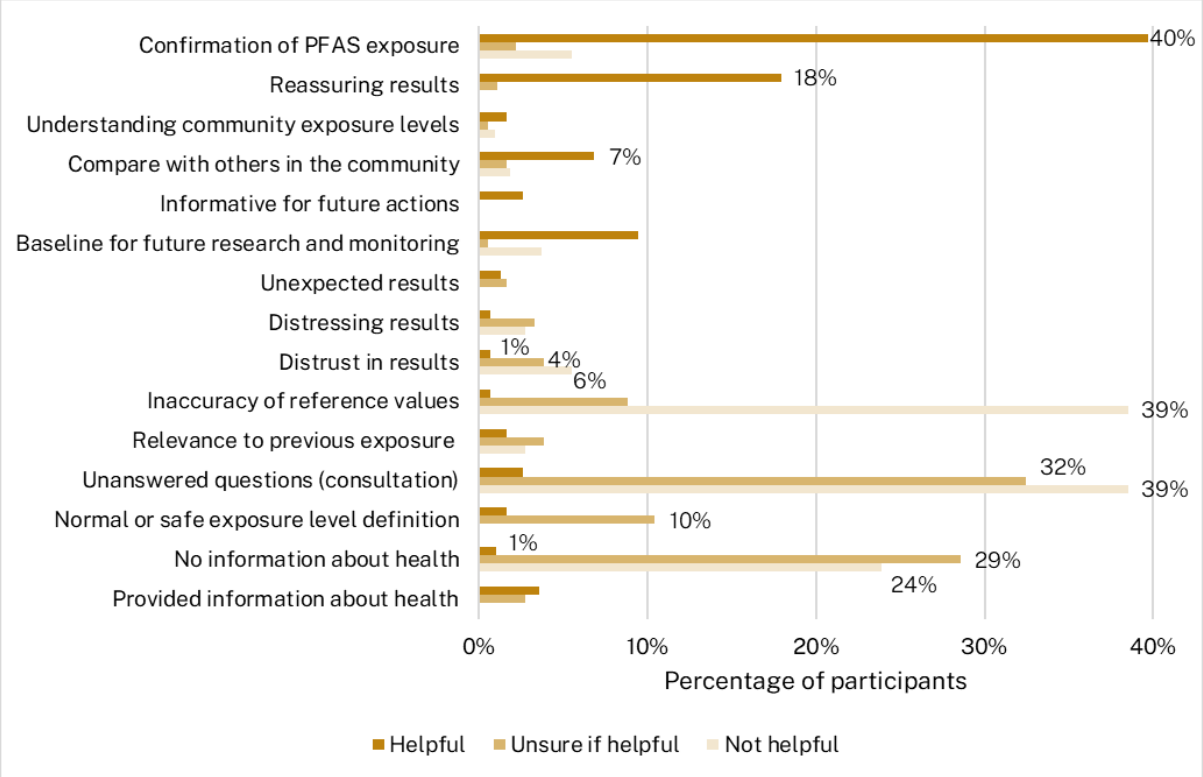
Of the participants who found the blood test helpful, 40% (122/307) described the test as informative and a confirmation of their exposure to PFAS, and 18% (55/307) found the results reassuring. One participant stated:

> *'[The results] eased my mind about long term contamination.'*

However, of these participants, some considered a blood test result within the estimated Australian reference values (based on the estimated 95th percentiles for PFOA and PFOS in the Australian population, 2011–2012)[85] to be 'safe':

> *'The test provided peace of mind that my levels were within the acceptable 'safe' range.'*

Figure 7. Participant experiences and perceptions of the VBTP in PFAS Management Areas, 2019.



Participant experiences and perceptions of the VBTP (*y*-axis) grouped by whether they found the blood test helpful, not helpful, or were unsure. Percentages are relative to the number of participants in each group (307 participants reported that the blood test was helpful, 182 participants were unsure, and 109 reported that the blood test was not helpful). Participants could report more than one experience or perception, so the sum of the percentages in each group is not necessarily 100%.

In addition, 7% (21/307) of participants who reported that the blood test was helpful described how they used the results to compare their exposure to the exposure of others in their community, including their children and other members of their family. In comparing their exposure to others, participants also considered the differences in exposure pathways, such as bore water ingestion and local produce consumption. One participant stated:

> *'I understand that my results were relatively low, I think mostly because I drank rainwater most of the time, and rarely drank bore water. I feel it can be a useful comparison when looking at other results from people who have mostly or always drank [bore water].'*

In contrast, 39% (42/109) of the participants who found that the blood test was not helpful had unanswered questions, primarily in relation to their post-test consultation with their GP. These participants indicated that they had questions about the meaning of their results, referring to the scientific language used to explain the results, or in some cases, the lack of an explanation from their GP. One participant referred to the results as *'too ambiguous for a non-clinical person'*. Similarly, 32% (59/182) of participants who reported that they were unsure if the test was helpful described a similar experience at their post-test consultation. Participants stated:

> *'I didn't know much about PFAS at the time. The results were hard to decipher and it was difficult to get the results. The whole process was not very good or informative.'*
>
> *'My GP [had] little understanding of this testing and results.'*

Participants referred to the uncertainty in the reference values used to compare exposure levels between community members and the general Australian population, as well as to the uncertainty in the health effects and long-term outcomes. Of the participants who reported that the blood test

was not helpful, 39% (42/109) referred to the estimated Australian reference values, questioning the relevance and meaning of the comparison to the general population and pointing to the differences between Australian and international reference values. One participant stated:

> *'The blood tests provided a baseline at a moment in time. There was little information available at the time that defines reasonable limits and what is considered abnormal results.'*

Further, 10% (19/182) of participants who were unsure whether the test was helpful differentiated between the reference values and a 'safe' level of PFAS exposure, referring to the insufficient information available on harmful exposure levels. Related to this, 29% (52/182) of this group of participants recalled that the blood test results were not meaningful in understanding their health, either currently or in the future. We observed the same experience in 24% (26/109) of participants who found that the blood test was not helpful, but only in 1% (3/307) of participants who found the test helpful. Participants stated:

> *'No one is aware of what blood concentration levels may cause health issues.'*
>
> *'Not enough is known about the long-term effects of PFAS to make any meaningful assessment of the blood test results.'*

In contrast, several participants (2%; 16/724) drew conclusions about their current or future health status, including disease diagnoses, in relation to their test results. However, participants did not describe how they came to these conclusions. No participants reported drawing conclusions on their health based on feedback at the post-test consultation with their GP.

Across all categories of experiences, 2% (15/724) of participants described distress in relation to their blood test results, and none of these participants reported that the test was helpful. Further, no participants who reported the test was unhelpful were reassured by their results, and 6% (6/109) described their distrust of the results, referring to the differences between testing facilities and between the results of people who had received multiple tests. In addition, 4% (7/182) of participants who were unsure whether the test was helpful reported distrust in the results. In contrast, less than 1% (2/307) of participants who reported that the test was helpful expressed distrust of their results, highlighting differences in participant experiences and perceptions of testing. However, these differences were not necessarily reflected in the blood serum PFAS concentrations of participants across the categories, relative to the reference values used in the reporting of results. For participants who reported that the test was helpful, 9% (28/307) had blood serum PFOS or PFOA concentrations above the reference values, compared to 6% (7/109) of participants who reported that the test was not helpful and 4% (7/182) who were unsure.

Overall, participants also highlighted the additional contributions of the VBTP, referring to contributions to their community, research, and future government decisions. A small number of participants stated that the test results were a justification for following the current precautions and restrictions in their community (less than 3% of participants who reported that the test was helpful, and less than 1% overall). Further, across all categories of experiences, 5% (34/724) described the value of the test as a baseline of PFAS exposure to compare with future tests. Participants stated:

> *'I am interested in the results purely for the purpose of the long term survey and possible future prevention.'*
>
> '*It gave me and my family a baseline result. We are now able to get follow up tests and determine if we are still being contaminated while living here and following [government] guidelines.'*

Discussion

In the Cross-sectional Survey, we compared the prevalence of self-reported health and psychological distress outcomes between participants of the PFAS Management Areas in Katherine, Oakey, and Williamtown and the comparison communities of Alice Springs, Dalby, and Kiama and Shellharbour. We examined the association between blood serum concentrations of three PFAS chemicals and self-reported physical and mental health outcomes. In the exposed communities, we also assessed participant health and other concerns in relation to residing or working in PFAS Management Areas and participant experiences and perceptions of the PFAS blood testing in the VBTP. We focused on health outcomes in adults due to low child participation rates in the survey. A summary of findings from the analyses of self-reported physical and mental health outcomes is presented in Box 3.

Self-reported health

We observed higher sample prevalence of several health outcomes in some exposed communities compared to comparison communities, including cancer and liver disease in Katherine compared to Alice Springs, and rheumatoid arthritis, hypercholesterolaemia, type II diabetes, and fertility problems in Williamtown compared to Kiama and Shellharbour. While this provides insight into the differences between the communities, sample prevalence may differ for reasons not related to PFAS, including self-selection bias, differences in the exposed and comparison communities with regard to non-PFAS risk factors for the health outcomes, and the use of self-reported measures of health.

We also assessed relationships between self-reported health outcomes and PFAS concentrations in blood serum. While few of our observations were consistent across communities and specific PFAS, with higher PFAS serum concentrations, we observed: lower prevalence of cancer and rheumatoid arthritis; higher prevalence of hypercholesterolaemia; associations in opposing directions for different PFAS for kidney disease and hypothyroidism; and no clear associations for cardiovascular disease, liver disease, or reproductive outcomes.

Interpretation of the findings in the context of previous research

Cancer

The cross-sectional survey asked participants about their lifetime history of diagnosis with any of 10 cancers. Very few cases of cancer were reported by participants of the exposed communities (between 0 and 15 cases of any given cancer) and some of the cases may have occurred prior to living or working in the exposed communities. Based on these few cases, we observed decreased prevalence of breast cancer in Katherine and decreased prevalence of prostate cancer and any cancer combined in Oakey with higher PFOS and PFHxS serum concentrations.

These cross-sectional associations were in the opposite direction to what we would expect to observe if PFAS plays a role in cancer development, and should not be considered as evidence of a protective effect of PFAS on cancer risk. The direction of the associations may have been due to survivor bias, the ‘healthy worker effect’ (i.e., participants with higher occupational exposure may have been required to meet physical fitness and health criteria to be employed in their occupation), and/or self-selection bias (e.g., individuals with cancer who were concerned about their exposure to firefighting foam may have been more likely to participate in the survey; however, it is also possible that individuals with a history of cancer may have engaged in health-promoting behaviours to avoid exposure since their diagnosis).

We were not able to identify any studies of breast or prostate cancer prevalence related to PFAS exposure in the literature. While our cross-sectional analysis cannot provide evidence for or against a causal hypothesis, our findings are consistent with two studies that reported a negative association between PFAS exposure and breast cancer incidence.[86,87] However, case-control and

cohort studies assessing breast cancer incidence have predominantly reported no associations[34] in communities with PFAS-contaminated water[87,88] or in occupational studies.[89,90]

In the case of prostate cancer incidence, case-control and cohort studies have found no associations for PFAS exposure at 'background' levels in the general population,[91,92] in communities exposed to PFAS-contaminated drinking water,[87,88] or in occupational studies.[90,93] For further discussion, we refer the reader to the comprehensive assessment of cancer outcomes in the Data Linkage Study.[66]

Box 3. Summary of findings of self-reported physical and mental health outcomes in the Cross-sectional Survey.

Outcome	Summary of findings
Self-reported health outcomes	
Cancer	Very few cancer cases were reported in the Cross-sectional Survey. Self-reported cancer was more common in Katherine than Alice Springs. However, there was no clear evidence for positive associations between PFAS concentrations in blood serum and cancer prevalence in any exposed community.
Cardiovascular outcomes	Self-reported hypercholesterolaemia was more common in Williamtown than Kiama and Shellharbour. We observed small positive cross-sectional associations between PFOA concentrations in blood serum and self-reported hypercholesterolaemia in Katherine, but not with PFOS or PFHxS concentrations. PFAS concentrations in blood serum were not clearly associated with increased prevalence of self-reported heart attacks, high blood pressure, and stroke.
Liver disease	Self-reported liver disease was more common in Katherine than Alice Springs. However, we had no clear evidence of associations between PFAS serum concentrations and prevalence of self-reported liver disease in any exposed community.
Kidney disease	We had evidence of positive cross-sectional associations between PFOA serum concentrations and self-reported gout and any kidney disease among participants in Katherine and Williamtown and limited evidence of inverse cross-sectional associations for PFOS and PFHxS.
Autoimmune disease	Self-reported rheumatoid arthritis was more common in Williamtown than Kiama and Shellharbour. However, we had some limited evidence of lower, rather than higher, prevalence of self-reported rheumatoid arthritis and any autoimmune disease per doubling in PFAS serum concentrations. We observed no meaningful difference in self-reported asthma prevalence per doubling in PFAS serum concentrations.
Endocrine outcomes	Self-reported type II diabetes was more common in Williamtown than Kiama and Shellharbour. However, PFAS serum concentrations were not clearly associated with self-reported type II diabetes, hypothyroidism, or hyperthyroidism. There was some evidence of positive associations for hypothyroidism with PFOA and inverse associations with PFHxS in Katherine, but these observations were based on few cases of hypothyroidism.
Reproductive outcomes	Self-reported problems with fertility were more common in Williamtown than Kiama and Shellharbour. However, PFAS serum concentrations were not clearly associated with self-reported problems with fertility or with early-onset menopause.
Psychological distress outcomes	
Psychological distress (K6, DQ5), somatisation (PHQ-15), and anxiety (GAD-7)	We observed substantially higher self-reported psychological distress in all exposed communities than comparison communities. However, there was no clear evidence for a positive association between PFAS concentrations in blood serum and psychological distress. Psychological distress was higher in participants who reported occupational exposure to AFFF, bore water use on their properties, or concerns about their health.

Cardiovascular outcomes

We observed a small positive cross-sectional association between PFOA and self-reported hypercholesterolaemia in Katherine. PFAS serum concentrations were also associated with elevated cholesterol levels in the Blood Serum Study.[65] While we cannot use our observed cross-sectional associations to infer causation, our findings are consistent with a substantial body of evidence suggesting that exposure to PFAS is associated with abnormal lipid profiles, particularly elevated total cholesterol levels.[14,27,30,94] Both cross-sectional and cohort studies have reported positive associations between PFOA exposure and elevated cholesterol levels: in a general population at 'background' exposure levels,[95] in communities exposed to PFAS-contaminated water,[15,96,97] and in occupational studies.[98-100] Mechanistic studies have demonstrated that PFAS have the ability to perturb cholesterol homeostasis; for example, by affecting the expression of genes involved in cholesterol transport.[30,94,101] However, uncontrolled confounding by diet and the enterohepatic cycling process of PFAS and bile acids have also been proposed as explanations for the positive associations reported in epidemiological studies.[30]

Although hypercholesterolaemia is an established risk factor for cardiovascular disease, PFAS serum concentrations were not clearly associated with increased prevalence of self-reported cardiovascular outcomes (including heart attack, high blood pressure, and stroke) in our study. The evidence for relationships between PFAS and specific cardiovascular diseases has been inconclusive.[14,30] Cross-sectional associations of PFAS with hypertension and cardiovascular disease prevalence have been reported in a representative sample of the United States (US) population in the National Health and Nutrition Examination Survey (NHANES).[102-104] However, the findings of prospective cohort studies and case-control studies of disease incidence, which offer better-quality evidence, have been inconsistent.[14] For example, studies of Italian and US communities exposed to PFAS-contaminated drinking water have reported both positive associations[105] and no associations[97] with the risk of hypertension and cardiovascular disease. Mortality due to coronary heart disease was also assessed in the Data Linkage Study.[66]

Liver disease

We observed no cross-sectional associations between PFAS serum concentrations and prevalence of self-reported history of liver disease in any exposed community. PFAS serum concentrations were also not consistently associated with biochemical markers of liver function in the Blood Serum Study.[65] Mechanistic studies suggest that PFAS exposure may contribute to the development and progression of non-alcoholic fatty liver disease and toxicant-associated fatty liver disease.[106] Despite this, epidemiological studies have inconsistently linked PFAS exposure to biomarkers of liver function and there is a paucity of studies that link PFAS directly to clinically diagnosed liver disease.[27,94] Positive associations have been primarily reported for PFOA and PFOS with elevated liver enzymes, such as alanine aminotransferase and alkaline phosphatase, in population-based cross-sectional studies and cohort studies in the USA,[107] China,[108] and Sweden,[109] in cross-sectional studies of communities exposed to PFAS-contaminated water,[110,111] and in occupational studies.[98] The findings have been inconsistent across specific PFAS and liver enzymes, and lack of associations have also been reported.[98,109-112] However, in the absence of clinical symptoms, biomarker values outside of reference intervals are not necessarily indicative of disease. Among studies that directly assessed liver disease, PFAS have been associated with mortality due to cirrhosis of the liver in one of two cohort studies of highly exposed workers,[113,114] but our findings are consistent with two cross-sectional studies of communities with PFAS-contaminated water that reported no associations with liver disease.[110,115] Liver disease mortality was also assessed in the Data Linkage Study.[66]

Kidney disease

We observed positive cross-sectional associations between PFOA exposure and self-reported history of gout among exposed participants in Katherine and Williamtown, but inverse cross-sectional associations between both PFOS and PFHxS exposure and history of gout in

Williamtown. No material associations were observed for gout in Oakey. We also analysed the combined outcome 'any kidney disease', which includes participants who have gout or chronic kidney disease, and observed a similar pattern of associations, which were probably driven by the relatively high prevalence of gout compared with chronic kidney disease among survey participants. In our Blood Serum Study,[65] PFAS were not associated with eGFR, a measure of kidney function, but PFOA and PFOS were positively associated with elevated uric acid levels, which is an established risk factor for the development of gout.

The associations observed for PFOS and PFHxS in the Cross-sectional Survey were in the opposite direction to what we would expect to see if increasing exposure to PFAS adversely affects kidney function. Our findings may be explained by the potential for: non-linear relationships between PFAS serum concentrations and kidney function,[79] reverse causation in cross-sectional analyses of PFAS and kidney disease,[79] medical treatments for gout and kidney disease that impact PFAS serum concentrations (measured after disease onset), and differential rates of excretion across PFAS chemicals.[23,24] PFAS are thought to be excreted at diminished rates in individuals with mild to moderate loss of kidney function, and at enhanced rates in cases of severe loss of kidney function.[79] Further, when kidney function is compromised, a build-up of uric acid can promote the development of gout. Adjusting for the eGFR, a measure of kidney function, did not attenuate all of the observed effects; however, we were not able to stratify our analysis by stages of kidney function decline.[79]

Studies assessing the relationships between PFAS exposure and kidney disease have predominantly focused on biomarkers of kidney function; few studies have assessed gout and kidney disease risk and prevalence. Consistent with our study, Scinicariello and colleagues (2020), in a large representative sample of the US population in the NHANES, reported positive cross-sectional associations between PFOA and odds of self-reported gout, but they reported positive, rather than negative, associations between PFHxS exposure and odds of self-reported gout.[116] PFOA and PFOS exposure have also been positively associated with chronic kidney disease prevalence in the NHANES,[117,118] but PFOA was not associated with chronic kidney disease risk at higher exposure levels in a large retrospective cohort that was part of the C8 Health Project.[119] We also assessed mortality due to chronic kidney disease in the Data Linkage Study.[66]

Autoimmune outcomes

PFAS serum concentrations were not clearly associated with self-reported autoimmune conditions in the Cross-sectional Survey. We had some limited evidence of lower prevalence of rheumatoid arthritis and any autoimmune disease with PFOA serum concentrations, which were at similar levels in the exposed and comparison communities (see Blood Serum Study).[65] While cross-sectional associations cannot be used to support or refute causation, the observed associations for PFOA were in the opposite direction to what we would expect to see if PFOA were to adversely affect autoimmune disease prevalence. In the C8 Health Project, a large community cohort exposed to PFAS-contaminated drinking water at higher levels than observed in our Blood Serum Study,[65] Steenland and colleagues (2013) reported no associations between model-based PFOA exposure estimates and odds of self-reported lifetime history of Crohn's disease, lupus, multiple sclerosis, or rheumatoid arthritis.[120] However, in an occupational subcohort of the C8 Health Project, with higher PFOA exposure levels, Steenland and colleagues (2015) separately reported positive associations between PFOA exposure, ulcerative colitis, and rheumatoid arthritis, and an inverse association with adult asthma.[93]

Endocrine outcomes

PFAS serum concentrations were not clearly associated with the self-reported lifetime history of type II diabetes. Evidence for associations between PFAS exposure and type II diabetes have been inconclusive.[27,121] Consistent with our observation, studies assessing relationships between PFAS exposure and type II diabetes have reported no cross-sectional associations[122,123] and no associations in a retrospective cohort study.[124] Several studies have reported positive cross-

sectional associations with exposure to PFOS[125] and PFOA,[126] as well as positive associations in prospective studies of US women[127] and highly exposed workers.[93] However, inverse associations[125,128] and non-linear associations[129] have also been reported. The evidence has also been inconclusive among studies assessing markers of diabetes risk, including blood insulin and fasting glucose levels.[27,121]

Higher PFOA serum concentrations were associated with higher prevalence of self-reported hypothyroidism, and higher PFHxS serum concentrations were associated with lower prevalence of self-reported hypothyroidism in Katherine. However, this pattern of associations was not observed in Oakey and Williamtown, was based on only nine cases of hypothyroidism, and the associations were attenuated when we imputed missing data. PFAS serum concentrations were also not clearly associated with self-reported hyperthyroidism in this study or with serum concentrations of thyroid-stimulating hormone and thyroid hormones in the Blood Serum Study.[65]

Experimental studies in cells and animals have demonstrated that PFAS can disrupt thyroid homeostasis.[130,131] Epidemiological studies have widely reported associations with thyroid stimulating hormone and thyroid hormone concentrations,[131-134] but few studies have assessed relationships between PFAS exposure and thyroid disease prevalence or incidence.[27] While our observation for PFOA in Katherine was probably due to small case numbers, PFOA was also positively associated with thyroid disease in a cross-sectional analysis of the US population in the NHANES[135] and in women, but not men, in the C8 Health Project;[136] however, PFOA was not associated with thyroid disease at higher exposure levels in an occupational cohort study in West Virginia.[93] Consistent with our finding, PFOS was not associated with thyroid disease in the NHANES[135] and no associations were reported in a large Swedish population exposed to PFOS- and PFHxS-contaminated water.[137] The differences in findings of cross-sectional studies may be explained by reverse causation, as renal function may be compromised in individuals with thyroid dysfunction,[138] which in turn may affect PFAS excretion.[139]

Reproductive outcomes

PFAS serum concentrations were not clearly associated with self-reported problems with fertility in the Cross-sectional Survey. Our analysis was limited to a non-specific measure of infertility that was not validated and was not sex-specific, even though environmental chemicals operate on male and female fertility through different mechanisms. While our analysis was cross-sectional and cannot provide evidence for or against a causal relationship between PFAS and infertility, a recent Australian study also reported no associations between PFAS exposure, measured in the follicular fluid of women undergoing *in vitro* fertilisation, and fertilisation rates.[140] In contrast, several cohort studies have reported positive associations between PFAS and infertility in women, defined as time to pregnancy greater than 12 months or treatment for infertility, but associations have been inconsistent for specific PFAS.[141-144] Positive associations have also been reported between PFAS and infertility due to endometriosis,[145] polycystic ovarian syndrome,[146] and premature ovarian insufficiency.[102] However, PFAS have been inconsistently associated with delayed time-to-pregnancy in females, which may be explained by differences in treatment of parity and inter-pregnancy intervals in analyses.[35,147] Few studies of PFAS exposure in men and couple fecundability (i.e., the ability to conceive a pregnancy) have been conducted[147] and PFAS have not been consistently associated with measures of semen quality.[148]

PFAS serum concentrations were also not clearly associated with early onset menopause in our study, which was based on self-reported dates of menopause onset that may be subject to recall bias. In contrast with our findings, PFAS serum concentrations have been associated with early menopause in a representative sample of the US population in the NHANES study[149] and in a large cohort of women from communities with PFAS-contaminated water supplies in the C8 Health Project in West Virginia.[150] However, these associations are thought to be due to reverse causation;[36,151,152] blood loss during menstruation is an elimination pathway for PFAS, so it is possible that women who experienced early menopause have higher PFAS serum concentrations.

Strengths and limitations

Strengths of our study included the use of an objective measure of PFAS exposure (i.e., PFAS concentrations measured in blood serum). A key limitation was our use of self-reported measures of health. Further limitations that are common to analyses of cross-sectional data may have affected our results, including the uncertain temporal relationship between exposure and outcome occurrence, selection bias, misclassification of outcome, exposure, and confounder variables, and residual confounding. These limitations are explained below.

Temporality

Cross-sectional surveys measure both health outcomes and exposures at the same point in time. We related self-reported lifetime history of particular health conditions to PFAS concentrations in serum measured at the time of blood collection in the Blood Serum Study.[65] Exposure measurement, therefore, occurred after disease onset, and we were not able to take into account whether participants were exposed to PFAS before or after disease onset, which precludes the use of our results alone to assess causation.

Selection bias

Community members chose whether to participate in the Cross-sectional Survey. The sample of participants in exposed communities was, therefore, not randomly selected but was 'self-selected'. Self-selection may bias effect estimates (e.g., prevalence ratios may be under- or overestimated). For example, if participants with health conditions were more likely to respond to the survey in exposed communities than in comparison communities, our prevalence ratio estimates may be artificially inflated (i.e., a disease may incorrectly appear to be more common in an exposed community than it is). Likewise, in the exposed communities, awareness of exposure status (e.g., due to occupational use of firefighting foam or bore water consumption) and the perception that PFAS adversely affects health may have influenced participation in the Cross-sectional Survey.

Outcome, exposure, and confounder misclassification

Outcome misclassification (where a person is considered to have a disease when they do not, or vice versa) is a possibility as outcome assessment was based on self-reported information that was not validated using medical records. Recall of potentially confounding variables may have been biased; that is, participants diagnosed with a disease may have better recall of the information related to the risk factors of that disease.

Exposure measurement error may also have affected our findings. The patterns of exposure to PFAS in participants varied depending on their movement in and out of exposed communities over time. We measured exposure at a single time point, which does not reflect variation in PFAS serum concentrations over time, is an imperfect measure of cumulative exposure levels, and may not reflect exposure levels at pertinent times for disease development. While PFAS have biological half-lives of several years, serum concentrations of PFOA, PFOS, and PFHxS have been decreasing in the Australian population since 2002.[2] Thus, in sensitivity analyses we restricted our sample to participants who resided in the exposed communities in the 5, 10, and 15 years prior to the survey.

Control for confounding

We assessed the sensitivity of our results to assumptions on the relationships between the outcomes, exposures, and confounders. However, our findings may be explained by residual confounding due to the coarse resolution of some confounder data.

Statistical limitations

We performed numerous analyses without correcting for multiple testing, therefore some of the associations that we observed may have been due to chance. Our sample size was small, which limited the statistical power that we had to detect associations. In addition to low power, our

analysis may have been affected by sparse data bias in analyses of outcomes with low prevalence (i.e., bias due to small numbers of participants with an outcome).

Psychological distress and participant concerns

We found substantially higher levels of self-reported psychological distress, somatisation, and anxiety in exposed communities than in comparison communities; however, we found limited evidence to suggest that psychological distress was associated with PFAS serum concentrations in the exposed communities. Instead, we found higher self-reported psychological distress among participants who were occupationally exposed to AFFF and, to a lesser extent, among participants who used bore water on their properties. We also found an increasing trend in the prevalence of self-reported psychological distress, somatisation, and anxiety with increasing participant concerns about physical health and mental health. Our findings suggest that the perception of risks to health, rather than direct PFAS exposure, contributed to psychological distress in the exposed communities.

Interpretation of the findings in the context of previous research

Direct effects of PFAS exposure on psychological distress have seldom been studied. Experimental studies have suggested a potential impact of PFAS exposure on neurotransmitters such as dopamine;[153,154] however, this has not been confirmed in human studies. Inverse cross-sectional associations were reported for depressive symptoms in adults and exposure to PFOA and PFHxS, but not PFOS, in a representative sample of the US population in the NHANES.[155] However, no associations were reported between depressive symptoms and PFAS in prospective cohort studies of sensitive subgroups, including mothers followed for eight years postpartum[156] and children and young adults.[157] Another mechanism for direct effects of PFAS on psychological health is through impacts on thyroid function;[131,133,158] however, our analyses of self-reported hypo- and hyperthyroidism diagnosis in this study, and thyroid function biomarkers in the Blood Serum Study,[65] gave limited evidence of associations with thyroid function.

Beyond the hypothetical direct effects of PFAS on psychological health, the experience of residing in a contaminated area is a documented risk factor for poor psychological health.[81,159,160] A recent systematic review and meta-analysis of the impact of chronic environmental contamination in communities, not limited to PFAS, on psychological health, reported “robust” effects on anxiety, stress, and depression.[159] The uncertainty surrounding the health effects of the contamination and individual health concerns, whether real or perceived, have been identified as major stressors.[81,159,161,162] In contrast with acute environmental disasters, which follow more defined stages from warning and threat through to eventual recovery and rehabilitation, individuals in chronically contaminated areas are thought to be caught in a perpetual state of warning and threat.[81] The prolonged and uncertain effects on physical health are thought to immeasurably tax coping resources and amplify the effects of other stressors, including those not related to the contamination.[81] In support of this, we found that the perceived risk of ever having been exposed to PFAS, rather than current residence or length of time residing in an exposed community, was associated with psychological distress outcomes.

Other than concerns about the health effects of exposure, potential stressors include concerns about financial impacts through loss of livelihood and property devaluation, the role of the media, and social stigma.[81,159,161] Vulnerable community members may be disproportionately affected, such as individuals with lower socio-economic status, the parents of young children, and individuals with pre-existing mental or physical health conditions.[81,159,161] These stressors were evident both in our Focus Groups Study[6] and in our assessment of health and other concerns among exposed participants in the Cross-sectional Survey. A substantial proportion of participants reported concerns about their general health and mental health, and with increasing concerns we found steeply increasing prevalence of self-reported psychological distress, somatisation, and anxiety

at levels that may be of clinical significance. Individuals in exposed communities also reported concern about the health of their partner and children, and concern about their finances and social stigma. Psychological responses to environmental contamination are likely to have complex interactions with perceived risk, health status, financial, social and relational impacts.

Stressors at the community level that arise due to social and cultural responses to the contamination are also thought to contribute to individual psychological distress.[81,159] Psychosocial stress may be promoted by 'institutional delegitimisation';[159] that is, the perceived minimisation of the potential impacts of environmental contamination by government and health professionals,[159,163] and the potential for dismissal of community health concerns as somatic or attributable to lifestyle by health professionals, in the absence of definitive scientific evidence of the health effects of the contaminants.[159,163] These social risk factors were evident in our assessment of community concerns in the focus groups, which identified community disillusionment with the initial government response in the exposed communities.[6]

Strengths and limitations

Strengths of our analysis of psychological distress outcomes included the survey of both exposed and comparison communities, and the concurrent assessment of both the direct effects of PFAS and factors that may affect the perceived risk of PFAS exposure on psychological distress.

The same limitations described in the section on self-reported health apply to the analysis of psychological distress, including the cross-sectional nature of the study, selection bias, outcome, exposure, and confounder misclassification, residual confounding, and multiple testing. In particular, self-selected participation in the survey may have artificially inflated our effect estimates, as individuals with greater distress and concern about their health may have been more likely to respond to the survey.

Outcome misclassification is a possibility. It was not feasible in the context of a large epidemiological study to administer a clinical diagnostic interview to participants. Meeting clinical criteria on a self-report measure is not the same as having a clinical diagnosis. Although we used well-validated mental health measures, it is possible that the use of cut-points for self-report measures of mental health may have misclassified individuals, with the possibility of false positives (participants identified with clinical symptoms who would not have met diagnostic criteria for a mental disorder) and false negatives (individuals identified as not meeting criteria who have a mental disorder). In addition, differences in mental health scores may be statistically significant but not necessarily reflect clinical states or meaningful differences in the severity of symptoms.

An important limitation is the potential for the observed cross-sectional associations to be due to reverse causation. For example, pre-existing mental health symptoms may increase health concerns. It is also important to note that factors such as use of mental health services, psychological treatments and psychotropic medications were not accounted for in analyses. A further limitation is that we were not able to assess the effects of stress-toxicant interactions on health due to the small sample size.[160]

Our comparison communities responded to the survey during the COVID-19 pandemic, which may have increased psychological distress in those communities. Nevertheless, our comparison of psychological health outcomes between exposed and comparison communities revealed higher levels of psychological distress, somatisation, and anxiety in the exposed communities.

PFAS blood test experiences and perceptions

There were thematic differences in the experiences and perceptions of the blood testing provided through the VBTP between participants who found the blood test helpful and those who did not find the test helpful or who were unsure. Participants who reported that the blood testing was helpful communicated that the results confirmed their exposure to PFAS, allowed them to compare their exposure levels with those of other community members and to reflect on potential exposure

pathways. However, some participants assumed that the test results could be used to make inferences to their current and future health or that levels within reference values can be considered 'safe'. In contrast, participants who indicated that the test was not helpful, or who were unsure, reflected on the uncertainty in scientific knowledge regarding the relationships between exposure at different levels and potential health effects. These participants were more likely to express that they had unanswered questions about their results after their post-test consultation. This may indicate that the training provided to health practitioners was not sufficient to ensure post-test consultations were informative, or it may reflect the lack of available evidence for informing clinical action.

Participants also reported that the VBTP was beneficial in setting a baseline for PFAS exposure levels within the community, which could be used in future assessments of the effectiveness of measures intended to limit exposure. This was also a recommendation of the Inquiry into the management of PFAS contamination in and around Defence bases in November 2018,[164] and is the focus of an ongoing longitudinal study that is being conducted by the Queensland Alliance for Environmental Health Sciences at the University of Queensland (funded by the Australian Government National Health and Medical Research Council).

Summary and conclusions

We conducted a cross-sectional survey of individuals who participated in the VBTP in the PFAS Management Areas in Katherine, Oakey, and Williamtown, and individuals in the comparison communities in Alice Springs, Dalby, and Kiama and Shellharbour. We evaluated the effects of PFAS on physical and mental health, and concurrently assessed participant health concerns and other concerns related to living or working in a PFAS Management Area and participant experiences of the PFAS blood testing in the VBTP.

We found that the prevalence of several self-reported health outcomes was higher in PFAS Management Areas than comparison communities, including cancer and liver disease in Katherine compared to Alice Springs, and rheumatoid arthritis, hypercholesterolaemia, type II diabetes, and fertility problems in Williamtown compared to Kiama and Shellharbour. However, PFAS concentrations in blood serum were not clearly associated with most self-reported health conditions. Positive associations were observed for gout, hypercholesterolaemia, and hypothyroidism with blood serum concentrations of PFOA, which is a relatively minor component of the firefighting foams used in Australia, whereas small inverse associations were observed for cancer, gout and kidney disease, and hypothyroidism with blood serum concentrations of PFOS and/or PFHxS, which are the PFAS of highest average concentration in the blood serum of exposed participants in the Blood Serum Study.[65]

In contrast with our observations for self-reported physical health, we observed substantially higher prevalence of self-reported psychological distress in exposed communities compared to the comparison communities. When we examined factors contributing to psychological distress in the exposed communities, our findings suggested that the perception of the risks of PFAS to health, rather than direct PFAS exposure, contributed to psychological distress. Our findings for psychological distress are consistent with the literature on the mental health effects of chronic environmental contamination and highlight the need for government initiatives to support communities exposed to environmental contamination.

In the Cross-sectional Survey, we collected data on a broad range of health outcomes, and surveyed participants in both exposed and comparison communities. This allowed us to compare the prevalence of health outcomes between communities as well as to consider consistency in effects across exposed communities. However, our study had several limitations; notably, the potential for self-selection bias, outcome misclassification and recall bias due to self-report, statistical limitations due to small sample size and multiple comparisons, and the potential for cross-sectional associations to be subject to reverse causation. While our cross-sectional

observations elucidate differences in health between the communities, they cannot be used to infer cause and may not be generalisable to the broader populations of Katherine, Oakey, and Williamtown. Nevertheless, our findings add to the evidence on the health effects of PFAS exposure and psychological effects of chronic environmental contamination in communities exposed to PFAS-contaminated land and water.

Glossary

Adjustment — the modification of an estimate to account for potential confounders (see *confounding*).

Aqueous Film-Forming Foam (firefighting foams) — a highly effective flame suppressing foam, commonly used in the aviation industry to extinguish aircraft fires.

Association — a relationship between two variables. A *positive association* is where the value of one variable tends to increase as the value of another variable increases. An *inverse association* is where the value of one variable tends to decrease as the value of another variable increases. A *null association* is where there is no relationship between two variables.

Bias — any systematic error that results in an incorrect effect estimate (see *effect estimate*).

Causal relationship — where one variable (for example, exposure) causes another (for example, a health outcome). As opposed to 'association', where one variable is related to, but does not necessarily cause, the other.

Chance/random error — some study results may reflect a true effect; however, some results can arise simply because of chance (randomness).

Comparison communities — specific communities in NT, Qld, and NSW that have similar sociodemographic characteristics to Katherine, Oakey, and Williamtown, respectively.

Confidence interval — a range of probable values for an estimate. The point estimate and its confidence interval are collectively known as the interval estimate.

Confounding — occurs if the characteristics of the exposed population do not match the characteristics of the comparison population, and it is these characteristics that cause an effect (see *effect*) to be observed. This makes the effect estimate biased (see *bias*). For example, if we compare an older population to a younger population, age may be the reason why a difference in rates of disease is observed. Age is a confounding factor here unless appropriately accounted for.

Convergence — see *non-convergence*.

Crude statistic — an estimated statistic prior to any adjustments (see *adjustment*).

Effect — the influence of one condition (for example, exposure) on another (for example, a health effect).

Effect estimate/point estimate — the value of a measurement used to estimate an effect (see *effect*). For example, the estimated prevalence ratio or difference in means.

Elevated level — blood serum PFAS concentration above the background level of exposure observed in the comparison communities, i.e. above the 95th percentile of age-specific serum PFAS concentrations in the comparison population.

Exposed communities — areas with known PFAS contamination, that is, the PFAS Management Areas.

Exposure levels — the level of a population's exposure to PFAS. *Background levels* reflect exposure to low levels of PFAS typically seen in the general population who have not experienced high levels of exposure. *Community exposure levels* reflect exposure to high levels of PFAS through environmental contamination of residential areas located close to facilities that use or produce PFAS. *Occupational levels* represent exposure to very high levels of PFAS through work at facilities that use or produce PFAS.

Exposed population — all individuals who lived in the exposed communities.

Geocoded — providing geographical coordinates corresponding to a location.

Internal validity — the extent to which the findings of a study represent the population being studied, as opposed to 'external validity' which is whether findings of a study can be applied to a population beyond the study in a different setting.

Log-transformation — a type of data transformation used to change the values from a skewed distribution to a symmetric distribution in order to make patterns in the data more interpretable.

Mean — the arithmetic mean or average is a measure of the central value of a set of values; i.e., the sum of the values divided by the number of values. For example, the mean of 1,2,2,2,4,4,5 is 2.85 (20 divided by 7).

Measurement error — incorrectly measured values (see also *misclassification*).

Median — the midpoint of a set of values. For example, the median value of 1,2,2,**2**,4,4,5 is 2. The median can be more useful than the mean when there are many extreme values.

Misclassification — when someone or something is assigned to an incorrect category. For example, someone could be misclassified as non-Indigenous if they did not identify as Aboriginal and/or Torres Strait Islander (see also *measurement error*).

Non-convergence — when an algorithm is not able to find a solution.

Percentile — a score below which a certain percentage of the population falls. For example, 91% of the population falls below an IQ score of 120 (which is the 91st percentile).

PFAS Management Areas — the areas in Katherine, Oakey, and Williamtown, within boundaries defined by the Australian Department of Defence, that have known PFAS contamination. All street addresses within the PFAS Management Areas are captured in the PFAS Address Database.

Prevalence — the proportion or percentage of a population with a specific characteristic during a given time period.

Prevalence ratio — the proportion of a health condition in one group of people relative to the proportion in another group of people.

Regression — a statistical method used to quantify the relationship between two or more variables.

Selection bias — occurs when there is a systematic difference between people who are included in the study and those who are not.

Sociodemographic — a combination of social and demographic factors.

Socioeconomic — a combination of social and economic factors.

Standard deviation — a measure of the spread of a set of values. A low standard deviation means values are closer to the mean, while a large standard deviation means the values are spread over a wider range.

Standard error — the standard deviation of a sampling distribution, which measures the variability of a statistic.

Statistical power — the ability of a study to detect an effect (see *effect*), if there is actually an effect. This depends on the number of people in the study (sample size), how common the health outcome is, how large the variance (spread) of the measure, and how large the expected effect is. The smaller the expected effect, the more power required.

Variable — a characteristic that varies among individuals. A *binary variable* is a variable where there can only be two possible values (for example, 'yes' or 'no'). A *categorical variable* is a variable where there can only be a limited number of values. For example, BMI can be considered a categorical variable with four possible values 'underweight', 'normal', 'overweight', and 'obese'). Note BMI can also be treated as a continuous variable. A *continuous variable* is a variable whose values can take any number including decimal places. For example, age is a continuous variable.

Appendix 1: Sensitivity analyses for self-reported health outcomes

Table A1-1. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from comparison communities, 2020.

	Alice Springs (NT)		Dalby (Qld)		Kiama and Shellharbour (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	154 (4)	1.34 (0.53,3.42)	124 (3)	NC	299 (11)	0.75 (0.48,1.17)
PFOA	154 (4)	1.08 (0.43,2.74)	125 (3)	NC	299 (11)	0.75 (0.46,1.20)
PFHxS	154 (4)	0.80 (0.46,1.40)	125 (3)	NC	299 (11)	0.66 (0.33,1.35)
Prostate cancer						
PFOS	61 (1)	NC	56 (7)	0.73 (0.43,1.23)	131 (11)	1.07 (0.51,2.25)
PFOA	61 (1)	NC	56 (7)	0.68 (0.47,0.98)	131 (11)	0.97 (0.54,1.72)
PFHxS	61 (1)	NC	56 (7)	0.64 (0.45,0.90)	131 (11)	0.98 (0.57,1.69)
Any cancer^						
PFOS	154 (6)	1.29 (0.64,2.57)	124 (11)	0.81 (0.50,1.32)	300 (33)	0.90 (0.66,1.21)
PFOA	154 (6)	1.21 (0.59,2.46)	125 (11)	0.49 (0.34,0.70)	300 (33)	0.98 (0.72,1.33)
PFHxS	154 (6)	1.19 (0.67,2.14)	125 (11)	0.69 (0.48,0.98)	300 (33)	0.83 (0.61,1.12)
Heart attack						
PFOS	153 (5)	NC	123 (5)	0.82 (0.45,1.51)	299 (17)	0.84 (0.55,1.27)
PFOA	153 (5)	NC	124 (5)	1.22 (0.67,2.22)	299 (17)	1.21 (0.80,1.82)
PFHxS	153 (5)	NC	124 (5)	1.24 (0.68,2.27)	299 (17)	1.35 (0.84,2.18)
High blood pressure						
PFOS	154 (37)	1.16 (0.88,1.54)	122 (24)	0.79 (0.58,1.08)	300 (92)	0.97 (0.83,1.14)
PFOA	154 (37)	0.91 (0.64,1.29)	123 (24)	0.82 (0.62,1.09)	300 (92)	1.06 (0.89,1.26)

	Alice Springs (NT)		Dalby (Qld)		Kiama and Shellharbour (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFHxS	154 (37)	0.97 (0.78,1.22)	123 (24)	1.01 (0.73,1.38)	300 (92)	0.99 (0.85,1.15)
Any cardiovascular disease^						
PFOS	154 (40)	1.11 (0.86,1.44)	123 (27)	0.76 (0.58,0.99)	300 (99)	0.93 (0.81,1.08)
PFOA	154 (40)	0.91 (0.66,1.26)	124 (27)	0.85 (0.65,1.12)	300 (99)	1.03 (0.88,1.21)
PFHxS	154 (40)	0.97 (0.78,1.20)	124 (27)	1.05 (0.77,1.42)	300 (99)	0.97 (0.85,1.12)
Hypercholesterolaemia						
PFOS	154 (31)	0.98 (0.72,1.33)	123 (19)	1.15 (0.72,1.85)	299 (86)	1.07 (0.90,1.28)
PFOA	154 (31)	0.72 (0.54,0.96)[S]	124 (19)	0.94 (0.56,1.56)	299 (86)	1.05 (0.86,1.28)
PFHxS	154 (31)	0.87 (0.70,1.09)	124 (19)	1.06 (0.65,1.71)	299 (86)	1.00 (0.86,1.15)
Fatty liver disease						
PFOS	154 (3)	0.86 (0.33,2.22)	123 (3)	0.44 (0.17,1.15)	299 (12)	0.71 (0.45,1.13)
PFOA	154 (3)	0.56 (0.19,1.62)	124 (3)	1.07 (0.56,2.06)	299 (12)	0.75 (0.43,1.33)
PFHxS	154 (3)	0.76 (0.22,2.55)	124 (3)	0.68 (0.25,1.83)	299 (12)	0.63 (0.32,1.23)
Any liver disease^						
PFOS	154 (4)	1.02 (0.44,2.37)	123 (7)	0.74 (0.35,1.58)	300 (17)	0.87 (0.55,1.36)
PFOA	154 (4)	0.63 (0.28,1.44)	124 (7)	1.20 (0.71,2.04)	300 (17)	0.92 (0.56,1.50)
PFHxS	154 (4)	0.91 (0.35,2.34)	124 (7)	1.01 (0.47,2.18)	300 (17)	0.69 (0.41,1.16)
Gout						
PFOS	153 (5)	0.48 (0.24,0.97)	123 (5)	0.52 (0.31,0.86)	300 (20)	1.16 (0.83,1.62)[S]
PFOA	153 (5)	1.37 (0.42,4.54)	124 (5)	1.28 (0.53,3.08)	300 (20)	1.42 (0.88,2.29)
PFHxS	153 (5)	0.92 (0.51,1.67)	124 (5)	0.74 (0.42,1.32)	300 (20)	1.44 (1.10,1.89)[S]
Any kidney disease^						

	Alice Springs (NT)		Dalby (Qld)		Kiama and Shellharbour (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOS	154 (6)	0.58 (0.31,1.06)	123 (6)	0.47 (0.32,0.70)[S]	300 (23)	1.10 (0.80,1.51)[S]
PFOA	154 (6)	1.22 (0.44,3.33)	124 (6)	0.68 (0.36,1.28)	300 (23)	1.27 (0.84,1.91)
PFHxS	154 (6)	0.98 (0.57,1.68)	124 (6)	0.58 (0.42,0.80)	300 (23)	1.38 (1.06,1.81)[S]
Asthma						
PFOS	153 (26)	0.80 (0.59,1.09)	123 (23)	0.68 (0.47,0.98)[S]	299 (40)	1.11 (0.85,1.46)
PFOA	153 (26)	0.88 (0.59,1.32)	124 (23)	0.99 (0.67,1.48)	299 (40)	1.12 (0.80,1.57)
PFHxS	153 (26)	0.90 (0.69,1.17)	124 (23)	0.78 (0.56,1.08)	299 (40)	1.14 (0.85,1.52)
Rheumatoid arthritis						
PFOS	154 (5)	0.93 (0.38,2.24)	123 (2)	NC	298 (11)	1.14 (0.67,1.95)
PFOA	154 (5)	0.33 (0.12,0.85)	124 (2)	NC	298 (11)	1.16 (0.70,1.91)
PFHxS	154 (5)	1.01 (0.50,2.04)	124 (2)	NC	298 (11)	1.64 (1.04,2.58)
Any autoimmune disease^						
PFOS	154 (29)	0.85 (0.63,1.13)	123 (25)	0.64 (0.45,0.91)[S]	299 (53)	0.96 (0.76,1.20)
PFOA	154 (29)	0.83 (0.57,1.20)	124 (25)	0.96 (0.66,1.39)	299 (53)	1.03 (0.78,1.36)
PFHxS	154 (29)	0.89 (0.71,1.13)	124 (25)	0.73 (0.54,1.00)	299 (53)	1.12 (0.88,1.43)
Type II diabetes						
PFOS	154 (14)	0.63 (0.42,0.93)	123 (4)	2.09 (0.96,4.51)[S]	299 (21)	0.91 (0.59,1.41)
PFOA	154 (14)	0.77 (0.50,1.17)	124 (4)	1.31 (0.73,2.36)	299 (21)	0.85 (0.57,1.26)
PFHxS	154 (14)	0.88 (0.60,1.29)	124 (4)	3.17 (1.22,8.25)[S]	299 (21)	1.00 (0.71,1.41)
Hypothyroidism						
PFOS	154 (19)	0.84 (0.51,1.38)	123 (11)	0.92 (0.52,1.63)	299 (20)	0.80 (0.50,1.29)
PFOA	154 (19)	0.78 (0.44,1.40)[S]	124 (11)	1.52 (0.82,2.82)	299 (20)	0.90 (0.56,1.44)

	Alice Springs (NT)		Dalby (Qld)		Kiama and Shellharbour (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFHxS	154 (19)	0.93 (0.62,1.39)	124 (11)	0.78 (0.53,1.14)	299 (20)	0.94 (0.61,1.46)
Hyperthyroidism						
PFOS	151 (1)	0.84 (0.53,1.35)	123 (4)	0.31 (0.06,1.50)	298 (10)	1.02 (0.56,1.85)
PFOA	151 (1)	0.58 (0.40,0.85)	124 (4)	0.13 (0.07,0.26)[S]	298 (10)	1.16 (0.45,2.97)
PFHxS	151 (1)	0.47 (0.33,0.67)[S]	124 (4)	0.44 (0.19,1.04)	298 (10)	0.93 (0.53,1.63)
Problems with fertility						
PFOS	127 (17)	0.90 (0.57,1.44)	104 (18)	0.93 (0.62,1.40)	270 (22)	0.86 (0.61,1.20)
PFOA	127 (17)	0.91 (0.54,1.54)	105 (18)	1.21 (0.89,1.64)	270 (22)	1.00 (0.64,1.54)
PFHxS	127 (17)	1.03 (0.71,1.49)	105 (18)	1.15 (0.80,1.64)	270 (22)	0.84 (0.57,1.23)
Early onset menopause						
PFOS	93 (7)	2.04 (0.98,4.24)[S]	68 (5)	NC	170 (17)	1.42 (0.86,2.37)
PFOA	93 (7)	1.41 (0.86,2.31)[S]	69 (5)	NC	170 (17)	1.34 (0.77,2.34)
PFHxS	93 (7)	1.25 (0.69,2.28)	69 (5)	NC	170 (17)	1.20 (0.83,1.74)

N: sample size; NC: convergence not achieved; PR: prevalence ratio; CI: confidence interval; S: significantly different to the exposed community at a 5% significance level. Sample sizes differ to those in Table 3 because of missing values in confounders.

† Adjusted for age, sex, level of education, gross household annual income, smoking status, alcohol consumption, and estimated glomerular filtration rate. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table A1-2. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: adjusting for additional potential confounders that arise if kidney function is assumed to affect PFAS serum concentrations.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	247 (13)	0.67 (0.48,0.93)	144 (2)	NC	267 (7)	1.15 (0.69,1.92)
PFOA	247 (13)	0.54 (0.31,0.95)	144 (2)	NC	267 (7)	0.68 (0.28,1.64)
PFHxS	247 (13)	0.75 (0.61,0.94)	144 (2)	NC	267 (7)	1.20 (0.88,1.65)
Prostate cancer						
PFOS	137 (3)	NC	105 (3)	0.53 (0.32,0.89)	180 (10)	0.76 (0.44,1.31)
PFOA	137 (3)	NC	105 (3)	0.55 (0.32,0.96)	180 (10)	0.80 (0.34,1.89)
PFHxS	137 (3)	NC	105 (3)	0.51 (0.23,1.09)	180 (10)	0.71 (0.42,1.20)
Any cancer^						
PFOS	247 (21)	0.81 (0.61,1.08)	144 (7)	0.63 (0.40,0.97)	269 (21)	0.93 (0.71,1.22)
PFOA	247 (21)	0.72 (0.47,1.10)	144 (7)	0.72 (0.45,1.14)	269 (21)	0.87 (0.57,1.32)
PFHxS	247 (21)	0.87 (0.73,1.05)	144 (7)	0.72 (0.53,0.98)	269 (21)	0.84 (0.67,1.03)
Heart attack						
PFOS	248 (2)	1.39 (1.07,1.80)	145 (12)	0.90 (0.63,1.28)	269 (17)	0.91 (0.69,1.21)
PFOA	248 (2)	0.90 (0.44,1.85)	145 (12)	1.06 (0.70,1.62)	269 (17)	0.83 (0.53,1.29)
PFHxS	248 (2)	1.24 (0.76,2.03)	145 (12)	0.99 (0.77,1.27)	269 (17)	0.92 (0.74,1.15)
High blood pressure						
PFOS	247 (53)	0.93 (0.81,1.08)	145 (33)	0.81 (0.65,1.00)	269 (67)	1.09 (0.94,1.26)
PFOA	247 (53)	0.85 (0.70,1.04)	145 (33)	0.88 (0.69,1.12)	269 (67)	0.98 (0.81,1.20)
PFHxS	247 (53)	0.91 (0.83,1.01)	145 (33)	0.92 (0.79,1.08)	269 (67)	1.06 (0.94,1.20)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	248 (53)	0.93 (0.80,1.08)	145 (34)	0.82 (0.66,1.01)	269 (70)	1.08 (0.94,1.24)
PFOA	248 (53)	0.84 (0.68,1.03)	145 (34)	0.89 (0.70,1.12)	269 (70)	0.98 (0.81,1.19)
PFHxS	248 (53)	0.91 (0.82,1.01)	145 (34)	0.93 (0.80,1.08)	269 (70)	1.06 (0.95,1.20)
Hypercholesterolaemia						
PFOS	247 (45)	1.05 (0.91,1.22)	144 (32)	1.07 (0.85,1.35)	269 (71)	1.05 (0.92,1.20)
PFOA	247 (45)	1.30 (1.02,1.67)	144 (32)	1.13 (0.83,1.55)	269 (71)	1.13 (0.91,1.40)
PFHxS	247 (45)	1.02 (0.91,1.15)	144 (32)	1.00 (0.85,1.18)	269 (71)	1.08 (0.96,1.22)
Fatty liver disease						
PFOS	247 (17)	1.05 (0.77,1.43)	145 (8)	1.12 (0.75,1.66)	269 (14)	0.89 (0.56,1.42)
PFOA	247 (17)	1.11 (0.74,1.66)	145 (8)	1.26 (0.74,2.13)	269 (14)	1.18 (0.77,1.82)
PFHxS	247 (17)	1.03 (0.80,1.32)	145 (8)	1.38 (0.99,1.93)	269 (14)	0.90 (0.67,1.22)
Any liver disease^						
PFOS	248 (23)	1.01 (0.79,1.29)	145 (9)	0.97 (0.69,1.37)	269 (17)	0.93 (0.61,1.40)
PFOA	248 (23)	1.03 (0.76,1.39)	145 (9)	1.34 (0.77,2.31)	269 (17)	1.06 (0.74,1.53)
PFHxS	248 (23)	1.06 (0.88,1.27)	145 (9)	1.24 (0.96,1.61)	269 (17)	0.90 (0.70,1.17)
Gout						
PFOS	247 (19)	0.78 (0.60,1.00)	145 (10)	0.75 (0.52,1.09)	270 (30)	0.73 (0.60,0.88)
PFOA	247 (19)	1.40 (0.91,2.16)	145 (10)	0.95 (0.50,1.78)	270 (30)	1.65 (1.10,2.48)
PFHxS	247 (19)	0.92 (0.76,1.10)	145 (10)	0.86 (0.58,1.27)	270 (30)	0.83 (0.69,1.00)
Any kidney disease^						
PFOS		N/A		N/A		N/A

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA		N/A		N/A		N/A
PFHxS		N/A		N/A		N/A
Asthma						
PFOS	248 (40)	0.96 (0.80,1.15)	145 (21)	0.99 (0.77,1.27)	269 (37)	1.14 (0.93,1.40)
PFOA	248 (40)	0.84 (0.66,1.07)	145 (21)	0.75 (0.53,1.05)	269 (37)	0.92 (0.63,1.34)
PFHxS	248 (40)	0.99 (0.87,1.14)	145 (21)	0.99 (0.77,1.27)	269 (37)	0.99 (0.81,1.22)
Rheumatoid arthritis						
PFOS	247 (9)	0.82 (0.56,1.20)	145 (9)	NC	269 (15)	0.74 (0.50,1.10)
PFOA	247 (9)	0.74 (0.47,1.17)	145 (9)	NC	269 (15)	0.74 (0.35,1.56)
PFHxS	247 (9)	0.92 (0.73,1.14)	145 (9)	NC	269 (15)	0.97 (0.72,1.32)
Any autoimmune disease^						
PFOS	248 (55)	0.88 (0.75,1.03)	145 (32)	0.96 (0.76,1.20)	269 (58)	0.94 (0.77,1.15)
PFOA	248 (55)	0.81 (0.67,0.98)	145 (32)	0.83 (0.65,1.07)	269 (58)	0.82 (0.62,1.09)
PFHxS	248 (55)	0.91 (0.81,1.02)	145 (32)	0.97 (0.80,1.19)	269 (58)	1.00 (0.86,1.16)
Type II diabetes						
PFOS	248 (14)	0.88 (0.70,1.10)	145 (12)	0.81 (0.57,1.15)	269 (20)	0.89 (0.62,1.27)
PFOA	248 (14)	0.78 (0.56,1.11)	145 (12)	0.78 (0.52,1.19)	269 (20)	0.68 (0.43,1.08)
PFHxS	248 (14)	0.93 (0.78,1.11)	145 (12)	0.82 (0.63,1.07)	269 (20)	0.99 (0.76,1.29)
Hypothyroidism						
PFOS	247 (7)	0.89 (0.68,1.17)	145 (6)	0.71 (0.46,1.10)	269 (9)	0.80 (0.56,1.15)
PFOA	247 (7)	2.19 (1.27,3.77)	145 (6)	0.95 (0.47,1.94)	269 (9)	1.14 (0.67,1.94)
PFHxS	247 (7)	0.75 (0.58,0.96)	145 (6)	0.91 (0.50,1.66)	269 (9)	1.18 (0.90,1.53)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	247 (10)	0.89 (0.71,1.12)	145 (4)	1.04 (0.62,1.77)	269 (9)	1.04 (0.82,1.32)
PFOA	247 (10)	0.74 (0.54,1.01)	145 (4)	1.26 (0.58,2.76)	269 (9)	0.68 (0.35,1.33)
PFHxS	247 (10)	0.95 (0.78,1.15)	145 (4)	1.18 (0.72,1.94)	269 (9)	0.87 (0.59,1.28)
Problems with fertility						
PFOS	208 (22)	0.81 (0.53,1.25)	119 (13)	1.14 (0.79,1.63)	231 (34)	0.86 (0.67,1.10)
PFOA	208 (22)	0.74 (0.47,1.19)	119 (13)	0.83 (0.62,1.13)	231 (34)	0.87 (0.63,1.20)
PFHxS	208 (22)	0.84 (0.66,1.07)	119 (13)	1.26 (0.92,1.72)	231 (34)	0.76 (0.63,0.92)
Early onset menopause						
PFOS	113 (8)	0.88 (0.57,1.37)#	39 (0)	NC	89 (8)	0.87 (0.65,1.17)
PFOA	113 (8)	0.76 (0.47,1.24)#	39 (0)	NC	89 (8)	0.88 (0.48,1.61)
PFHxS	113 (8)	0.91 (0.67,1.24)#	39 (0)	NC	89 (8)	0.98 (0.70,1.36)

N: sample size; NC: convergence not achieved; N/A: not applicable; PR: prevalence ratio; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, smoking status, alcohol consumption, and estimated glomerular filtration rate. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table A1-3. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: excluding exposed participants who now live in comparison communities.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	265 (13)	0.63 (0.44,0.90)	127 (2)	NC	283 (7)	1.12 (0.71,1.79)
PFOA	265 (13)	0.60 (0.30,1.21)	127 (2)	NC	283 (7)	0.73 (0.31,1.72)
PFHxS	265 (13)	0.70 (0.55,0.89)	127 (2)	NC	283 (7)	1.16 (0.86,1.55)
Prostate cancer						
PFOS	144 (4)	NC	96 (2)	0.57 (0.32,1.00)	191 (9)	0.77 (0.45,1.30)
PFOA	144 (4)	NC	96 (2)	0.57 (0.27,1.21)	191 (9)	0.79 (0.30,2.11)
PFHxS	144 (4)	NC	96 (2)	0.48 (0.31,0.74)	191 (9)	0.60 (0.35,1.03)
Any cancer^						
PFOS	265 (21)	0.75 (0.56,1.02)	127 (6)	0.58 (0.39,0.89)	286 (21)	0.98 (0.73,1.31)
PFOA	265 (21)	0.70 (0.44,1.12)	127 (6)	0.72 (0.42,1.26)	286 (21)	0.96 (0.59,1.57)
PFHxS	265 (21)	0.82 (0.67,1.00)	127 (6)	0.63 (0.46,0.84)	286 (21)	0.81 (0.66,1.01)
Heart attack						
PFOS	267 (4)	NC	128 (10)	0.76 (0.51,1.15)	285 (19)	0.85 (0.65,1.12)
PFOA	267 (4)	NC	128 (10)	1.20 (0.79,1.81)	285 (19)	0.87 (0.57,1.32)
PFHxS	267 (4)	NC	128 (10)	0.92 (0.75,1.13)	285 (19)	0.87 (0.71,1.08)
High blood pressure						
PFOS	266 (56)	0.98 (0.85,1.13)	128 (29)	0.86 (0.69,1.08)	286 (79)	1.05 (0.92,1.20)
PFOA	266 (56)	0.93 (0.74,1.17)	128 (29)	0.92 (0.68,1.23)	286 (79)	0.95 (0.78,1.15)
PFHxS	266 (56)	0.93 (0.84,1.04)	128 (29)	0.96 (0.81,1.14)	286 (79)	1.00 (0.88,1.12)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	267 (57)	0.98 (0.85,1.12)	128 (30)	0.87 (0.70,1.08)	286 (82)	1.04 (0.92,1.19)
PFOA	267 (57)	0.93 (0.75,1.16)	128 (30)	0.92 (0.70,1.22)	286 (82)	0.95 (0.79,1.14)
PFHxS	267 (57)	0.92 (0.83,1.03)	128 (30)	0.96 (0.81,1.14)	286 (82)	1.00 (0.89,1.12)
Hypercholesterolaemia						
PFOS	267 (48)	1.07 (0.93,1.24)	126 (29)	0.99 (0.79,1.24)	285 (74)	1.04 (0.91,1.18)
PFOA	267 (48)	1.36 (1.07,1.73)	126 (29)	1.15 (0.80,1.65)	285 (74)	1.15 (0.94,1.41)
PFHxS	267 (48)	1.04 (0.92,1.17)	126 (29)	0.94 (0.80,1.11)#	285 (74)	1.06 (0.94,1.20)
Fatty liver disease						
PFOS	266 (18)	1.00 (0.72,1.38)	128 (5)	1.21 (0.78,1.90)	285 (12)	0.94 (0.54,1.63)
PFOA	266 (18)	1.10 (0.75,1.62)	128 (5)	1.57 (0.49,4.98)	285 (12)	1.10 (0.73,1.67)
PFHxS	266 (18)	0.97 (0.74,1.27)	128 (5)	1.20 (0.84,1.73)	285 (12)	1.02 (0.76,1.36)
Any liver disease^						
PFOS	267 (23)	1.02 (0.77,1.34)	128 (5)	1.19 (0.80,1.75)	285 (15)	0.98 (0.61,1.56)
PFOA	267 (23)	1.11 (0.80,1.54)	128 (5)	1.57 (0.45,5.48)	285 (15)	1.04 (0.73,1.48)
PFHxS	267 (23)	1.07 (0.86,1.32)	128 (5)	1.23 (0.86,1.76)	285 (15)	0.98 (0.76,1.28)
Gout						
PFOS	266 (20)	0.81 (0.66,1.01)	127 (12)	0.67 (0.50,0.91)	283 (29)	0.75 (0.62,0.92)
PFOA	266 (20)	1.51 (1.00,2.29)	127 (12)	0.82 (0.52,1.29)	283 (29)	1.51 (1.01,2.26)
PFHxS	266 (20)	0.90 (0.76,1.07)	127 (12)	0.80 (0.62,1.03)	283 (29)	0.90 (0.73,1.10)
Any kidney disease^						
PFOS	267 (22)	0.87 (0.72,1.06)	128 (15)	0.71 (0.56,0.90)	285 (31)	0.75 (0.62,0.91)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA	267 (22)	1.33 (0.74,2.41)	128 (15)	0.93 (0.59,1.46)	285 (31)	1.45 (0.99,2.13)
PFHxS	267 (22)	0.91 (0.78,1.07)	128 (15)	0.87 (0.67,1.14)	285 (31)	0.86 (0.71,1.05)
Asthma						
PFOS	267 (41)	1.00 (0.84,1.20)	128 (18)	1.02 (0.76,1.37)	285 (43)	1.16 (0.95,1.41)
PFOA	267 (41)	0.84 (0.66,1.07)	128 (18)	0.67 (0.47,0.95)	285 (43)	0.93 (0.66,1.32)
PFHxS	267 (41)	1.03 (0.89,1.19)	128 (18)	0.98 (0.74,1.31)	285 (43)	1.04 (0.86,1.26)
Rheumatoid arthritis						
PFOS	266 (10)	0.79 (0.54,1.16)	128 (10)	NC	285 (17)	0.72 (0.50,1.03)
PFOA	266 (10)	0.55 (0.33,0.90)	128 (10)	NC	285 (17)	0.67 (0.35,1.28)
PFHxS	266 (10)	0.92 (0.73,1.16)	128 (10)	NC	285 (17)	0.96 (0.71,1.29)
Any autoimmune disease^						
PFOS	267 (57)	0.91 (0.78,1.07)	128 (29)	0.92 (0.73,1.16)	285 (66)	0.93 (0.78,1.12)
PFOA	267 (57)	0.81 (0.66,0.98)	128 (29)	0.78 (0.60,1.02)	285 (66)	0.81 (0.63,1.05)
PFHxS	267 (57)	0.94 (0.83,1.06)	128 (29)	0.92 (0.77,1.11)	285 (66)	0.99 (0.86,1.15)
Type II diabetes						
PFOS	267 (12)	0.92 (0.67,1.26)	127 (11)	0.74 (0.53,1.05)	286 (24)	0.91 (0.68,1.22)
PFOA	267 (12)	0.76 (0.50,1.16)	127 (11)	0.86 (0.48,1.52)	286 (24)	0.75 (0.51,1.11)
PFHxS	267 (12)	1.06 (0.86,1.31)	127 (11)	0.77 (0.61,0.98)	286 (24)	0.97 (0.77,1.20)
Hypothyroidism						
PFOS	268 (8)	0.78 (0.56,1.10)	128 (3)	1.16 (0.86,1.56)	283 (10)	0.75 (0.53,1.05)
PFOA	268 (8)	1.88 (1.18,3.00)	128 (3)	8.44 (1.08,66.01)	283 (10)	1.17 (0.71,1.94)
PFHxS	268 (8)	0.74 (0.55,0.99)	128 (3)	1.73 (1.00,3.01)	283 (10)	1.05 (0.81,1.37)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	266 (10)	0.88 (0.68,1.14)	128 (4)	1.01 (0.58,1.78)	284 (12)	1.04 (0.81,1.35)
PFOA	266 (10)	0.78 (0.49,1.26)	128 (4)	1.09 (0.31,3.88)	284 (12)	0.93 (0.56,1.56)
PFHxS	266 (10)	0.91 (0.73,1.14)	128 (4)	1.12 (0.62,2.02)	284 (12)	0.93 (0.69,1.24)
Problems with fertility						
PFOS	224 (27)	0.97 (0.74,1.27)	104 (14)	1.09 (0.79,1.50)	243 (36)	0.90 (0.71,1.14)
PFOA	224 (27)	0.91 (0.64,1.28)	104 (14)	0.87 (0.55,1.36)	243 (36)	0.90 (0.66,1.23)
PFHxS	224 (27)	0.93 (0.76,1.13)	104 (14)	1.20 (0.88,1.63)	243 (36)	0.81 (0.66,0.99)
Early onset menopause						
PFOS	128 (9)	0.95 (0.71,1.27)	31 (0)	NC	96 (9)	0.94 (0.67,1.31)
PFOA	128 (9)	0.59 (0.36,0.96)	31 (0)	NC	96 (9)	0.96 (0.53,1.74)
PFHxS	128 (9)	1.04 (0.84,1.28)	31 (0)	NC	96 (9)	0.92 (0.68,1.25)

N: sample size; NC: convergence not achieved; N/A: not applicable; PR: prevalence ratio; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table A1-4. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: excluding exposed participants who have not lived in the exposed communities in the last 15 years.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	259 (12)	0.64 (0.43,0.95)	152 (2)	NC	266 (4)	0.93 (0.64,1.36)
PFOA	259 (12)	0.51 (0.21,1.25)	152 (2)	NC	266 (4)	0.42 (0.15,1.20)
PFHxS	259 (12)	0.67 (0.51,0.87)	152 (2)	NC	266 (4)	1.00 (0.81,1.23)
Prostate cancer						
PFOS	142 (4)	NC	111 (3)	0.59 (0.37,0.95)	190 (10)	0.78 (0.47,1.32)
PFOA	142 (4)	NC	111 (3)	0.56 (0.30,1.04)	190 (10)	0.80 (0.32,1.98)
PFHxS	142 (4)	NC	111 (3)	0.55 (0.39,0.78)	190 (10)	0.71 (0.43,1.19)
Any cancer^						
PFOS	259 (21)	0.77 (0.58,1.03)	154 (9)	0.64 (0.44,0.92)	269 (19)	0.93 (0.70,1.25)
PFOA	259 (21)	0.66 (0.41,1.08)	154 (9)	0.76 (0.46,1.25)	269 (19)	0.86 (0.51,1.45)
PFHxS	259 (21)	0.80 (0.65,0.98)	154 (9)	0.67 (0.51,0.88)	269 (19)	0.80 (0.64,1.00)
Heart attack						
PFOS	261 (4)	NC	154 (14)	0.77 (0.56,1.07)	268 (19)	0.86 (0.65,1.14)
PFOA	261 (4)	NC	154 (14)	1.14 (0.78,1.66)	268 (19)	0.85 (0.55,1.30)
PFHxS	261 (4)	NC	154 (14)	0.94 (0.78,1.12)	268 (19)	0.89 (0.73,1.10)
High blood pressure						
PFOS	260 (57)	1.00 (0.87,1.15)	155 (40)	0.82 (0.67,1.00)	269 (76)	1.06 (0.91,1.22)
PFOA	260 (57)	0.94 (0.74,1.18)	155 (40)	0.90 (0.71,1.13)	269 (76)	0.93 (0.76,1.14)
PFHxS	260 (57)	0.93 (0.83,1.04)	155 (40)	0.94 (0.80,1.10)	269 (76)	1.00 (0.89,1.13)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	261 (58)	0.99 (0.86,1.14)	155 (41)	0.83 (0.68,1.00)	269 (79)	1.05 (0.91,1.21)
PFOA	261 (58)	0.93 (0.74,1.17)	155 (41)	0.90 (0.72,1.13)	269 (79)	0.93 (0.77,1.13)
PFHxS	261 (58)	0.92 (0.82,1.03)	155 (41)	0.94 (0.81,1.10)	269 (79)	1.01 (0.90,1.13)
Hypercholesterolaemia						
PFOS	261 (49)	1.07 (0.92,1.23)	153 (35)	0.99 (0.81,1.20)	268 (73)	1.05 (0.92,1.20)
PFOA	261 (49)	1.32 (1.02,1.70)	153 (35)	1.07 (0.81,1.42)	268 (73)	1.18 (0.95,1.45)
PFHxS	261 (49)	1.02 (0.90,1.16)	153 (35)	0.97 (0.82,1.14)	268 (73)	1.04 (0.92,1.17)
Fatty liver disease						
PFOS	260 (18)	1.00 (0.72,1.40)	154 (7)	1.12 (0.75,1.69)	268 (14)	0.88 (0.53,1.45)
PFOA	260 (18)	1.10 (0.74,1.65)	154 (7)	1.15 (0.69,1.91)	268 (14)	1.19 (0.80,1.77)
PFHxS	260 (18)	0.97 (0.73,1.29)	154 (7)	1.25 (0.92,1.69)	268 (14)	0.90 (0.67,1.20)
Any liver disease^						
PFOS	261 (24)	1.00 (0.76,1.32)	154 (8)	0.94 (0.65,1.35)	268 (17)	0.92 (0.60,1.42)
PFOA	261 (24)	1.11 (0.81,1.52)	154 (8)	1.17 (0.71,1.92)	268 (17)	1.11 (0.78,1.57)
PFHxS	261 (24)	1.04 (0.83,1.29)	154 (8)	1.15 (0.89,1.50)	268 (17)	0.89 (0.69,1.15)
Gout						
PFOS	260 (20)	0.82 (0.66,1.02)	153 (13)	0.77 (0.56,1.04)	267 (32)	0.77 (0.64,0.93)
PFOA	260 (20)	1.54 (1.01,2.35)	153 (13)	0.88 (0.52,1.47)	267 (32)	1.58 (1.06,2.34)
PFHxS	260 (20)	0.90 (0.76,1.07)	153 (13)	0.83 (0.63,1.10)	267 (32)	0.87 (0.73,1.04)
Any kidney disease^						
PFOS	261 (23)	0.87 (0.72,1.05)	154 (17)	0.74 (0.60,0.93)	269 (34)	0.77 (0.64,0.92)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA	261 (23)	1.28 (0.73,2.24)	154 (17)	0.91 (0.58,1.44)	269 (34)	1.51 (1.04,2.19)
PFHxS	261 (23)	0.89 (0.76,1.04)	154 (17)	0.89 (0.68,1.15)	269 (34)	0.84 (0.71,1.00)
Asthma						
PFOS	261 (41)	1.02 (0.86,1.21)	154 (24)	1.02 (0.79,1.31)	268 (40)	1.13 (0.93,1.39)
PFOA	261 (41)	0.90 (0.70,1.16)	154 (24)	0.74 (0.55,1.00)	268 (40)	0.86 (0.59,1.24)
PFHxS	261 (41)	1.03 (0.89,1.20)	154 (24)	0.98 (0.77,1.25)	268 (40)	1.03 (0.85,1.27)
Rheumatoid arthritis						
PFOS	260 (11)	0.72 (0.46,1.14)	154 (10)	NC	268 (16)	0.64 (0.49,0.83)
PFOA	260 (11)	0.52 (0.29,0.91)	154 (10)	NC	268 (16)	0.63 (0.32,1.23)
PFHxS	260 (11)	0.88 (0.66,1.16)	154 (10)	NC	268 (16)	0.92 (0.70,1.21)
Any autoimmune disease^						
PFOS	261 (58)	0.90 (0.76,1.06)	154 (34)	0.97 (0.78,1.19)	268 (61)	0.87 (0.73,1.05)
PFOA	261 (58)	0.85 (0.69,1.04)	154 (34)	0.81 (0.64,1.03)	268 (61)	0.74 (0.56,0.97)
PFHxS	261 (58)	0.93 (0.81,1.06)	154 (34)	0.96 (0.80,1.15)	268 (61)	0.97 (0.84,1.13)
Type II diabetes						
PFOS	261 (13)	0.92 (0.68,1.24)	153 (13)	0.74 (0.53,1.03)	268 (20)	0.93 (0.65,1.34)
PFOA	261 (13)	0.86 (0.53,1.40)	153 (13)	0.82 (0.49,1.38)	268 (20)	0.70 (0.45,1.09)
PFHxS	261 (13)	1.01 (0.82,1.25)	153 (13)	0.80 (0.61,1.06)	268 (20)	1.02 (0.80,1.30)
Hypothyroidism						
PFOS	262 (8)	0.76 (0.53,1.09)	155 (7)	0.71 (0.46,1.09)	266 (8)	0.74 (0.48,1.12)
PFOA	262 (8)	2.07 (1.20,3.57)	155 (7)	0.87 (0.42,1.82)	266 (8)	1.20 (0.71,2.02)
PFHxS	262 (8)	0.69 (0.48,0.98)	155 (7)	0.84 (0.47,1.49)	266 (8)	1.00 (0.73,1.35)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	260 (9)	0.90 (0.69,1.16)	154 (5)	1.00 (0.63,1.59)	266 (9)	0.99 (0.73,1.34)
PFOA	260 (9)	0.69 (0.41,1.14)	154 (5)	1.03 (0.48,2.20)	266 (9)	0.91 (0.48,1.72)
PFHxS	260 (9)	0.97 (0.80,1.18)	154 (5)	1.04 (0.61,1.78)	266 (9)	0.81 (0.58,1.12)
Problems with fertility						
PFOS	221 (24)	1.00 (0.74,1.34)	123 (16)	1.11 (0.82,1.50)	231 (31)	0.86 (0.67,1.10)
PFOA	221 (24)	0.78 (0.55,1.11)	123 (16)	0.86 (0.61,1.21)	231 (31)	0.89 (0.64,1.24)
PFHxS	221 (24)	0.96 (0.76,1.21)	123 (16)	1.21 (0.91,1.60)	231 (31)	0.80 (0.64,1.00)
Early onset menopause						
PFOS	123 (10)	0.85 (0.59,1.24)	42 (0)	NC	79 (7)	0.92 (0.59,1.46)
PFOA	123 (10)	0.57 (0.34,0.98)	42 (0)	NC	79 (7)	1.16 (0.59,2.29)
PFHxS	123 (10)	0.95 (0.73,1.23)	42 (0)	NC	79 (7)	1.12 (0.80,1.57)

N: sample size; NC: convergence not achieved; N/A: not applicable; PR: prevalence ratio; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table A1-5. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: excluding exposed participants who have not lived in the exposed communities in the last 10 years.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	253 (10)	0.63 (0.43,0.92)	149 (2)	NC	260 (4)	0.93 (0.64,1.35)
PFOA	253 (10)	0.41 (0.16,1.06)	149 (2)	NC	260 (4)	0.42 (0.15,1.19)
PFHxS	253 (10)	0.66 (0.51,0.86)	149 (2)	NC	260 (4)	0.99 (0.81,1.23)
Prostate cancer						
PFOS	140 (4)	NC	110 (3)	0.59 (0.37,0.95)	185 (10)	0.78 (0.47,1.31)
PFOA	140 (4)	NC	110 (3)	0.56 (0.30,1.04)	185 (10)	0.81 (0.32,2.03)
PFHxS	140 (4)	NC	110 (3)	0.55 (0.39,0.78)	185 (10)	0.72 (0.43,1.19)
Any cancer^						
PFOS	253 (19)	0.75 (0.56,1.01)	151 (9)	0.64 (0.44,0.92)	263 (19)	0.93 (0.70,1.24)
PFOA	253 (19)	0.61 (0.37,1.00)	151 (9)	0.76 (0.46,1.24)	263 (19)	0.86 (0.51,1.45)
PFHxS	253 (19)	0.80 (0.64,0.99)	151 (9)	0.67 (0.51,0.88)	263 (19)	0.80 (0.64,1.00)
Heart attack						
PFOS	255 (4)	NC	151 (13)	0.78 (0.55,1.10)	262 (18)	0.85 (0.64,1.14)
PFOA	255 (4)	NC	151 (13)	1.15 (0.76,1.75)	262 (18)	0.78 (0.52,1.18)
PFHxS	255 (4)	NC	151 (13)	0.93 (0.77,1.12)	262 (18)	0.90 (0.72,1.11)
High blood pressure						
PFOS	254 (54)	1.00 (0.87,1.16)	152 (39)	0.82 (0.68,1.00)	263 (75)	1.05 (0.91,1.22)
PFOA	254 (54)	0.91 (0.72,1.17)	152 (39)	0.89 (0.70,1.14)	263 (75)	0.92 (0.75,1.13)
PFHxS	254 (54)	0.94 (0.84,1.06)	152 (39)	0.94 (0.80,1.10)	263 (75)	1.00 (0.89,1.13)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	255 (55)	0.99 (0.86,1.15)	152 (40)	0.83 (0.69,1.01)	263 (78)	1.05 (0.91,1.21)
PFOA	255 (55)	0.91 (0.72,1.15)	152 (40)	0.90 (0.71,1.14)	263 (78)	0.92 (0.76,1.12)
PFHxS	255 (55)	0.93 (0.83,1.05)	152 (40)	0.94 (0.81,1.10)	263 (78)	1.01 (0.89,1.13)
Hypercholesterolaemia						
PFOS	255 (47)	1.08 (0.93,1.25)	150 (35)	0.99 (0.81,1.20)	262 (72)	1.05 (0.92,1.20)
PFOA	255 (47)	1.35 (1.04,1.75)	150 (35)	1.07 (0.81,1.41)	262 (72)	1.16 (0.94,1.43)
PFHxS	255 (47)	1.04 (0.91,1.18)	150 (35)	0.97 (0.82,1.13)	262 (72)	1.04 (0.92,1.17)
Fatty liver disease						
PFOS	254 (18)	1.00 (0.72,1.39)	151 (7)	1.12 (0.74,1.68)	262 (12)	0.98 (0.59,1.63)
PFOA	254 (18)	1.12 (0.75,1.68)	151 (7)	1.13 (0.68,1.87)	262 (12)	1.08 (0.73,1.59)
PFHxS	254 (18)	0.96 (0.72,1.28)	151 (7)	1.25 (0.92,1.68)	262 (12)	0.92 (0.67,1.27)
Any liver disease^						
PFOS	255 (24)	0.99 (0.76,1.31)	151 (8)	0.93 (0.65,1.34)	262 (15)	1.02 (0.66,1.57)
PFOA	255 (24)	1.13 (0.82,1.54)	151 (8)	1.16 (0.71,1.90)	262 (15)	1.01 (0.72,1.42)
PFHxS	255 (24)	1.02 (0.81,1.28)	151 (8)	1.15 (0.89,1.50)	262 (15)	0.91 (0.69,1.20)
Gout						
PFOS	254 (20)	0.82 (0.66,1.02)	150 (13)	0.77 (0.56,1.04)	261 (32)	0.77 (0.64,0.93)
PFOA	254 (20)	1.54 (1.01,2.35)	150 (13)	0.88 (0.52,1.47)	261 (32)	1.59 (1.07,2.36)
PFHxS	254 (20)	0.90 (0.76,1.07)	150 (13)	0.83 (0.63,1.10)	261 (32)	0.86 (0.72,1.03)
Any kidney disease^						
PFOS	255 (22)	0.86 (0.70,1.04)	151 (17)	0.74 (0.60,0.93)	263 (34)	0.77 (0.64,0.92)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA	255 (22)	1.15 (0.69,1.94)	151 (17)	0.91 (0.58,1.44)	263 (34)	1.52 (1.05,2.22)
PFHxS	255 (22)	0.90 (0.77,1.05)	151 (17)	0.89 (0.68,1.15)	263 (34)	0.84 (0.71,1.00)
Asthma						
PFOS	255 (40)	1.00 (0.84,1.19)	151 (24)	1.01 (0.79,1.30)	262 (40)	1.12 (0.91,1.37)
PFOA	255 (40)	0.88 (0.69,1.12)	151 (24)	0.74 (0.55,1.00)	262 (40)	0.85 (0.59,1.23)
PFHxS	255 (40)	1.03 (0.89,1.20)	151 (24)	0.98 (0.77,1.24)	262 (40)	1.02 (0.83,1.25)
Rheumatoid arthritis						
PFOS	254 (11)	0.72 (0.46,1.13)	151 (10)	NC	262 (16)	0.64 (0.49,0.83)
PFOA	254 (11)	0.52 (0.30,0.91)	151 (10)	NC	262 (16)	0.63 (0.32,1.24)
PFHxS	254 (11)	0.87 (0.66,1.15)	151 (10)	NC	262 (16)	0.92 (0.70,1.20)
Any autoimmune disease^						
PFOS	255 (57)	0.89 (0.75,1.05)	151 (34)	0.96 (0.78,1.19)	262 (61)	0.86 (0.72,1.03)
PFOA	255 (57)	0.83 (0.68,1.02)	151 (34)	0.81 (0.64,1.02)	262 (61)	0.74 (0.56,0.97)
PFHxS	255 (57)	0.92 (0.80,1.05)	151 (34)	0.96 (0.80,1.15)	262 (61)	0.96 (0.83,1.11)
Type II diabetes						
PFOS	255 (13)	0.91 (0.68,1.23)	150 (12)	0.75 (0.54,1.06)	262 (19)	0.93 (0.64,1.36)
PFOA	255 (13)	0.87 (0.53,1.42)	150 (12)	0.81 (0.47,1.42)	262 (19)	0.65 (0.42,0.99)
PFHxS	255 (13)	1.00 (0.81,1.24)	150 (12)	0.79 (0.59,1.05)	262 (19)	1.03 (0.80,1.32)
Hypothyroidism						
PFOS	256 (7)	0.78 (0.53,1.14)	152 (7)	0.71 (0.46,1.09)	260 (8)	0.74 (0.49,1.12)
PFOA	256 (7)	2.10 (1.17,3.77)	152 (7)	0.87 (0.42,1.80)	260 (8)	1.19 (0.71,2.00)
PFHxS	256 (7)	0.70 (0.47,1.03)	152 (7)	0.84 (0.48,1.48)	260 (8)	0.99 (0.73,1.34)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	254 (9)	0.89 (0.69,1.15)	151 (5)	0.99 (0.63,1.57)	260 (9)	0.98 (0.72,1.33)
PFOA	254 (9)	0.69 (0.42,1.15)	151 (5)	0.96 (0.47,1.99)	260 (9)	0.92 (0.48,1.74)
PFHxS	254 (9)	0.95 (0.77,1.17)	151 (5)	1.03 (0.61,1.75)	260 (9)	0.80 (0.58,1.11)
Problems with fertility						
PFOS	216 (24)	0.99 (0.74,1.33)	121 (16)	1.11 (0.83,1.48)	227 (31)	0.85 (0.67,1.09)
PFOA	216 (24)	0.78 (0.55,1.11)	121 (16)	0.83 (0.60,1.15)	227 (31)	0.88 (0.64,1.23)
PFHxS	216 (24)	0.95 (0.75,1.20)	121 (16)	1.19 (0.90,1.57)	227 (31)	0.79 (0.64,0.99)
Early onset menopause						
PFOS	119 (9)	0.86 (0.58,1.28)	40 (0)	NC	78 (7)	0.92 (0.59,1.45)
PFOA	119 (9)	0.57 (0.33,1.00)	40 (0)	NC	78 (7)	1.15 (0.58,2.27)
PFHxS	119 (9)	0.99 (0.77,1.27)	40 (0)	NC	78 (7)	1.11 (0.80,1.56)

N: sample size; NC: convergence not achieved; N/A: not applicable; PR: prevalence ratio; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table A1-6. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: excluding exposed participants who have not lived in the exposed communities in the last 5 years.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	245 (10)	0.61 (0.42,0.90)	149 (2)	NC	256 (4)	0.92 (0.64,1.33)
PFOA	245 (10)	0.42 (0.16,1.06)	149 (2)	NC	256 (4)	0.42 (0.15,1.17)
PFHxS	245 (10)	0.66 (0.51,0.85)	149 (2)	NC	256 (4)	0.99 (0.81,1.22)
Prostate cancer						
PFOS	139 (4)	NC	110 (3)	0.59 (0.37,0.95)	184 (10)	0.80 (0.47,1.37)
PFOA	139 (4)	NC	110 (3)	0.56 (0.30,1.04)	184 (10)	0.74 (0.29,1.86)
PFHxS	139 (4)	NC	110 (3)	0.55 (0.39,0.78)	184 (10)	0.72 (0.44,1.19)
Any cancer^						
PFOS	245 (19)	0.73 (0.54,0.99)	151 (9)	0.64 (0.44,0.92)	259 (19)	0.94 (0.70,1.25)
PFOA	245 (19)	0.61 (0.37,1.01)	151 (9)	0.76 (0.46,1.24)	259 (19)	0.83 (0.50,1.39)
PFHxS	245 (19)	0.79 (0.64,0.98)	151 (9)	0.67 (0.51,0.88)	259 (19)	0.80 (0.64,0.99)
Heart attack						
PFOS	247 (4)	NC	151 (13)	0.78 (0.55,1.10)	258 (18)	0.86 (0.64,1.15)
PFOA	247 (4)	NC	151 (13)	1.15 (0.76,1.75)	258 (18)	0.76 (0.51,1.14)
PFHxS	247 (4)	NC	151 (13)	0.93 (0.77,1.12)	258 (18)	0.90 (0.73,1.11)
High blood pressure						
PFOS	246 (53)	1.00 (0.87,1.16)	152 (39)	0.82 (0.68,1.00)	259 (75)	1.06 (0.91,1.22)
PFOA	246 (53)	0.91 (0.71,1.16)	152 (39)	0.89 (0.70,1.14)	259 (75)	0.90 (0.74,1.10)
PFHxS	246 (53)	0.94 (0.83,1.06)	152 (39)	0.94 (0.80,1.10)	259 (75)	1.00 (0.89,1.13)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	247 (54)	1.00 (0.86,1.15)	152 (40)	0.83 (0.69,1.01)	259 (78)	1.05 (0.91,1.21)
PFOA	247 (54)	0.91 (0.71,1.15)	152 (40)	0.90 (0.71,1.14)	259 (78)	0.91 (0.75,1.10)
PFHxS	247 (54)	0.93 (0.83,1.04)	152 (40)	0.94 (0.81,1.10)	259 (78)	1.01 (0.90,1.13)
Hypercholesterolaemia						
PFOS	247 (47)	1.07 (0.92,1.24)	150 (35)	0.99 (0.81,1.20)	258 (71)	1.05 (0.92,1.21)
PFOA	247 (47)	1.34 (1.04,1.74)	150 (35)	1.07 (0.81,1.41)	258 (71)	1.15 (0.93,1.42)
PFHxS	247 (47)	1.03 (0.91,1.17)	150 (35)	0.97 (0.82,1.13)	258 (71)	1.03 (0.92,1.16)
Fatty liver disease						
PFOS	246 (18)	0.98 (0.70,1.37)	151 (7)	1.12 (0.74,1.68)	258 (12)	0.98 (0.59,1.62)
PFOA	246 (18)	1.12 (0.75,1.67)	151 (7)	1.13 (0.68,1.87)	258 (12)	1.07 (0.72,1.58)
PFHxS	246 (18)	0.94 (0.71,1.26)	151 (7)	1.25 (0.92,1.68)	258 (12)	0.92 (0.66,1.26)
Any liver disease^						
PFOS	247 (23)	1.01 (0.76,1.33)	151 (8)	0.93 (0.65,1.34)	258 (15)	1.02 (0.66,1.57)
PFOA	247 (23)	1.13 (0.82,1.55)	151 (8)	1.16 (0.71,1.90)	258 (15)	1.00 (0.71,1.40)
PFHxS	247 (23)	1.00 (0.79,1.28)	151 (8)	1.15 (0.89,1.50)	258 (15)	0.91 (0.69,1.20)
Gout						
PFOS	246 (20)	0.82 (0.66,1.02)	150 (13)	0.77 (0.56,1.04)	257 (32)	0.77 (0.64,0.93)
PFOA	246 (20)	1.53 (1.00,2.33)	150 (13)	0.88 (0.52,1.47)	257 (32)	1.57 (1.06,2.33)
PFHxS	246 (20)	0.90 (0.75,1.07)	150 (13)	0.83 (0.63,1.10)	257 (32)	0.87 (0.73,1.03)
Any kidney disease^						
PFOS	247 (22)	0.84 (0.69,1.01)	151 (17)	0.74 (0.60,0.93)	259 (34)	0.77 (0.64,0.92)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA	247 (22)	1.13 (0.66,1.91)	151 (17)	0.91 (0.58,1.44)	259 (34)	1.50 (1.03,2.18)
PFHxS	247 (22)	0.89 (0.76,1.04)	151 (17)	0.89 (0.68,1.15)	259 (34)	0.84 (0.71,1.00)
Asthma						
PFOS	247 (39)	0.99 (0.83,1.18)	151 (24)	1.01 (0.79,1.30)	258 (40)	1.11 (0.91,1.36)
PFOA	247 (39)	0.89 (0.69,1.14)	151 (24)	0.74 (0.55,1.00)	258 (40)	0.85 (0.59,1.23)
PFHxS	247 (39)	1.02 (0.88,1.19)	151 (24)	0.98 (0.77,1.24)	258 (40)	1.01 (0.83,1.24)
Rheumatoid arthritis						
PFOS	246 (11)	0.72 (0.46,1.12)	151 (10)	NC	258 (16)	0.64 (0.50,0.83)
PFOA	246 (11)	0.52 (0.30,0.91)	151 (10)	NC	258 (16)	0.62 (0.32,1.22)
PFHxS	246 (11)	0.87 (0.65,1.15)	151 (10)	NC	258 (16)	0.92 (0.70,1.20)
Any autoimmune disease^						
PFOS	247 (56)	0.87 (0.73,1.04)	151 (34)	0.96 (0.78,1.19)	258 (61)	0.86 (0.72,1.03)
PFOA	247 (56)	0.84 (0.68,1.03)	151 (34)	0.81 (0.64,1.02)	258 (61)	0.73 (0.56,0.96)
PFHxS	247 (56)	0.91 (0.79,1.04)	151 (34)	0.96 (0.80,1.15)	258 (61)	0.96 (0.82,1.11)
Type II diabetes						
PFOS	247 (13)	0.91 (0.68,1.22)	150 (12)	0.75 (0.54,1.06)	258 (19)	0.93 (0.64,1.36)
PFOA	247 (13)	0.86 (0.53,1.41)	150 (12)	0.81 (0.47,1.42)	258 (19)	0.63 (0.42,0.97)
PFHxS	247 (13)	1.00 (0.80,1.24)	150 (12)	0.79 (0.59,1.05)	258 (19)	1.03 (0.80,1.32)
Hypothyroidism						
PFOS	248 (6)	0.80 (0.53,1.21)	152 (7)	0.71 (0.46,1.09)	256 (8)	0.74 (0.49,1.11)
PFOA	248 (6)	2.27 (1.22,4.26)	152 (7)	0.87 (0.42,1.80)	256 (8)	1.18 (0.70,1.99)
PFHxS	248 (6)	0.63 (0.41,0.96)	152 (7)	0.84 (0.48,1.48)	256 (8)	0.98 (0.72,1.33)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	246 (8)	0.93 (0.73,1.18)	151 (5)	0.99 (0.63,1.57)	256 (8)	0.99 (0.72,1.38)
PFOA	246 (8)	0.69 (0.41,1.16)	151 (5)	0.96 (0.47,1.99)	256 (8)	0.94 (0.46,1.92)
PFHxS	246 (8)	0.92 (0.74,1.14)	151 (5)	1.03 (0.61,1.75)	256 (8)	0.77 (0.54,1.08)
Problems with fertility						
PFOS	210 (24)	0.96 (0.70,1.30)	121 (16)	1.11 (0.83,1.48)	223 (31)	0.84 (0.66,1.07)
PFOA	210 (24)	0.78 (0.55,1.11)	121 (16)	0.83 (0.60,1.15)	223 (31)	0.89 (0.65,1.24)
PFHxS	210 (24)	0.92 (0.73,1.17)	121 (16)	1.19 (0.90,1.57)	223 (31)	0.78 (0.63,0.97)
Early onset menopause						
PFOS	112 (9)	0.84 (0.56,1.25)#	40 (0)	NC	75 (7)	0.91 (0.59,1.42)
PFOA	112 (9)	0.58 (0.34,1.00)#	40 (0)	NC	75 (7)	1.13 (0.57,2.24)
PFHxS	112 (9)	0.97 (0.76,1.25)#	40 (0)	NC	75 (7)	1.12 (0.80,1.55)

N: sample size; NC: convergence not achieved; N/A: not applicable; PR: prevalence ratio; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table A1-7. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: excluding exposed participants who have not lived in the exposed communities in the last 10 years and past workers.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	235 (9)	0.65 (0.43,0.98)	114 (2)	NC	197 (3)	1.02 (0.74,1.42)
PFOA	235 (9)	0.52 (0.17,1.55)	114 (2)	NC	197 (3)	0.62 (0.18,2.11)
PFHxS	235 (9)	0.63 (0.44,0.89)	114 (2)	NC	197 (3)	0.97 (0.74,1.26)
Prostate cancer						
PFOS	127 (4)	NC	78 (2)	0.69 (0.40,1.19)	130 (6)	0.87 (0.58,1.30)
PFOA	127 (4)	NC	78 (2)	0.90 (0.35,2.29)	130 (6)	0.99 (0.42,2.31)
PFHxS	127 (4)	NC	78 (2)	0.52 (0.29,0.93)	130 (6)	0.67 (0.41,1.08)
Any cancer^						
PFOS	235 (18)	0.73 (0.53,1.00)	116 (7)	0.73 (0.49,1.08)	199 (12)	0.93 (0.75,1.17)
PFOA	235 (18)	0.62 (0.36,1.09)	116 (7)	1.03 (0.56,1.91)	199 (12)	0.91 (0.49,1.70)
PFHxS	235 (18)	0.74 (0.58,0.96)	116 (7)	0.73 (0.51,1.05)	199 (12)	0.69 (0.52,0.91)
Heart attack						
PFOS	237 (4)	NC	115 (9)	0.64 (0.40,1.02)	198 (12)	0.94 (0.67,1.31)
PFOA	237 (4)	NC	115 (9)	1.11 (0.59,2.09)	198 (12)	1.03 (0.52,2.02)
PFHxS	237 (4)	NC	115 (9)	0.88 (0.69,1.13)	198 (12)	0.81 (0.61,1.08)
High blood pressure						
PFOS	236 (48)	1.03 (0.88,1.20)	116 (29)	0.78 (0.61,0.99)	199 (57)	1.19 (1.02,1.38)
PFOA	236 (48)	0.93 (0.69,1.24)	116 (29)	0.87 (0.66,1.16)	199 (57)	0.98 (0.75,1.28)
PFHxS	236 (48)	0.93 (0.81,1.08)	116 (29)	0.90 (0.74,1.10)	199 (57)	1.01 (0.87,1.19)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	237 (49)	1.02 (0.87,1.19)	116 (30)	0.79 (0.63,0.99)	199 (59)	1.19 (1.03,1.38)
PFOA	237 (49)	0.92 (0.69,1.22)	116 (30)	0.87 (0.67,1.15)	199 (59)	0.99 (0.77,1.29)
PFHxS	237 (49)	0.92 (0.79,1.06)	116 (30)	0.91 (0.75,1.09)	199 (59)	1.01 (0.87,1.18)
Hypercholesterolaemia						
PFOS	237 (40)	1.04 (0.88,1.24)	114 (25)	0.95 (0.76,1.19)	198 (52)	1.04 (0.90,1.20)
PFOA	237 (40)	1.32 (0.98,1.79)	114 (25)	0.99 (0.75,1.31)	198 (52)	1.11 (0.86,1.45)
PFHxS	237 (40)	1.01 (0.88,1.18)	114 (25)	0.94 (0.78,1.13)	198 (52)	0.97 (0.84,1.11)
Fatty liver disease						
PFOS	236 (15)	0.90 (0.62,1.30)	115 (5)	0.85 (0.55,1.32)	198 (8)	1.02 (0.57,1.82)
PFOA	236 (15)	0.96 (0.66,1.41)	115 (5)	0.80 (0.45,1.41)	198 (8)	1.11 (0.68,1.80)
PFHxS	236 (15)	0.89 (0.64,1.25)	115 (5)	1.19 (0.81,1.74)	198 (8)	0.78 (0.52,1.18)
Any liver disease^						
PFOS	237 (20)	0.93 (0.68,1.28)	115 (5)	0.88 (0.59,1.31)	198 (10)	1.06 (0.65,1.74)
PFOA	237 (20)	0.99 (0.76,1.30)	115 (5)	0.99 (0.53,1.84)	198 (10)	1.03 (0.67,1.60)
PFHxS	237 (20)	0.99 (0.76,1.30)	115 (5)	1.18 (0.86,1.63)	198 (10)	0.77 (0.54,1.10)
Gout						
PFOS	236 (18)	0.75 (0.59,0.96)	114 (9)	0.68 (0.46,1.00)	197 (22)	0.72 (0.59,0.89)
PFOA	236 (18)	1.38 (0.87,2.19)	114 (9)	0.86 (0.36,2.01)	197 (22)	2.11 (1.24,3.61)
PFHxS	236 (18)	0.85 (0.68,1.04)	114 (9)	0.76 (0.50,1.15)	197 (22)	0.76 (0.59,0.98)
Any kidney disease^						
PFOS	237 (20)	0.80 (0.63,1.01)	115 (12)	0.66 (0.51,0.85)	199 (24)	0.72 (0.59,0.88)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA	237 (20)	1.01 (0.58,1.76)	115 (12)	0.84 (0.42,1.69)	199 (24)	1.92 (1.16,3.17)
PFHxS	237 (20)	0.84 (0.70,1.01)	115 (12)	0.75 (0.57,0.98)	199 (24)	0.73 (0.58,0.93)
Asthma						
PFOS	237 (35)	0.97 (0.81,1.16)	115 (20)	1.17 (0.93,1.47)	198 (28)	1.10 (0.88,1.38)
PFOA	237 (35)	0.85 (0.65,1.12)	115 (20)	1.01 (0.71,1.44)	198 (28)	0.79 (0.49,1.26)
PFHxS	237 (35)	0.99 (0.84,1.17)	115 (20)	1.15 (0.94,1.41)	198 (28)	1.02 (0.82,1.27)
Rheumatoid arthritis						
PFOS	236 (8)	0.74 (0.42,1.30)	115 (6)	NC	198 (13)	0.58 (0.46,0.73)
PFOA	236 (8)	0.35 (0.18,0.71)	115 (6)	NC	198 (13)	0.50 (0.20,1.25)
PFHxS	236 (8)	0.91 (0.68,1.22)	115 (6)	NC	198 (13)	0.87 (0.64,1.18)
Any autoimmune disease^						
PFOS	237 (50)	0.88 (0.74,1.05)	115 (27)	1.04 (0.84,1.29)	198 (41)	0.82 (0.67,1.01)
PFOA	237 (50)	0.82 (0.65,1.03)	115 (27)	0.97 (0.73,1.30)	198 (41)	0.70 (0.48,1.00)
PFHxS	237 (50)	0.90 (0.78,1.05)	115 (27)	1.05 (0.88,1.27)	198 (41)	0.94 (0.78,1.12)
Type II diabetes						
PFOS	237 (11)	0.81 (0.61,1.07)	114 (9)	0.78 (0.49,1.25)	198 (13)	0.94 (0.60,1.47)
PFOA	237 (11)	0.74 (0.43,1.28)	114 (9)	0.92 (0.46,1.85)	198 (13)	0.51 (0.29,0.89)
PFHxS	237 (11)	0.88 (0.70,1.11)	114 (9)	0.93 (0.67,1.30)	198 (13)	0.98 (0.73,1.31)
Hypothyroidism						
PFOS	238 (7)	0.75 (0.51,1.11)	116 (6)	0.68 (0.43,1.07)	196 (5)	1.00 (0.74,1.35)
PFOA	238 (7)	2.05 (1.14,3.69)	116 (6)	0.77 (0.30,1.98)	196 (5)	1.16 (0.56,2.38)
PFHxS	238 (7)	0.65 (0.43,1.00)	116 (6)	0.60 (0.38,0.94)	196 (5)	1.23 (0.91,1.65)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	236 (9)	0.88 (0.68,1.14)	115 (5)	0.98 (0.64,1.49)	196 (7)	1.13 (0.90,1.41)
PFOA	236 (9)	0.69 (0.41,1.15)	115 (5)	0.86 (0.48,1.54)	196 (7)	0.95 (0.41,2.18)
PFHxS	236 (9)	0.93 (0.74,1.16)	115 (5)	1.01 (0.59,1.71)	196 (7)	0.84 (0.55,1.29)
Problems with fertility						
PFOS	200 (24)	0.98 (0.73,1.32)	96 (15)	1.14 (0.85,1.52)	170 (23)	0.70 (0.56,0.89)
PFOA	200 (24)	0.78 (0.55,1.11)	96 (15)	0.86 (0.62,1.20)	170 (23)	0.63 (0.43,0.93)
PFHxS	200 (24)	0.93 (0.72,1.18)	96 (15)	1.23 (0.92,1.63)	170 (23)	0.75 (0.58,0.96)
Early onset menopause						
PFOS	114 (8)	0.84 (0.54,1.30)	37 (0)	NC	69 (6)	0.88 (0.50,1.57)#
PFOA	114 (8)	0.56 (0.31,1.01)	37 (0)	NC	69 (6)	0.98 (0.42,2.28)#
PFHxS	114 (8)	0.93 (0.69,1.25)	37 (0)	NC	69 (6)	1.17 (0.82,1.67)#

N: sample size; NC: convergence not achieved; N/A: not applicable; PR: prevalence ratio; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table A1-8. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: excluding participants under 25 years of age.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	268 (13)	0.71 (0.52,0.97)	156 (2)	NC	280 (7)	1.15 (0.73,1.83)
PFOA	268 (13)	0.65 (0.35,1.21)	156 (2)	NC	280 (7)	0.73 (0.30,1.78)
PFHxS	268 (13)	0.77 (0.62,0.94)	156 (2)	NC	280 (7)	1.19 (0.90,1.58)
Prostate cancer						
PFOS	145 (4)	NC	113 (3)	0.58 (0.36,0.94)	187 (10)	0.79 (0.46,1.34)
PFOA	145 (4)	NC	113 (3)	0.55 (0.30,1.02)	187 (10)	0.81 (0.32,2.03)
PFHxS	145 (4)	NC	113 (3)	0.55 (0.39,0.77)	187 (10)	0.72 (0.43,1.20)
Any cancer^						
PFOS	268 (22)	0.81 (0.63,1.05)	158 (9)	0.63 (0.44,0.91)	283 (22)	0.99 (0.74,1.32)
PFOA	268 (22)	0.75 (0.49,1.12)	158 (9)	0.74 (0.47,1.19)	283 (22)	0.95 (0.59,1.54)
PFHxS	268 (22)	0.86 (0.73,1.02)	158 (9)	0.67 (0.51,0.88)	283 (22)	0.86 (0.69,1.07)
Heart attack						
PFOS	270 (4)	NC	158 (14)	0.76 (0.55,1.06)	282 (19)	0.86 (0.65,1.14)
PFOA	270 (4)	NC	158 (14)	1.09 (0.76,1.57)	282 (19)	0.83 (0.54,1.28)
PFHxS	270 (4)	NC	158 (14)	0.93 (0.78,1.12)	282 (19)	0.90 (0.73,1.11)
High blood pressure						
PFOS	269 (61)	0.97 (0.85,1.11)	159 (40)	0.81 (0.66,0.99)	283 (80)	1.07 (0.93,1.22)
PFOA	269 (61)	0.91 (0.74,1.12)	159 (40)	0.87 (0.69,1.09)	283 (80)	0.95 (0.78,1.15)
PFHxS	269 (61)	0.93 (0.84,1.02)	159 (40)	0.94 (0.81,1.10)	283 (80)	1.02 (0.90,1.14)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	270 (62)	0.96 (0.84,1.10)	159 (41)	0.82 (0.67,0.99)	283 (83)	1.06 (0.93,1.21)
PFOA	270 (62)	0.90 (0.74,1.11)	159 (41)	0.87 (0.70,1.09)	283 (83)	0.95 (0.78,1.15)
PFHxS	270 (62)	0.92 (0.84,1.01)	159 (41)	0.95 (0.81,1.10)	283 (83)	1.02 (0.91,1.14)
Hypercholesterolaemia						
PFOS	270 (51)	1.09 (0.96,1.24)	157 (36)	1.00 (0.82,1.22)	282 (76)	1.03 (0.91,1.18)
PFOA	270 (51)	1.37 (1.09,1.73)	157 (36)	1.06 (0.81,1.39)	282 (76)	1.12 (0.92,1.38)
PFHxS	270 (51)	1.04 (0.93,1.16)	157 (36)	0.97 (0.83,1.14)	282 (76)	1.06 (0.94,1.19)
Fatty liver disease						
PFOS	269 (17)	1.06 (0.78,1.45)	158 (8)	1.13 (0.79,1.62)	282 (14)	0.88 (0.53,1.45)
PFOA	269 (17)	1.16 (0.79,1.69)	158 (8)	1.26 (0.74,2.16)	282 (14)	1.15 (0.78,1.69)
PFHxS	269 (17)	1.04 (0.81,1.35)	158 (8)	1.29 (0.98,1.69)	282 (14)	0.90 (0.67,1.21)
Any liver disease^						
PFOS	270 (23)	1.06 (0.83,1.36)	158 (9)	0.97 (0.70,1.36)	282 (17)	0.92 (0.60,1.42)
PFOA	270 (23)	1.14 (0.84,1.55)	158 (9)	1.31 (0.77,2.24)	282 (17)	1.07 (0.76,1.50)
PFHxS	270 (23)	1.10 (0.91,1.33)	158 (9)	1.20 (0.94,1.53)	282 (17)	0.90 (0.69,1.16)
Gout						
PFOS	269 (20)	0.89 (0.73,1.09)	157 (13)	0.76 (0.56,1.03)	281 (33)	0.74 (0.61,0.89)
PFOA	269 (20)	1.61 (1.09,2.37)	157 (13)	0.86 (0.52,1.41)	281 (33)	1.53 (1.05,2.24)
PFHxS	269 (20)	0.96 (0.82,1.13)	157 (13)	0.83 (0.63,1.09)	281 (33)	0.84 (0.69,1.01)
Any kidney disease^						
PFOS	270 (23)	0.93 (0.78,1.11)	158 (17)	0.74 (0.59,0.92)	283 (35)	0.74 (0.61,0.89)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA	270 (23)	1.35 (0.83,2.20)	158 (17)	0.89 (0.58,1.38)	283 (35)	1.47 (1.03,2.11)
PFHxS	270 (23)	0.95 (0.82,1.09)	158 (17)	0.88 (0.68,1.15)	283 (35)	0.82 (0.68,0.98)
Asthma						
PFOS	270 (42)	1.01 (0.84,1.20)	158 (25)	1.02 (0.80,1.30)	282 (41)	1.16 (0.95,1.42)
PFOA	270 (42)	0.87 (0.68,1.10)	158 (25)	0.78 (0.58,1.04)	282 (41)	0.92 (0.65,1.31)
PFHxS	270 (42)	1.02 (0.89,1.17)	158 (25)	1.00 (0.79,1.26)	282 (41)	1.04 (0.86,1.26)
Rheumatoid arthritis						
PFOS	269 (11)	0.78 (0.53,1.15)	158 (10)	NC	282 (17)	0.72 (0.49,1.05)
PFOA	269 (11)	0.57 (0.36,0.90)	158 (10)	NC	282 (17)	0.66 (0.34,1.26)
PFHxS	269 (11)	0.94 (0.75,1.17)	158 (10)	NC	282 (17)	0.99 (0.74,1.31)
Any autoimmune disease^						
PFOS	270 (59)	0.91 (0.78,1.07)	158 (36)	0.97 (0.80,1.19)	282 (64)	0.94 (0.77,1.13)
PFOA	270 (59)	0.83 (0.69,1.01)	158 (36)	0.85 (0.68,1.07)	282 (64)	0.80 (0.61,1.05)
PFHxS	270 (59)	0.94 (0.84,1.05)	158 (36)	0.97 (0.82,1.15)	282 (64)	1.01 (0.87,1.17)
Type II diabetes						
PFOS	270 (14)	0.86 (0.64,1.15)	157 (13)	0.73 (0.52,1.01)	283 (24)	0.92 (0.68,1.23)
PFOA	270 (14)	0.76 (0.51,1.13)	157 (13)	0.80 (0.49,1.29)	283 (24)	0.74 (0.50,1.09)
PFHxS	270 (14)	0.97 (0.78,1.20)	157 (13)	0.80 (0.61,1.06)	283 (24)	0.99 (0.80,1.22)
Hypothyroidism						
PFOS	271 (9)	0.82 (0.61,1.09)	159 (8)	0.77 (0.50,1.16)	280 (10)	0.76 (0.54,1.07)
PFOA	271 (9)	2.02 (1.25,3.27)	159 (8)	1.02 (0.49,2.09)	280 (10)	1.19 (0.72,1.97)
PFHxS	271 (9)	0.75 (0.58,0.96)	159 (8)	0.92 (0.56,1.52)	280 (10)	1.08 (0.84,1.40)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	269 (10)	0.92 (0.73,1.17)	158 (5)	1.00 (0.62,1.60)	281 (12)	1.04 (0.81,1.34)
PFOA	269 (10)	0.82 (0.52,1.28)	158 (5)	1.00 (0.48,2.06)	281 (12)	0.94 (0.56,1.57)
PFHxS	269 (10)	0.95 (0.77,1.16)	158 (5)	1.05 (0.62,1.80)	281 (12)	0.95 (0.72,1.27)
Problems with fertility						
PFOS	233 (28)	0.90 (0.64,1.27)	129 (16)	1.10 (0.81,1.50)	247 (36)	0.93 (0.73,1.17)
PFOA	233 (28)	0.83 (0.56,1.23)	129 (16)	0.88 (0.63,1.22)	247 (36)	0.94 (0.69,1.28)
PFHxS	233 (28)	0.90 (0.73,1.12)	129 (16)	1.22 (0.94,1.60)	247 (36)	0.82 (0.67,1.00)
Early onset menopause						
PFOS	130 (10)	0.92 (0.67,1.26)	44 (0)	NC	97 (9)	0.98 (0.71,1.37)
PFOA	130 (10)	0.64 (0.40,1.03)	44 (0)	NC	97 (9)	0.99 (0.54,1.83)
PFHxS	130 (10)	1.00 (0.80,1.25)	44 (0)	NC	97 (9)	1.00 (0.75,1.35)

N: sample size; NC: convergence not achieved; N/A: not applicable; PR: prevalence ratio; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table A1-9. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: PFAS serum concentrations below the limit of quantification imputed using multiple imputation by chained equations, rather than using a single plug-in value of the limit/sqrt(2).

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	278 (13)	0.71 (0.52,0.97)	158 (2)	NC	291 (7)	1.15 (0.72,1.83)
PFOA	278 (13)	0.64 (0.33,1.22)	158 (2)	NC	291 (7)	0.73 (0.30,1.77)
PFHxS	278 (13)	0.76 (0.62,0.94)	158 (2)	NC	291 (7)	1.19 (0.90,1.57)
Prostate cancer						
PFOS	151 (4)	NC	113 (3)	0.58 (0.36,0.94)	196 (10)	0.78 (0.44,1.36)
PFOA	151 (4)	NC	113 (3)	0.55 (0.28,1.05)	196 (10)	0.81 (0.32,2.03)
PFHxS	151 (4)	NC	113 (3)	0.55 (0.39,0.80)	196 (10)	0.72 (0.43,1.19)
Any cancer^						
PFOS	278 (22)	0.82 (0.63,1.05)	160 (9)	0.63 (0.44,0.91)	294 (22)	1.00 (0.74,1.34)
PFOA	278 (22)	0.74 (0.48,1.12)	160 (9)	0.75 (0.46,1.22)	294 (22)	0.96 (0.59,1.57)
PFHxS	278 (22)	0.87 (0.73,1.03)	160 (9)	0.68 (0.52,0.89)	294 (22)	0.87 (0.70,1.08)
Heart attack						
PFOS	280 (4)	NC	160 (14)	0.76 (0.55,1.06)	293 (19)	0.86 (0.65,1.15)
PFOA	280 (4)	NC	160 (14)	1.09 (0.75,1.57)	293 (19)	0.83 (0.54,1.28)
PFHxS	280 (4)	NC	160 (14)	0.93 (0.78,1.12)	293 (19)	0.90 (0.73,1.11)
High blood pressure						
PFOS	279 (61)	0.97 (0.85,1.11)	161 (40)	0.81 (0.66,0.99)	294 (80)	1.07 (0.93,1.22)
PFOA	279 (61)	0.90 (0.73,1.11)	161 (40)	0.86 (0.68,1.09)	294 (80)	0.95 (0.78,1.15)
PFHxS	279 (61)	0.93 (0.84,1.02)	161 (40)	0.94 (0.81,1.10)	294 (80)	1.02 (0.91,1.14)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	280 (62)	0.96 (0.84,1.10)	161 (41)	0.82 (0.67,0.99)	294 (83)	1.06 (0.93,1.21)
PFOA	280 (62)	0.90 (0.74,1.10)	161 (41)	0.87 (0.69,1.09)	294 (83)	0.95 (0.78,1.15)
PFHxS	280 (62)	0.92 (0.84,1.02)	161 (41)	0.95 (0.81,1.10)	294 (83)	1.02 (0.91,1.14)
Hypercholesterolaemia						
PFOS	280 (51)	1.09 (0.95,1.24)	159 (36)	1.00 (0.82,1.22)	293 (76)	1.04 (0.91,1.18)
PFOA	280 (51)	1.36 (1.08,1.71)	159 (36)	1.06 (0.80,1.40)	293 (76)	1.13 (0.92,1.38)
PFHxS	280 (51)	1.04 (0.93,1.16)	159 (36)	0.97 (0.83,1.14)	293 (76)	1.06 (0.94,1.19)
Fatty liver disease						
PFOS	279 (18)	1.04 (0.78,1.40)	160 (8)	1.13 (0.79,1.62)	293 (14)	0.88 (0.53,1.46)
PFOA	279 (18)	1.14 (0.79,1.65)	160 (8)	1.26 (0.74,2.17)	293 (14)	1.14 (0.78,1.68)
PFHxS	279 (18)	1.02 (0.80,1.30)	160 (8)	1.29 (0.98,1.70)	293 (14)	0.91 (0.68,1.21)
Any liver disease^						
PFOS	280 (24)	1.05 (0.82,1.34)	160 (9)	0.97 (0.70,1.36)	293 (17)	0.92 (0.59,1.43)
PFOA	280 (24)	1.15 (0.84,1.57)	160 (9)	1.31 (0.76,2.25)	293 (17)	1.07 (0.76,1.50)
PFHxS	280 (24)	1.08 (0.89,1.32)	160 (9)	1.20 (0.94,1.53)	293 (17)	0.90 (0.70,1.16)
Gout						
PFOS	279 (20)	0.90 (0.74,1.09)	159 (13)	0.76 (0.56,1.03)	292 (33)	0.74 (0.60,0.90)
PFOA	279 (20)	1.61 (1.09,2.38)	159 (13)	0.86 (0.51,1.44)	292 (33)	1.54 (1.06,2.24)
PFHxS	279 (20)	0.96 (0.82,1.13)	159 (13)	0.83 (0.63,1.09)	292 (33)	0.84 (0.70,1.01)
Any kidney disease^						
PFOS	280 (23)	0.93 (0.78,1.11)	160 (17)	0.74 (0.59,0.92)	294 (35)	0.74 (0.61,0.90)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA	280 (23)	1.36 (0.83,2.23)	160 (17)	0.89 (0.57,1.40)	294 (35)	1.48 (1.04,2.12)
PFHxS	280 (23)	0.95 (0.82,1.09)	160 (17)	0.88 (0.68,1.15)	294 (35)	0.82 (0.69,0.98)
Asthma						
PFOS	280 (44)	1.00 (0.84,1.19)	160 (25)	1.02 (0.80,1.31)	293 (43)	1.16 (0.96,1.42)
PFOA	280 (44)	0.85 (0.66,1.08)	160 (25)	0.78 (0.57,1.07)	293 (43)	0.93 (0.65,1.32)
PFHxS	280 (44)	1.03 (0.90,1.18)	160 (25)	1.01 (0.80,1.27)	293 (43)	1.05 (0.88,1.27)
Rheumatoid arthritis						
PFOS	279 (11)	0.78 (0.53,1.15)	160 (10)	NC	293 (17)	0.72 (0.48,1.08)
PFOA	279 (11)	0.56 (0.35,0.90)	160 (10)	NC	293 (17)	0.66 (0.34,1.26)
PFHxS	279 (11)	0.94 (0.75,1.17)	160 (10)	NC	293 (17)	0.99 (0.74,1.31)
Any autoimmune disease^						
PFOS	280 (61)	0.91 (0.78,1.06)	160 (36)	0.97 (0.80,1.19)	293 (66)	0.94 (0.78,1.13)
PFOA	280 (61)	0.82 (0.67,1.00)	160 (36)	0.85 (0.67,1.08)	293 (66)	0.81 (0.62,1.05)
PFHxS	280 (61)	0.95 (0.85,1.07)	160 (36)	0.97 (0.82,1.16)	293 (66)	1.02 (0.88,1.17)
Type II diabetes						
PFOS	280 (14)	0.85 (0.64,1.15)	159 (13)	0.73 (0.52,1.01)	294 (24)	0.92 (0.68,1.25)
PFOA	280 (14)	0.75 (0.50,1.14)	159 (13)	0.79 (0.49,1.30)	294 (24)	0.74 (0.50,1.09)
PFHxS	280 (14)	0.97 (0.78,1.20)	159 (13)	0.81 (0.61,1.07)	294 (24)	0.99 (0.80,1.23)
Hypothyroidism						
PFOS	281 (9)	0.83 (0.63,1.10)	161 (8)	0.76 (0.50,1.16)	291 (10)	0.75 (0.52,1.07)
PFOA	281 (9)	2.13 (1.30,3.47)	161 (8)	1.00 (0.48,2.08)	291 (10)	1.18 (0.71,1.97)
PFHxS	281 (9)	0.75 (0.58,0.97)	161 (8)	0.92 (0.55,1.53)	291 (10)	1.08 (0.83,1.39)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	279 (10)	0.92 (0.73,1.16)	160 (5)	1.00 (0.62,1.60)	292 (12)	1.06 (0.82,1.37)
PFOA	279 (10)	0.77 (0.48,1.24)	160 (5)	0.99 (0.48,2.07)	292 (12)	0.93 (0.55,1.58)
PFHxS	279 (10)	0.95 (0.77,1.16)	160 (5)	1.05 (0.61,1.80)	292 (12)	0.95 (0.71,1.27)
Problems with fertility						
PFOS	236 (28)	0.90 (0.64,1.27)	130 (16)	1.10 (0.81,1.50)	251 (37)	0.90 (0.71,1.14)
PFOA	236 (28)	0.80 (0.54,1.21)	130 (16)	0.86 (0.62,1.21)	251 (37)	0.92 (0.68,1.26)
PFHxS	236 (28)	0.90 (0.73,1.12)	130 (16)	1.22 (0.93,1.61)	251 (37)	0.83 (0.68,1.01)
Early onset menopause						
PFOS	134 (10)	0.91 (0.67,1.25)	46 (0)	NC	99 (9)	0.98 (0.70,1.37)
PFOA	134 (10)	0.61 (0.38,1.00)	46 (0)	NC	99 (9)	0.99 (0.53,1.82)
PFHxS	134 (10)	1.00 (0.80,1.25)	46 (0)	NC	99 (9)	1.00 (0.74,1.35)

Among survey respondents, detection rates were 99.5-100% for PFOS, 97.8-99.5% for PFOA, and 93.6-96.2% for PFHxS.

N: sample size; NC: convergence not achieved; N/A: not applicable; PR: prevalence ratio; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Table A1-10. Adjusted prevalence ratios of self-reported health outcomes per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: missing values in confounding variables imputed using multiple imputation by chained equations.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Breast cancer						
PFOS	365 (15)	0.72 (0.53,0.99)	204 (3)	0.77 (0.51,1.15)	399 (9)	1.16 (0.77,1.73)
PFOA	365 (15)	0.82 (0.42,1.61)	204 (3)	1.01 (0.64,1.61)	399 (9)	0.73 (0.35,1.54)
PFHxS	365 (15)	0.78 (0.63,0.97)	204 (3)	0.80 (0.59,1.09)	399 (9)	1.14 (0.89,1.47)
Prostate cancer						
PFOS	196 (4)	1.28 (0.82,2.00)	147 (6)	0.60 (0.38,0.96)	267 (12)	0.64 (0.43,0.97)
PFOA	196 (4)	0.63 (0.33,1.22)	147 (6)	0.60 (0.40,0.88)	267 (12)	0.68 (0.32,1.44)
PFHxS	196 (4)	1.34 (0.88,2.05)	147 (6)	0.67 (0.48,0.92)	267 (12)	0.59 (0.38,0.92)
Any cancer^						
PFOS	365 (25)	0.85 (0.67,1.09)	207 (13)	0.66 (0.48,0.90)	402 (32)	0.89 (0.69,1.14)
PFOA	365 (25)	0.89 (0.57,1.37)	207 (13)	0.75 (0.50,1.13)	402 (32)	0.81 (0.56,1.17)
PFHxS	365 (25)	0.90 (0.75,1.08)	207 (13)	0.72 (0.57,0.91)	402 (32)	0.87 (0.72,1.06)
Heart attack						
PFOS	367 (9)	NC	206 (19)	0.86 (0.63,1.19)	399 (28)	0.98 (0.76,1.26)
PFOA	367 (9)	NC	206 (19)	1.31 (0.86,1.99)	399 (28)	1.06 (0.72,1.55)
PFHxS	367 (9)	NC	206 (19)	0.96 (0.79,1.16)	399 (28)	1.03 (0.84,1.26)
High blood pressure						
PFOS	367 (85)	0.96 (0.84,1.09)	208 (58)	0.90 (0.71,1.15)	405 (116)	1.07 (0.97,1.18)
PFOA	367 (85)	0.93 (0.77,1.12)	208 (58)	0.88 (0.71,1.09)	405 (116)	1.05 (0.90,1.24)
PFHxS	367 (85)	0.92 (0.83,1.01)	208 (58)	0.97 (0.85,1.11)	405 (116)	1.06 (0.97,1.15)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Any cardiovascular disease^						
PFOS	368 (90)	0.97 (0.86,1.10)	208 (61)	0.93 (0.74,1.17)	405 (122)	1.08 (0.98,1.18)
PFOA	368 (90)	0.96 (0.80,1.15)	208 (61)	0.91 (0.74,1.13)	405 (122)	1.07 (0.92,1.25)
PFHxS	368 (90)	0.93 (0.85,1.02)	208 (61)	0.98 (0.86,1.12)	405 (122)	1.07 (0.98,1.16)
Hypercholesterolaemia						
PFOS	367 (74)	1.07 (0.94,1.22)	204 (55)	1.05 (0.92,1.20)	402 (110)	1.07 (0.96,1.19)
PFOA	367 (74)	1.23 (1.00,1.52)	204 (55)	1.09 (0.86,1.39)	402 (110)	1.12 (0.94,1.34)
PFHxS	367 (74)	1.03 (0.93,1.15)	204 (55)	1.00 (0.89,1.13)	402 (110)	1.07 (0.97,1.19)
Fatty liver disease						
PFOS	364 (24)	0.93 (0.70,1.24)	204 (11)	0.90 (0.69,1.17)	401 (24)	0.87 (0.61,1.22)
PFOA	364 (24)	0.97 (0.69,1.35)	204 (11)	0.98 (0.59,1.64)	401 (24)	1.15 (0.80,1.65)
PFHxS	364 (24)	0.97 (0.79,1.20)	204 (11)	0.96 (0.73,1.25)	401 (24)	0.96 (0.77,1.19)
Any liver disease^						
PFOS	365 (32)	0.93 (0.73,1.19)	204 (12)	0.86 (0.66,1.13)	401 (29)	0.84 (0.62,1.15)
PFOA	365 (32)	0.99 (0.74,1.34)	204 (12)	1.02 (0.63,1.64)	401 (29)	1.05 (0.77,1.45)
PFHxS	365 (32)	1.03 (0.87,1.22)	204 (12)	0.95 (0.75,1.22)	401 (29)	0.96 (0.79,1.16)
Gout						
PFOS	364 (25)	0.87 (0.70,1.07)	203 (18)	0.83 (0.58,1.18)	398 (39)	0.74 (0.61,0.90)
PFOA	364 (25)	1.35 (0.90,2.03)	203 (18)	0.89 (0.52,1.55)	398 (39)	1.37 (0.94,2.01)
PFHxS	364 (25)	0.91 (0.77,1.07)	203 (18)	0.82 (0.64,1.05)	398 (39)	0.82 (0.68,0.97)
Any kidney disease^						
PFOS	365 (28)	0.89 (0.73,1.08)	204 (22)	0.78 (0.59,1.03)	402 (43)	0.73 (0.62,0.88)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PFOA	365 (28)	1.24 (0.79,1.95)	204 (22)	0.92 (0.56,1.51)	402 (43)	1.17 (0.81,1.67)
PFHxS	365 (28)	0.89 (0.76,1.04)	204 (22)	0.86 (0.68,1.09)	402 (43)	0.80 (0.68,0.95)
Asthma						
PFOS	364 (56)	0.97 (0.82,1.15)	203 (32)	1.08 (0.88,1.32)	399 (62)	1.07 (0.91,1.25)
PFOA	364 (56)	0.84 (0.67,1.04)	203 (32)	0.87 (0.62,1.23)	399 (62)	0.90 (0.68,1.18)
PFHxS	364 (56)	1.03 (0.90,1.18)	203 (32)	1.09 (0.89,1.34)	399 (62)	1.02 (0.87,1.19)
Rheumatoid arthritis						
PFOS	363 (16)	0.82 (0.59,1.13)	203 (11)	0.89 (0.65,1.21)	400 (31)	0.88 (0.64,1.21)
PFOA	363 (16)	0.81 (0.48,1.36)	203 (11)	0.64 (0.38,1.08)	400 (31)	0.75 (0.51,1.11)
PFHxS	363 (16)	0.91 (0.75,1.12)	203 (11)	0.90 (0.64,1.28)	400 (31)	1.01 (0.83,1.24)
Any autoimmune disease^						
PFOS	365 (80)	0.91 (0.78,1.05)	203 (45)	1.03 (0.86,1.22)	401 (98)	0.93 (0.81,1.06)
PFOA	365 (80)	0.85 (0.70,1.01)	203 (45)	0.93 (0.71,1.22)	401 (98)	0.80 (0.66,0.98)
PFHxS	365 (80)	0.96 (0.85,1.07)	203 (45)	1.04 (0.89,1.23)	401 (98)	0.99 (0.88,1.11)
Type II diabetes						
PFOS	365 (17)	0.87 (0.64,1.20)	203 (20)	0.92 (0.62,1.38)	403 (36)	0.96 (0.76,1.22)
PFOA	365 (17)	0.77 (0.50,1.18)	203 (20)	0.85 (0.56,1.30)	403 (36)	0.77 (0.59,1.02)
PFHxS	365 (17)	0.97 (0.78,1.22)	203 (20)	0.89 (0.71,1.12)	403 (36)	1.04 (0.86,1.25)
Hypothyroidism						
PFOS	367 (16)	0.82 (0.64,1.06)	204 (10)	0.80 (0.56,1.14)	397 (14)	0.68 (0.51,0.91)
PFOA	367 (16)	1.40 (0.85,2.31)#	204 (10)	0.90 (0.50,1.64)	397 (14)	1.05 (0.67,1.63)
PFHxS	367 (16)	0.94 (0.72,1.22)#	204 (10)	0.87 (0.57,1.32)	397 (14)	0.93 (0.75,1.16)

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
Hyperthyroidism						
PFOS	361 (14)	0.95 (0.77,1.16)	203 (9)	1.09 (0.79,1.51)	399 (16)	0.95 (0.76,1.19)
PFOA	361 (14)	0.87 (0.58,1.30)	203 (9)	0.93 (0.60,1.43)	399 (16)	0.92 (0.58,1.47)
PFHxS	361 (14)	1.05 (0.87,1.28)	203 (9)	1.24 (0.88,1.74)	399 (16)	0.93 (0.73,1.20)
Problems with fertility						
PFOS	308 (30)	0.90 (0.64,1.25)	166 (21)	1.14 (0.91,1.43)	336 (40)	0.96 (0.77,1.20)
PFOA	308 (30)	0.78 (0.53,1.15)	166 (21)	0.95 (0.67,1.35)	336 (40)	0.99 (0.72,1.35)
PFHxS	308 (30)	0.94 (0.76,1.16)	166 (21)	1.19 (0.96,1.49)	336 (40)	0.89 (0.73,1.08)
Early onset menopause						
PFOS	181 (17)	0.82 (0.60,1.12)	59 (1)	0.70 (0.50,0.99)	138 (12)	0.80 (0.60,1.09)
PFOA	181 (17)	0.65 (0.42,1.00)	59 (1)	0.71 (0.44,1.14)	138 (12)	0.92 (0.49,1.71)
PFHxS	181 (17)	0.95 (0.78,1.15)	59 (1)	1.01 (0.78,1.30)	138 (12)	0.89 (0.71,1.13)

N: sample size; NC: convergence not achieved; N/A: not applicable; PR: prevalence ratio; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income. Age was modelled using a restricted cubic spline with 3 knots.

^ Any cancer includes bone, brain, breast, kidney, leukaemia, liver, ovarian, prostate, testicular, and thyroid cancers. Any cardiovascular disease includes heart attack, high blood pressure, and stroke. Any liver disease includes non-infectious hepatitis, fatty liver disease, and cirrhosis of the liver. Any kidney disease includes gout and chronic kidney disease. Any autoimmune disease includes asthma, rheumatoid arthritis, lupus, ulcerative colitis, Crohn's disease, and multiple sclerosis. Type I diabetes and neurological outcomes were not analysed due to low prevalence.

Appendix 2: Sensitivity analysis for psychological distress outcomes

Table A2-1. Adjusted differences in mean self-reported psychological distress scores between participants from PFAS Management Areas, 2019–2020, and comparison communities, 2020.

	Katherine vs. Alice Springs (NT)		Oakey vs. Dalby (Qld)		Williamtown vs. Kiama and Shellharbour (NSW)	
	Exposed N; Comparison N	Adjusted difference (95% CI)†	Exposed N; Comparison N	Adjusted difference (95% CI)†	Exposed N; Comparison N	Adjusted difference (95% CI)†
PHQ-15 score	281; 180	2.5 (1.8,3.3)	164; 133	3.0 (1.9,4.1)	293; 345	2.1 (1.4,2.9)
K6 score	291; 184	1.0 (0.3,1.6)#	171; 140	2.1 (1.1,3.0)	300; 349	1.1 (0.4,1.7)#
DQ5 score	293; 184	1.1 (0.4,1.8)	172; 140	2.3 (1.4,3.1)	300; 347	1.2 (0.5,1.9)
GAD-7 score	291; 184	0.9 (0.3,1.6)#	172; 138	2.0 (1.0,3.0)	301; 349	0.8 (0.2,1.5)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income.

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-2. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores in participants from PFAS Management Areas, 2019–2020, versus comparison communities, 2020. Sensitivity analysis: additional adjustment for marital status.

	Katherine vs. Alice Springs (NT)		Oakey vs. Dalby (Qld)		Williamtown vs. Kiama and Shellharbour (NSW)	
	Exposed N (cases); Comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); Comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); Comparison N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10	280 (71); 180 (12)	3.76 (2.12,6.68)	164 (43); 133 (22)	1.91 (1.23,2.97)	293 (72); 345 (39)	2.26 (1.56,3.28)
K6 score ≥ 13	290 (61); 184 (21)	1.76 (1.12,2.76)	170 (37); 140 (19)	1.95 (1.17,3.26)	300 (62); 349 (40)	1.59 (1.07,2.37)
DQ5 score ≥ 14	292 (56); 184 (24)	1.40 (0.92,2.12)	172 (38); 140 (11)	3.60 (1.92,6.72)	300 (54); 347 (43)	1.29 (0.87,1.91)
GAD-7 score ≥ 10	290 (28); 184 (5)	2.85 (1.18,6.89)	171 (21); 138 (6)	3.46 (1.33,9.01)	301 (28); 349 (21)	1.24 (0.67,2.29)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, and marital status.

Table A2-3. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores in participants from PFAS Management Areas, 2019–2020, versus comparison communities, 2020. Sensitivity analysis: excluding exposed participants who now live in comparison communities.

	Katherine vs. Alice Springs (NT)		Oakey vs. Dalby (Qld)		Williamtown vs. Kiama and Shellharbour (NSW)	
	Exposed N (cases); Comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); Comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); Comparison N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10	263 (69); 180 (12)	3.82 (2.13,6.86)	132 (36); 133 (22)	2.11 (1.33,3.35)	282 (70); 345 (39)	2.33 (1.60,3.39)
K6 score ≥ 13	273 (56); 184 (21)	1.66 (1.06,2.60)	136 (29); 140 (19)	2.31 (1.33,4.01)	289 (59); 349 (40)	1.58 (1.05,2.37)
DQ5 score ≥ 14	275 (52); 184 (24)	1.28 (0.84,1.97)	138 (30); 140 (11)	3.87 (2.09,7.17)	289 (52); 347 (43)	1.27 (0.85,1.91)
GAD-7 score ≥ 10	273 (27); 184 (5)	2.89 (1.18,7.05)	137 (17); 138 (6)	4.33 (1.65,11.37)	290 (26); 349 (21)	1.19 (0.63,2.24)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income.

Table A2-4. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores in participants from PFAS Management Areas, 2019–2020, versus comparison communities, 2020. Sensitivity analysis: missing values in confounding variables imputed using multiple imputation by chained equations.

	Katherine vs. Alice Springs (NT)		Oakey vs. Dalby (Qld)		Williamtown vs. Kiama and Shellharbour (NSW)	
	Exposed N (cases); Comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); Comparison N (cases)	Adjusted PR (95% CI)†	Exposed N (cases); Comparison N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10	356 (84); 198 (16)	2.84 (1.71,4.74)	205 (58); 154 (27)	1.82 (1.21,2.74)	396 (105); 415 (50)	2.31 (1.69,3.17)
K6 score ≥ 13	372 (77); 203 (25)	1.53 (1.03,2.28)	216 (56); 164 (22)	2.20 (1.38,3.48)	413 (92); 425 (49)	1.77 (1.27,2.48)
DQ5 score ≥ 14	378 (75); 203 (29)	1.24 (0.85,1.81)	217 (60); 164 (13)	4.28 (2.44,7.52)	411 (85); 423 (52)	1.54 (1.10,2.15)
GAD-7 score ≥ 10	374 (36); 203 (8)	1.92 (0.93,3.97)	217 (38); 162 (8)	4.22 (1.95,9.13)#	412 (47); 426 (26)	1.63 (0.99,2.70)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income.

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-5. Adjusted differences in mean self-reported psychological distress scores between participants from PFAS Management Areas, 2019–2020, and comparison communities, 2020. Sensitivity analysis: additional adjustment for marital status.

	Katherine vs. Alice Springs (NT)		Oakey vs. Dalby (Qld)		Williamtown vs. Kiama and Shellharbour (NSW)	
	Exposed N; Comparison N	Adjusted difference (95% CI)†	Exposed N; Comparison N	Adjusted difference (95% CI)†	Exposed N; Comparison N	Adjusted difference (95% CI)†
PHQ-15 score	280; 180	2.5 (1.8,3.3)	164; 133	3.1 (2.0,4.2)	293; 345	2.1 (1.4,2.8)
K6 score	290; 184	1.1 (0.4,1.7)#	170; 140	2.1 (1.1,3.1)	300; 349	1.1 (0.4,1.8)#
DQ5 score	292; 184	1.3 (0.6,2.0)	172; 140	2.3 (1.4,3.1)	300; 347	1.2 (0.6,1.9)
GAD-7 score	290; 184	1.0 (0.3,1.7)#	171; 138	2.1 (1.1,3.0)	301; 349	0.8 (0.2,1.5)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, and marital status.

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-6. Adjusted differences in mean self-reported psychological distress scores between participants from PFAS Management Areas, 2019–2020, and comparison communities, 2020. Sensitivity analysis: excluding exposed participants who now live in comparison communities.

	Katherine vs. Alice Springs (NT)		Oakey vs. Dalby (Qld)		Williamtown vs. Kiama and Shellharbour (NSW)	
	Exposed N; Comparison N	Adjusted difference (95% CI)†	Exposed N; Comparison N	Adjusted difference (95% CI)†	Exposed N; Comparison N	Adjusted difference (95% CI)†
PHQ-15 score	263; 180	2.7 (1.9,3.5)	132; 133	3.2 (2.0,4.4)	282; 345	2.2 (1.5,2.9)
K6 score	273; 184	1.0 (0.3,1.6)#	136; 140	2.2 (1.1,3.2)	289; 349	1.0 (0.4,1.7)#
DQ5 score	275; 184	1.1 (0.4,1.8)	138; 140	2.6 (1.7,3.5)	289; 347	1.2 (0.5,1.9)
GAD-7 score	273; 184	1.0 (0.3,1.7)#	137; 138	2.2 (1.1,3.3)	290; 349	0.8 (0.2,1.5)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income.

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-7. Adjusted differences in mean self-reported psychological distress scores between participants from PFAS Management Areas, 2019–2020, and comparison communities, 2020. Sensitivity analysis: missing values in confounding variables imputed using multiple imputation by chained equations.

	Katherine vs. Alice Springs (NT)		Oakey vs. Dalby (Qld)		Williamtown vs. Kiama and Shellharbour (NSW)	
	Exposed N; Comparison N	Adjusted difference (95% CI)†	Exposed N; Comparison N	Adjusted difference (95% CI)†	Exposed N; Comparison N	Adjusted difference (95% CI)†
PHQ-15 score	356; 198	2.3 (1.6,3.1)	205; 154	3.1 (2.0,4.1)	396; 415	2.3 (1.6,2.9)
K6 score	372; 203	0.8 (0.2,1.4)#	216; 164	3.0 (1.4,4.5)	413; 425	1.5 (0.8,2.3)
DQ5 score	378; 203	1.1 (0.4,1.7)	217; 164	2.7 (1.8,3.5)	411; 423	1.3 (0.7,1.9)
GAD-7 score	374; 203	0.9 (0.2,1.5)#	217; 162	2.7 (1.7,3.8)#	412; 426	1.1 (0.5,1.8)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, and gross household annual income.

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-8. Adjusted differences in mean self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†
PHQ-15 score						
PFOS	260	-0.1 (-0.5,0.4)	149	-0.7 (-1.3,0.0)	280	-0.1 (-0.6,0.3)
PFOA	260	-0.7 (-1.5,0.0)	149	-0.6 (-1.3,0.2)	280	-0.4 (-1.2,0.3)
PFHxS	260	-0.1 (-0.4,0.3)	149	-0.2 (-0.7,0.4)	280	-0.0 (-0.4,0.4)
K6 score						
PFOS	268	-0.3 (-0.7,0.0)#	156	-0.3 (-0.8,0.3)	284	-0.0 (-0.4,0.3)#
PFOA	268	-0.7 (-1.4,-0.1)#	156	-0.5 (-1.2,0.2)	284	-0.3 (-0.9,0.3)#
PFHxS	268	-0.2 (-0.5,0.1)#	156	-0.1 (-0.5,0.3)	284	-0.2 (-0.5,0.1)#
DQ5 score						
PFOS	270	-0.2 (-0.6,0.2)	157	-0.4 (-1.0,0.1)	284	-0.0 (-0.4,0.4)
PFOA	270	-0.4 (-1.0,0.2)	157	-0.3 (-1.0,0.4)	284	0.1 (-0.5,0.7)
PFHxS	270	-0.1 (-0.4,0.2)	157	-0.1 (-0.5,0.3)	284	-0.0 (-0.4,0.3)
GAD-7 score						
PFOS	267	-0.2 (-0.6,0.1)#	157	-0.2 (-0.9,0.4)	285	0.1 (-0.3,0.5)
PFOA	267	-0.7 (-1.3,-0.0)#	157	-0.4 (-1.0,0.2)	285	0.2 (-0.5,0.9)
PFHxS	267	-0.2 (-0.5,0.2)#	157	-0.0 (-0.5,0.4)	285	0.0 (-0.3,0.4)

N: sample size; DQ5: Distress Questionnaire-5; K6: Kessler distress scale-6; GAD-7: Generalised Anxiety Disorder assessment-7; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), and occupational AFFF exposure (yes vs. no).

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-9. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: additional adjustment for smoking status and alcohol consumption.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10						
PFOS	248 (59)	0.96 (0.82,1.12)	145 (32)	0.81 (0.58,1.12)	271 (66)	0.91 (0.77,1.07)
PFOA	248 (59)	0.90 (0.72,1.13)	145 (32)	0.85 (0.64,1.13)	271 (66)	0.97 (0.77,1.22)
PFHxS	248 (59)	0.98 (0.86,1.12)	145 (32)	0.99 (0.79,1.24)	271 (66)	1.00 (0.88,1.13)
K6 score ≥13						
PFOS	256 (47)	0.96 (0.78,1.19)	149 (30)	0.86 (0.64,1.16)	275 (56)	0.98 (0.83,1.15)#
PFOA	256 (47)	0.75 (0.57,0.97)	149 (30)	0.82 (0.61,1.10)	275 (56)	0.87 (0.66,1.16)#
PFHxS	256 (47)	0.95 (0.81,1.11)	149 (30)	0.91 (0.75,1.12)	275 (56)	0.91 (0.78,1.05)#
DQ5 score ≥14						
PFOS	258 (43)	0.96 (0.78,1.19)	151 (30)	0.77 (0.59,1.02)	275 (48)	1.00 (0.84,1.19)
PFOA	258 (43)	0.81 (0.60,1.10)	151 (30)	0.89 (0.64,1.25)	275 (48)	1.06 (0.77,1.46)
PFHxS	258 (43)	0.93 (0.80,1.08)	151 (30)	0.90 (0.72,1.12)	275 (48)	0.93 (0.82,1.06)
GAD-7 score ≥10						
PFOS	256 (23)	0.96 (0.72,1.30)	150 (16)	1.01 (0.64,1.60)	276 (26)	0.94 (0.71,1.23)#
PFOA	256 (23)	0.80 (0.50,1.26)	150 (16)	0.89 (0.57,1.37)	276 (26)	1.00 (0.60,1.67)
PFHxS	256 (23)	0.89 (0.71,1.12)	150 (16)	1.01 (0.68,1.51)	276 (26)	0.91 (0.72,1.15)#

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), occupational AFFF exposure (yes vs. no), smoking status, and alcohol consumption.

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-10. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: additional adjustment for marital status.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10						
PFOS	259 (65)	0.93 (0.80,1.08)	149 (33)	0.79 (0.59,1.06)	280 (70)	0.89 (0.75,1.06)
PFOA	259 (65)	0.83 (0.68,1.02)	149 (33)	0.81 (0.62,1.06)	280 (70)	0.92 (0.73,1.15)
PFHxS	259 (65)	0.97 (0.86,1.10)	149 (33)	0.98 (0.80,1.20)	280 (70)	0.98 (0.85,1.12)
K6 score ≥13						
PFOS	267 (54)	0.95 (0.78,1.15)	155 (31)	0.85 (0.63,1.14)	284 (59)	0.99 (0.83,1.17)#
PFOA	267 (54)	0.71 (0.58,0.89)	155 (31)	0.78 (0.58,1.04)	284 (59)	0.87 (0.66,1.14)#
PFHxS	267 (54)	0.95 (0.82,1.11)	155 (31)	0.89 (0.72,1.10)	284 (59)	0.91 (0.78,1.06)#
DQ5 score ≥14						
PFOS	269 (49)	0.95 (0.78,1.16)	157 (32)	0.74 (0.57,0.96)	284 (50)	1.05 (0.88,1.24)
PFOA	269 (49)	0.75 (0.59,0.97)	157 (32)	0.83 (0.57,1.20)	284 (50)	1.05 (0.76,1.45)
PFHxS	269 (49)	0.94 (0.81,1.10)	157 (32)	0.87 (0.71,1.07)	284 (50)	0.96 (0.83,1.10)
GAD-7 score ≥10						
PFOS	266 (25)	0.82 (0.60,1.10)	156 (16)	0.98 (0.63,1.51)	285 (26)	0.94 (0.71,1.25)
PFOA	266 (25)	0.72 (0.46,1.11)	156 (16)	0.86 (0.56,1.31)	285 (26)	1.07 (0.61,1.89)
PFHxS	266 (25)	0.81 (0.65,1.01)	156 (16)	0.98 (0.67,1.42)	285 (26)	0.90 (0.70,1.15)

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), occupational AFFF exposure (yes vs. no), and marital status.

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-11. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: excluding exposed participants who now live in comparison communities.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10						
PFOS	247 (64)	0.93 (0.80,1.08)	120 (29)	0.80 (0.59,1.10)	272 (69)	0.88 (0.74,1.06)
PFOA	247 (64)	0.84 (0.68,1.04)	120 (29)	0.93 (0.71,1.22)	272 (69)	0.91 (0.72,1.14)
PFHxS	247 (64)	0.97 (0.86,1.10)	120 (29)	0.99 (0.79,1.23)	272 (69)	0.97 (0.84,1.11)
K6 score ≥13						
PFOS	255 (51)	0.97 (0.81,1.16)	124 (24)	0.81 (0.60,1.11)	276 (57)	1.00 (0.84,1.19)#
PFOA	255 (51)	0.73 (0.56,0.94)	124 (24)	0.93 (0.63,1.36)	276 (57)	0.87 (0.65,1.15)#
PFHxS	255 (51)	0.96 (0.83,1.12)	124 (24)	0.90 (0.71,1.13)	276 (57)	0.91 (0.78,1.07)#
DQ5 score ≥14						
PFOS	257 (47)	0.98 (0.82,1.18)	126 (25)	0.69 (0.54,0.89)	276 (49)	1.06 (0.89,1.26)
PFOA	257 (47)	0.79 (0.60,1.05)	126 (25)	0.82 (0.57,1.17)	276 (49)	1.04 (0.75,1.45)
PFHxS	257 (47)	0.96 (0.83,1.10)	126 (25)	0.80 (0.65,0.99)	276 (49)	0.95 (0.83,1.10)
GAD-7 score ≥10						
PFOS	254 (25)	0.83 (0.63,1.09)	125 (13)	0.96 (0.53,1.75)	277 (25)	0.99 (0.73,1.34)#
PFOA	254 (25)	0.70 (0.44,1.13)	125 (13)	1.00 (0.53,1.90)	277 (25)	1.07 (0.59,1.97)#
PFHxS	254 (25)	0.81 (0.66,1.00)	125 (13)	0.96 (0.60,1.53)	277 (25)	0.93 (0.71,1.22)#

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), and occupational AFFF exposure (yes vs. no).

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-12. Adjusted prevalence ratios of clinically-significant self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: PFAS serum concentrations below the limit of quantification imputed using multiple imputation by chained equations.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†	N (cases)	Adjusted PR (95% CI)†
PHQ-15 score ≥10						
PFOS	260 (65)	0.94 (0.81,1.09)	149 (33)	0.79 (0.58,1.07)	280 (70)	0.89 (0.75,1.06)
PFOA	260 (65)	0.83 (0.67,1.03)	149 (33)	0.80 (0.61,1.06)	280 (70)	0.92 (0.73,1.15)
PFHxS	260 (65)	0.98 (0.87,1.10)	149 (33)	0.97 (0.78,1.21)	280 (70)	0.98 (0.85,1.12)
K6 score ≥13						
PFOS	268 (54)	0.94 (0.78,1.13)	156 (31)	0.85 (0.64,1.13)	284 (59)	0.99 (0.83,1.17)#
PFOA	268 (54)	0.71 (0.56,0.89)	156 (31)	0.78 (0.58,1.04)	284 (59)	0.87 (0.66,1.15)#
PFHxS	268 (54)	0.95 (0.82,1.10)	156 (31)	0.90 (0.74,1.10)	284 (59)	0.91 (0.78,1.06)#
DQ5 score ≥14						
PFOS	270 (49)	0.95 (0.78,1.15)	157 (32)	0.74 (0.58,0.95)	284 (50)	1.05 (0.89,1.25)
PFOA	270 (49)	0.75 (0.57,0.98)	157 (32)	0.83 (0.58,1.20)	284 (50)	1.06 (0.76,1.48)
PFHxS	270 (49)	0.94 (0.82,1.09)	157 (32)	0.88 (0.72,1.07)	284 (50)	0.95 (0.83,1.10)
GAD-7 score ≥10						
PFOS	267 (25)	0.85 (0.65,1.10)	157 (16)	0.98 (0.63,1.53)	285 (26)	0.96 (0.72,1.30)#
PFOA	267 (25)	0.72 (0.45,1.13)	157 (16)	0.84 (0.54,1.32)	285 (26)	1.07 (0.61,1.88)
PFHxS	267 (25)	0.83 (0.67,1.01)	157 (16)	0.97 (0.66,1.43)	285 (26)	0.91 (0.70,1.18)#

Among survey respondents, detection rates were 99.5-100% for PFOS, 97.8-99.5% for PFOA, and 93.6-96.2% for PFHxS.

N: sample size; PR: prevalence ratio; DQ5: Distress Questionnaire-5; GAD-7: Generalised Anxiety Disorder assessment-7; K6: Kessler distress scale-6; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), and occupational AFFF exposure (yes vs. no).

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2- 13. Adjusted differences in mean self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: additional adjustment for smoking status and alcohol consumption.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†
PHQ-15 score						
PFOS	248	0.0 (-0.4,0.4)	145	-0.6 (-1.3,0.1)	271	-0.1 (-0.5,0.3)
PFOA	248	-0.5 (-1.2,0.3)	145	-0.5 (-1.2,0.3)	271	-0.4 (-1.1,0.3)
PFHxS	248	0.0 (-0.3,0.4)	145	-0.1 (-0.6,0.4)	271	0.0 (-0.4,0.4)
K6 score						
PFOS	256	-0.3 (-0.6,0.1)	149	-0.2 (-0.8,0.4)	275	-0.1 (-0.4,0.3)#
PFOA	256	-0.6 (-1.2,0.1)	149	-0.4 (-1.1,0.3)	275	-0.4 (-0.9,0.2)#
PFHxS	256	-0.2 (-0.5,0.1)	149	-0.1 (-0.5,0.4)	275	-0.2 (-0.5,0.1)#
DQ5 score						
PFOS	258	-0.1 (-0.5,0.2)	151	-0.3 (-0.9,0.3)	275	-0.0 (-0.4,0.3)
PFOA	258	-0.1 (-0.7,0.4)	151	-0.2 (-0.9,0.6)	275	0.1 (-0.5,0.7)
PFHxS	258	-0.2 (-0.5,0.1)	151	-0.1 (-0.5,0.4)	275	-0.1 (-0.4,0.3)
GAD-7 score						
PFOS	256	-0.2 (-0.5,0.2)	150	-0.2 (-0.9,0.5)	276	0.1 (-0.3,0.5)
PFOA	256	-0.5 (-1.1,0.1)	150	-0.3 (-0.9,0.4)	276	0.3 (-0.5,1.0)
PFHxS	256	-0.1 (-0.4,0.2)	150	0.0 (-0.5,0.5)	276	0.1 (-0.3,0.4)

N: sample size; DQ5: Distress Questionnaire-5; K6: Kessler distress scale-6; GAD-7: Generalised Anxiety Disorder assessment-7; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), occupational AFFF exposure (yes vs. no), smoking status, and alcohol consumption.

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-14. Adjusted differences in mean self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: additional adjustment for marital status.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†
PHQ-15 score						
PFOS	259	-0.1 (-0.5,0.4)	149	-0.7 (-1.4,0.0)	280	-0.1 (-0.6,0.3)
PFOA	259	-0.7 (-1.5,0.0)	149	-0.6 (-1.3,0.2)	280	-0.4 (-1.2,0.3)
PFHxS	259	-0.1 (-0.4,0.3)	149	-0.2 (-0.7,0.4)	280	-0.0 (-0.4,0.4)
K6 score						
PFOS	267	-0.3 (-0.7,0.1)#	155	-0.3 (-0.8,0.3)	284	-0.0 (-0.4,0.3)#
PFOA	267	-0.7 (-1.4,-0.1)#	155	-0.5 (-1.2,0.2)	284	-0.3 (-0.9,0.3)#
PFHxS	267	-0.2 (-0.5,0.1)#	155	-0.1 (-0.5,0.3)	284	-0.2 (-0.5,0.1)#
DQ5 score						
PFOS	269	-0.2 (-0.5,0.2)	157	-0.4 (-1.0,0.1)	284	-0.0 (-0.4,0.4)
PFOA	269	-0.5 (-1.0,0.1)	157	-0.3 (-1.0,0.4)	284	0.1 (-0.6,0.7)
PFHxS	269	-0.1 (-0.4,0.2)	157	-0.1 (-0.5,0.3)	284	-0.0 (-0.4,0.3)
GAD-7 score						
PFOS	266	-0.2 (-0.6,0.1)#	156	-0.2 (-0.9,0.4)	285	0.1 (-0.3,0.5)
PFOA	266	-0.7 (-1.3,-0.0)#	156	-0.4 (-1.0,0.3)	285	0.2 (-0.5,0.9)
PFHxS	266	-0.1 (-0.5,0.2)#	156	-0.0 (-0.5,0.5)	285	0.0 (-0.3,0.4)

N: sample size; DQ5: Distress Questionnaire-5; K6: Kessler distress scale-6; GAD-7: Generalised Anxiety Disorder assessment-7; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), occupational AFFF exposure (yes vs. no), and marital status.

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-15. Adjusted differences in mean self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: excluding exposed participants who now live in comparison communities.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†
PHQ-15 score						
PFOS	247	-0.1 (-0.5,0.4)	120	-0.6 (-1.4,0.1)	272	-0.1 (-0.6,0.3)
PFOA	247	-0.7 (-1.4,0.1)	120	-0.3 (-1.2,0.7)	272	-0.5 (-1.2,0.3)
PFHxS	247	-0.1 (-0.5,0.3)	120	-0.2 (-0.8,0.4)	272	-0.0 (-0.4,0.4)
K6 score						
PFOS	255	-0.3 (-0.7,0.1)#	124	-0.3 (-0.9,0.3)	276	0.0 (-0.3,0.4)#
PFOA	255	-0.6 (-1.3,0.0)#	124	-0.1 (-0.8,0.5)	276	-0.3 (-0.9,0.3)#
PFHxS	255	-0.2 (-0.5,0.1)#	124	-0.1 (-0.5,0.4)	276	-0.1 (-0.4,0.2)#
DQ5 score						
PFOS	257	-0.2 (-0.6,0.2)	126	-0.5 (-1.1,0.0)	276	0.0 (-0.3,0.4)
PFOA	257	-0.3 (-1.0,0.3)	126	-0.2 (-1.0,0.6)	276	0.1 (-0.6,0.7)
PFHxS	257	-0.1 (-0.4,0.2)	126	-0.2 (-0.7,0.2)	276	0.0 (-0.3,0.4)
GAD-7 score						
PFOS	254	-0.2 (-0.6,0.1)#	125	-0.3 (-1.0,0.4)	277	0.1 (-0.3,0.5)
PFOA	254	-0.7 (-1.3,0.0)#	125	0.0 (-0.7,0.7)	277	0.2 (-0.5,0.9)
PFHxS	254	-0.2 (-0.5,0.2)#	125	-0.0 (-0.6,0.5)	277	0.1 (-0.2,0.4)

N: sample size; DQ5: Distress Questionnaire-5; K6: Kessler distress scale-6; GAD-7: Generalised Anxiety Disorder assessment-7; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), and occupational AFFF exposure (yes vs. no).

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-16. Adjusted differences in mean self-reported psychological distress scores per doubling in PFAS serum concentrations in participants from PFAS Management Areas, 2016–2020. Sensitivity analysis: PFAS serum concentrations below the limit of quantification imputed using multiple imputation by chained equations.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†
PHQ-15 score						
PFOS	260	-0.1 (-0.5,0.4)	149	-0.7 (-1.3,0.0)	280	-0.1 (-0.6,0.3)
PFOA	260	-0.7 (-1.5,0.0)	149	-0.6 (-1.4,0.2)	280	-0.4 (-1.2,0.3)
PFHxS	260	-0.1 (-0.4,0.3)	149	-0.2 (-0.7,0.4)	280	-0.0 (-0.4,0.4)
K6 score						
PFOS	268	-0.3 (-0.7,0.0)#	156	-0.3 (-0.8,0.3)	284	-0.0 (-0.4,0.3)#
PFOA	268	-0.7 (-1.4,-0.1)#	156	-0.5 (-1.2,0.2)	284	-0.3 (-0.9,0.3)#
PFHxS	268	-0.2 (-0.5,0.1)#	156	-0.1 (-0.5,0.3)	284	-0.2 (-0.5,0.1)#
DQ5 score						
PFOS	270	-0.2 (-0.6,0.2)	157	-0.4 (-1.0,0.1)	284	-0.0 (-0.4,0.4)
PFOA	270	-0.4 (-1.1,0.2)	157	-0.3 (-1.0,0.5)	284	0.1 (-0.5,0.7)
PFHxS	270	-0.1 (-0.4,0.2)	157	-0.1 (-0.5,0.3)	284	-0.0 (-0.4,0.3)
GAD-7 score						
PFOS	267	-0.2 (-0.6,0.1)#	157	-0.2 (-0.9,0.4)	285	0.1 (-0.3,0.5)
PFOA	267	-0.7 (-1.3,-0.1)#	157	-0.4 (-1.1,0.2)	285	0.2 (-0.5,0.9)
PFHxS	267	-0.2 (-0.5,0.2)#	157	-0.0 (-0.5,0.4)	285	0.0 (-0.3,0.4)

Among survey respondents, detection rates were 99.5-100% for PFOS, 97.8-99.5% for PFOA, and 93.6-96.2% for PFHxS.

N: sample size; DQ5: Distress Questionnaire-5; K6: Kessler distress scale-6; GAD-7: Generalised Anxiety Disorder assessment-7; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), and occupational AFFF exposure (yes vs. no).

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

Table A2-17. Adjusted differences in mean self-reported psychological distress scores for factors that may affect the perceived risk of PFAS exposure in participants from PFAS Management Areas, 2019–2020.

	Katherine (NT)		Oakey (Qld)		Williamtown (NSW)	
	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†	N	Adjusted difference (95% CI)†
PHQ-15 score						
Current (vs. former) residence or work	259	-0.8 (-2.0,0.5)	157	-0.2 (-1.9,1.6)	278	-0.4 (-1.8,0.9)
Per decade of residence	259	0.1 (-0.5,0.7)	157	-0.8 (-1.4,-0.1)	278	-0.2 (-0.6,0.2)
Occupational AFFF exposure	259	2.8 (1.4,4.1)	157	0.5 (-1.1,2.2)	278	2.5 (1.2,3.7)
Bore water use	259	0.3 (-1.0,1.7)	157	1.3 (-0.5,3.1)	278	0.4 (-1.0,1.7)
K6 score						
Current (vs. former) residence or work	267	-0.4 (-1.4,0.7)#	161	0.5 (-1.0,2.0)	283	-0.7 (-1.7,0.3)#
Per decade of residence	267	-0.0 (-0.5,0.5)#	161	-0.4 (-1.0,0.1)	283	0.1 (-0.3,0.4)#
Occupational AFFF exposure	267	1.3 (0.2,2.5)#	161	0.8 (-0.8,2.4)	283	1.8 (0.7,2.9)#
Bore water use	267	0.7 (-0.7,2.1)#	161	0.7 (-0.9,2.4)	283	0.2 (-1.1,1.5)#
DQ5 score						
Current (vs. former) residence or work	269	-0.7 (-1.7,0.3)	163	-0.7 (-2.1,0.7)	283	-0.9 (-1.9,0.1)
Per decade of residence	269	-0.0 (-0.5,0.4)	163	-0.3 (-0.9,0.3)	283	0.0 (-0.3,0.4)
Occupational AFFF exposure	269	1.5 (0.3,2.6)	163	0.8 (-0.7,2.2)	283	2.0 (0.9,3.1)
Bore water use	269	0.7 (-0.5,2.0)	163	0.6 (-0.9,2.0)	283	1.0 (-0.2,2.3)
GAD-7 score						
Current (vs. former) residence or work	267	-0.6 (-1.6,0.5)#	162	0.2 (-1.4,1.9)	284	-0.7 (-1.7,0.3)
Per decade of residence	267	-0.1 (-0.6,0.4)#	162	-0.3 (-0.9,0.2)	284	-0.1 (-0.4,0.2)
Occupational AFFF exposure	267	1.4 (0.4,2.5)#	162	0.7 (-0.8,2.2)	284	2.0 (1.0,3.1)
Bore water use	267	1.2 (-0.2,2.6)#	162	1.0 (-0.7,2.7)	284	0.9 (-0.4,2.1)

N: sample size; DQ5: Distress Questionnaire-5; K6: Kessler distress scale-6; GAD-7: Generalised Anxiety Disorder assessment-7; PHQ-15: Patient Health Questionnaire-15; CI: confidence interval.

† Adjusted for age, sex, level of education, gross household annual income, current (vs. former) residence or work in a PFAS Management Area, length of time residing in a PFAS Management Area (per decade), bore water use (ever vs. never), and occupational AFFF exposure (yes vs. no).

Estimated assuming an independence (rather than exchangeable) within-cluster correlation structure and cluster-robust standard errors.

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