PNAS RESEARCH ARTICLE | SUSTAINABILITY SCIENCE

PFAS-contaminated drinking water harms infants

Robert Baluja[a], Bo Guo[b], Wesley Howden[c], Ashley Langer[d,e], and Derek Lemoine[d,e,1]

Affiliations are included on p. 9.

Edited by Catherine Kling, Cornell University, Ithaca, NY; received April 24, 2025; accepted October 22, 2025

There is evidence of widespread human exposure to per- and polyfluoroalkyl substances (PFAS) but limited evidence of the human health impacts of this exposure. Using data on New Hampshire births from 2010–2019, we show that mothers receiving water that had flowed beneath a PFAS-contaminated site, as opposed to comparable mothers receiving water that had flowed toward a PFAS-contaminated site, had 191% [95% CI: 83–298%] higher first-year infant mortality (611 [268–955] additional first-year deaths per 100k births); 168% [42–294%] more births before 28 wk of gestational age (466 [116–817] additional such births per 100k births); and 180% [57–302%] more births with weight below 1,000 g (607 [192–1022] additional such births per 100k births). Extrapolating to the contiguous U.S., PFAS contamination imposes annual social costs of approximately $8 billion. These health costs are substantially larger than current outside estimates of the cost of removing PFAS from the public water supply.

PFAS | water pollution | reproductive health | infant mortality | birthweight

Significance

Per- and polyfluoroalkyl substances (PFAS), so-called "forever chemicals," are pollutants of increasing concern. However, there is not yet firm evidence that the types of PFAS exposure occurring in daily life cause human health impacts. We show that New Hampshire mothers whose drinking water wells were downstream of PFAS releases had more extremely low-weight births, more extremely preterm births, and higher infant mortality than did mothers whose wells were upstream of PFAS releases. Mothers did not know the locations of their wells and so should be comparable but for their PFAS exposure. Extrapolating to the rest of the United States, PFAS impose billions of dollars of costs on U.S. residents each year by worsening infant health.

Policymakers and scientists are increasingly concerned about the potential for per- and polyfluoroalkyl substances (PFAS) to impact human health (1, 2). These extremely persistent chemicals serve a range of industrial and household purposes (3), from firefighting foams to nonstick coatings. PFAS have been found throughout the global environment (4, 5), including in drinking water systems (6–8), groundwater resources (9, 10), and human blood samples (11, 12). Regulatory bodies around the world are studying and enacting controls that include restrictions on production, imports, use, and disposal (13–15). However, a lack of epidemiological evidence for causal impacts on human health has hampered regulatory efforts to control PFAS (16). Here we provide evidence of a causal link from PFAS exposure to human health in real-world populations.

We study the impacts of PFAS on reproduction, an area of particular concern. Animal toxicology studies suggest that PFAS can lower birthweight (17), but whether the types of PFAS exposure that occur in daily life rather than in the lab affect human reproduction has not been clear, both because results are mixed and because human studies have nearly all been associational rather than causal (18). Human epidemiological studies have examined the relationship between PFAS measured in (maternal or cord) blood serum and birth outcomes. Results are mixed (19–22), with some studies finding an association with preterm births and/or low-weight births and other studies not finding an association with either or both of these outcomes. Moreover, even when an association is detected, it is possible that an additional health factor drives both PFAS measurements and birth outcomes. For example, poor maternal kidney functioning can cause both high blood PFAS levels and low birthweight (23, 24).

Here, we link exposure to PFAS through public water systems to infant mortality, preterm births, and low-weight births. When a population has PFAS in its water supply, drinking water becomes its primary source of exposure (22, 25, 26). Prior studies have measured the correlation between drinking water systems' PFAS and reproductive outcomes, again with mixed results (27, 28), and have tested how a community's installation of a water filtration system changed birth outcomes relative to other communities (29). However, these prior studies might not capture the causal effect of PFAS. First, households who choose to live in areas served by water systems with more PFAS contamination (27, 28), or in areas that have not yet cleaned up PFAS contamination (29), may be different from households who choose to live in areas with less contamination or that have cleaned up contamination (30). These differences may be due to health preferences or may be a response to PFAS contamination lowering house prices (31). Either way, households' sorting into and out of PFAS exposure might generate a spurious correlation between PFAS contamination and reproductive outcomes. Second, households may undertake preventive actions such as purchasing bottled water when public water systems either contain more PFAS or plan to install a filtration

Author contributions: R.B., B.G., W.H., A.L., and D.L. designed research; R.B., B.G., A.L., and D.L. performed research; R.B. analyzed data; W.H. administered the project; and D.L. wrote the paper.

Competing interest statement: A.L. and D.L. are codirectors of the newly established Consortium for Environmentally Resilient Business (CERB) at the Eller College of Management at the University of Arizona. This Consortium receives funding from electric utility affiliates. The present research does not use any data from CERB affiliates, and CERB affiliates have not reviewed this research prior to publication.

This article is a PNAS Direct Submission.

[1]To whom correspondence may be addressed. Email: dlemoine@arizona.edu.

This article contains supporting information online at https://www.pnas.org/lookup/suppl/doi:10.1073/pnas.2509801122/-/DCSupplemental.

Published December 8, 2025.

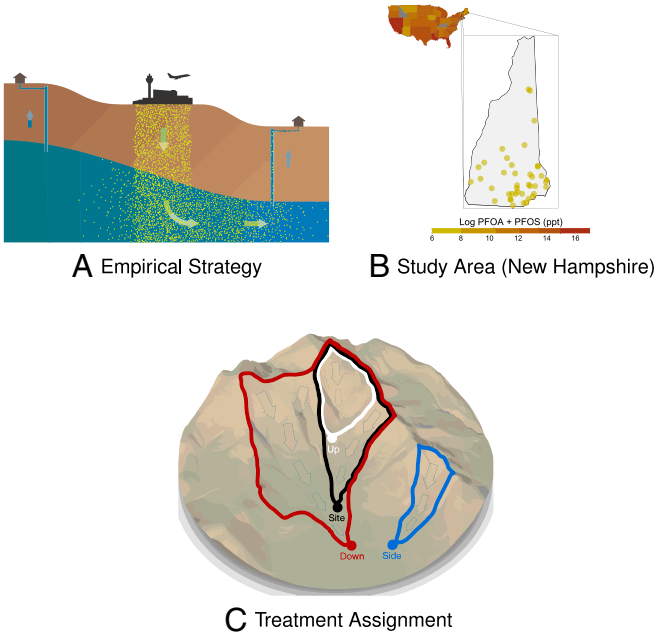

Fig. 1. (*A*) Our empirical strategy compares reproductive outcomes among households who receive water from wells that draw groundwater flowing either to or from a PFAS-contaminated site (here, an airport). (*B*) The study region comprises circles of 5 km radius centered on each PFAS-contaminated site. Circles are colored by the level of PFAS attributed to each site. The map of the United States is colored by the maximum level of confirmed PFAS contamination within each state. Both are measured in log-scale (ppt). (*C*) Groundwater flows downhill. The PFAS-contaminated site ("Site") is in the catchment area (outlined in red) of the downgradient well ("Down"). The upgradient well ("Up") is within the catchment area of the contaminated site (outlined in black). The remaining well ("Side") and the contaminated site are outside of each other's catchment areas (outlined in blue and black, respectively).

system (32). These preventive actions may obscure the true causal effect of PFAS contamination on reproductive outcomes.

We rely on variation in PFAS exposure that we argue is as good as random. In particular, we compare reproductive outcomes among mothers whose homes receive water piped from wells on groundwater flowing toward a source of PFAS contamination with reproductive outcomes among mothers whose homes receive water piped from wells on groundwater flowing from a source of PFAS contamination (Fig. 1*A*). To do so, we combine geocoded data on all births and infant deaths among New Hampshire residents from 2010–2019 with the locations of PFAS-contaminated sites and public drinking water wells (Fig. 1*B*). We compare mothers who live similar distances from contaminated sites. Because the locations of public water wells are confidential, these mothers should not know whether they receive water from a well whose groundwater flows to or from a contaminated site. As a result, we compare mothers who should not have differentially chosen where to live based on potential PFAS exposure and should not differentially take actions to mitigate PFAS exposure.

In our primary analysis, we study 41 sites with contamination of the two most prominent PFAS, perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS), above 1,000 ppt (*SI Appendix*, section A). These sites are mostly contaminated by industrial sources and landfills, but some are contaminated by fire fighting related activities. We focus on groundwater supplies for several reasons. First, it is more straightforward to connect water supplies to contamination when on groundwater because the contamination is typically persistent [driven by PFAS-impacted soils (33)] where PFOA and PFOS slowly leach to the water table (34). In contrast, surface water contamination can vary with current polluting activities and migrates further over a broader region. Second, preexisting treatment plants for groundwater systems rarely remove PFAS (35). Third, groundwater is more challenging to sample and test than surface water, making it more likely that mothers do not know their exposure. Finally, short-chain species of PFAS, which may have different health implications and may require different removal techniques, are generally much less abundant than PFOA and PFOS in groundwater (36, 37).

Framework for Testing the Effects of Receiving Water Flowing From a PFAS-Contaminated Site

To implement our empirical strategy, we develop a multivariate regression framework (38, 39) that relates birth outcomes to exposure to PFOA and PFOS (*Materials and Methods*). We study whether a baby dies within their first year of life ("infant mortality"), whether a birth occurs before 37 wk ("preterm"), and whether a baby's birthweight is below 2,500 grams ("low birthweight"). We also distinguish degrees of prematurity and degrees of low birthweight. Using conventional thresholds (40, 41), we separately test for effects of PFAS on moderately preterm births (between 32 and 36 wk), very preterm births (between 28 and 31 wk), and extremely preterm births (less than 28 wk). And again using conventional thresholds (39, 42), we separately test for the effects of PFAS on moderately low-weight births (between 1,500 and 2,500 grams), very low-weight births (between 1,000 and 1,500 grams), and extremely low-weight births (less than 1,000 grams).

A location's "catchment area" is the area upslope from it, so that water flows to a location from the points in its catchment area. PFAS released in a location's catchment area plausibly end up at the location, and PFAS released elsewhere plausibly do not. We proxy for the direction of groundwater flow with high-resolution data on land surface topography. The correlation between groundwater flow and surface topography is widely accepted and used in the hydrological sciences (43–45). *SI Appendix*, section L provides direct evidence of this correlation in our study area.

We calculate whether each public well in our data is within the catchment area of a PFAS-contaminated site and whether a PFAS-contaminated site is within the catchment area of the well (*Materials and Methods*). We classify a well as "downgradient" if at least one nearby (i.e., within 5 km) contaminated site is within its catchment area. If the well is not downgradient, then we classify it as "upgradient" if it is within the catchment area of every nearby contaminated site. Otherwise we classify the well as to the "side." Within our multivariate regression framework, the coefficient on the downgradient indicator gives the effect on birth outcomes of receiving water from a well downgradient of some nearby contaminated site relative to receiving water from a well upgradient of all nearby contaminated sites.

Fig. 1*C* illustrates this assignment. The *Top* of the figure is a hilltop. Groundwater flows from this peak toward a contaminated site (black dot). The well at the red dot is downhill from the contaminated site. Its catchment area (outlined in red) includes the contaminated site. Because PFAS flow from the contaminated site to the well at the red dot, we classify that well as downgradient.

In contrast, the well at the white dot is uphill from the contaminated site. The catchment area of the contaminated site

(outlined in black) includes the well at the white dot and all of its catchment area (outlined in white). Because PFAS flow from the well at the white dot to the contaminated site, we classify that well as upgradient with respect to this contaminated site.

The well at the blue dot is in the valley on the other side of a ridge from the contaminated site. The contaminated site is outside that well's catchment area (outlined in blue), and the well is outside the contaminated site's catchment area (outlined in black). We classify the well at the blue dot as to the side with respect to the contaminated site.

We test whether mothers who receive water from wells like that at the red dot have worse birth outcomes than mothers who receive water from wells such as that at the white dot. We use demographic, environmental, medical, house, and well control variables and both county and year-by-month fixed effects to make the mothers as similar as possible (*Materials and Methods* and *SI Appendix*, section B). Our regression framework compares births in the same month, in the same county, to observably similar mothers, some of whom are provided with water from public wells upgradient of a contaminated site and some of whom are provided with water from public wells downgradient of a contaminated site.

Birth Outcomes Are Worse Among Mothers Served by Wells Whose Water Flows From a PFAS-Contaminated Site

We find that being served by a well downgradient from a PFAS-contaminated site causes worse birth outcomes. Fig. 2 reports estimates as a percentage change in the base rate of that reproductive outcome in the sample (*SI Appendix*, section G reports results as additional births per 100,000 births). The panel across the top reports results for infant mortality. In the *Lower* panels, the *Left* column contains results for preterm births and the *Right* column contains results for low-weight births. Within each column, the *Upper* panels report results for all preterm or low-weight births and the *Lower* panels report results for extremely preterm or extremely low-weight births. Significance tests are relative to a null hypothesis of a weakly negative effect of PFAS exposure.

In our baseline specification (*Top* row in each panel), we estimate that being served by a well downgradient of a PFAS-contaminated site, as opposed to being served by a well upgradient of a PFAS-contaminated site, increases the chance of a baby dying during their first year of life by 191% [95% CI: 83%–298%, $P < 0.001$] (611 [268–955] additional deaths per 100k births), increases the chance of a preterm birth by 20% [−4.0–44%, $P = 0.06$] (1,475 [−291–3,240] additional preterm births per 100k births), and increases the chance of a low-weight birth by 43% [19–68%, $P < 0.001$] (2,639 [1,164–4,114] additional low-weight births per 100k births). *SI Appendix*, section G reports results for births that are moderately and very preterm or moderately and very low-weight. We do not have hypotheses regarding how the number of births in these less severe categories of preterm and low-weight births should change, because our estimated effects net out shifts into these less severe categories from healthier categories with shifts from these less severe categories into more severe categories. However, we do have hypotheses regarding how the number of births in the most severe categories should change. Indeed, Fig. 2 shows that we estimate large and statistically significant (at the 1% level) effects on the most severe categories of preterm and low-weight births: The chance of an extremely preterm birth increases by 168% [42–294%, $P = 0.007$] (466 [116–817] additional such births per 100k births), and the chance of an extremely low-weight birth increases by 180% [57–302%, $P = 0.004$] (607 [192–1,022] additional such births per 100k births).

The remaining rows demonstrate the robustness of our primary results. The second row drops mothers from the sample if their drinking water comes from a well within 1km of a contaminated site. These mothers are more likely to be misassigned because the topography-based groundwater flow direction may be less accurate at a smaller scale of 1km (e.g., groundwater flow and PFAS may transport from lower to higher elevation due to local spatial heterogeneities). These mothers may also be more likely to be aware of the contaminated site. The third row drops

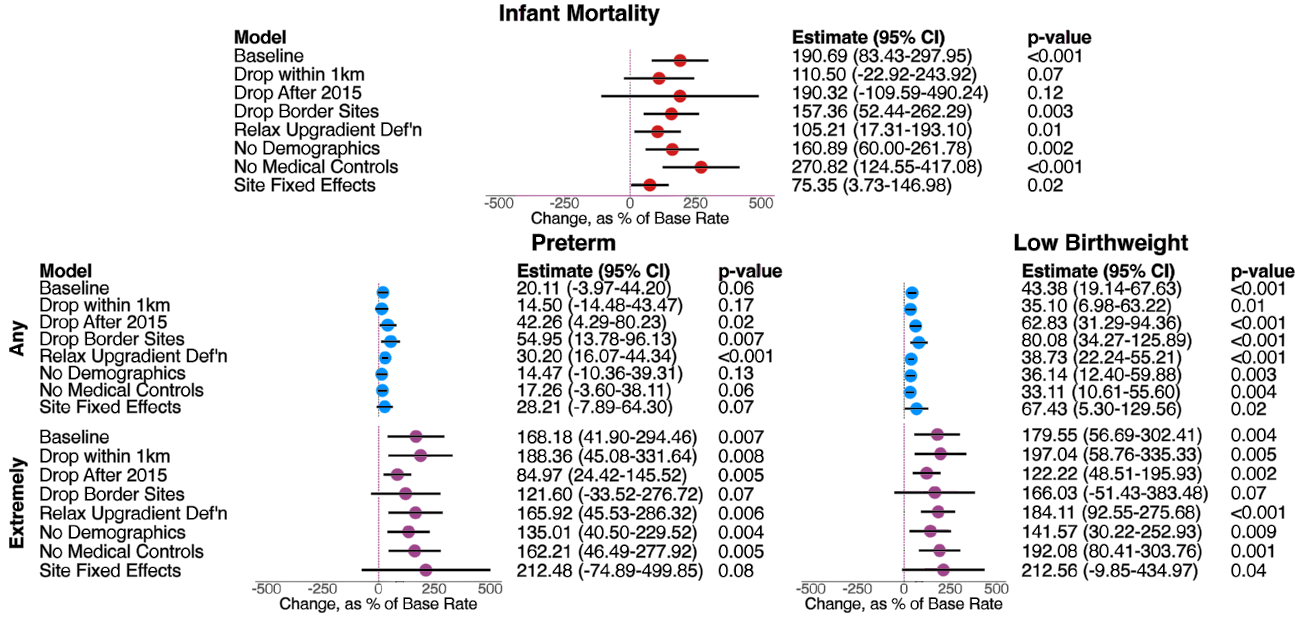

Fig. 2. Estimated effects of a mother being served by a downgradient well instead of an upgradient well, by reproductive outcome. Effects reported as percentage increases in the base rate of that reproductive outcome. Reported *P*-values are for one-sided tests of a null hypothesis of a weakly negative effect.

births after 2015 (so keeping births in 2010–2015), when the State of New Hampshire began a testing program. This testing program might have raised awareness of PFAS, which could have led households to attempt to sort on exposure or to take preventive actions regardless of exposure. The fourth row drops contaminated sites within 5 km of a state border, in case PFAS could have flowed to wells from unobserved contaminated sites beyond our study area. The fifth row applies a less stringent definition of upgradient, requiring only that a well be upgradient of *some* nearby contaminated site (rather than of *all* nearby contaminated sites) while not being downgradient of any nearby contaminated site. The control population becomes more likely to include individuals who have been treated to some degree, which would work to attenuate estimates (as we indeed see for infant mortality). The sixth and seventh rows remove all demographic and medical control variables, respectively. The final row includes contaminated site fixed effects so that, at the cost of power to detect a true effect, we compare mothers whose wells are near the same contaminated site. *SI Appendix*, section D changes the radius used to define "nearby" contaminated sites. Our results are remarkably robust to each of these alternate specifications.

Several additional results reinforce our primary analysis. *SI Appendix*, section G shows that being served by a well that is upgradient of a contaminated site (relative to a well classified as to the side) does not significantly increase the chance of infant mortality, of a preterm birth, or of a low-weight birth—and in some specifications significantly decreases the chance of those births. It also shows that being served by a well downgradient of a contaminated site increases the chance of a low-weight birth by 87% [37–137%, $P < 0.001$] even among full-term babies and, dropping the preterm and low-weight categorizations, reduces gestational length by 0.18 [0.004–0.4, $P = 0.03$] weeks and weight by 50 [12–88, $P = 0.008$] grams on average (with P-values for a null hypothesis of a weakly positive effect of PFAS exposure). It reports suggestive evidence that birth outcomes are worse for mothers who receive water from wells that are closer to contaminated sites. It shows that our results are not driven by exceptionally contaminated sites, by limiting our definition of downgradient to the well nearest a mother's residence, or by excluding surface water sources. And it shows that effects of being served by a well downgradient of a contaminated site are broadly similar for mothers who were long-term and short-term residents, with suggestive evidence of stronger effects on some outcomes for the longest-term residents.

Several additional tests suggest interpreting our results as causal. First, *SI Appendix*, section C shows that downgradient mothers tend to be of higher socioeconomic status, which one would expect to bias us against finding that PFAS exposure negatively affects infant health. *SI Appendix*, section E shows that if upgradient and downgradient mothers differed only in their observed socioeconomic and demographic characteristics (and not also in their exposure to PFAS), then we would expect infant health outcomes to be similar between the two groups—and if anything, we would expect infant mortality and birthweight to be better among downgradient mothers. Second, *SI Appendix*, section F tests whether we would estimate similarly significant effects of being downgradient if we assigned random locations around New Hampshire to be "contaminated sites." It shows that downgradient wells are not generically associated with worse birth outcomes than upgradient wells. Finally, *SI Appendix*, section M shows that we obtain qualitatively similar results for preterm births and low-weight births in an out-of-sample test that uses coarser data available for New York State.

PFAS Impose Costs Throughout the United States

We use our estimated health impacts to learn about the aggregate importance of PFAS as a public health problem in the United States. We calculate the social costs imposed by PFAS in three steps.

First, instead of estimating the effect of a mother being served by a downgradient well, we estimate a continuous measure of the effect of a mother being served by a well that we project as having greater PFAS in its groundwater. Fig. 3*A* reports the estimated effect of increasing the PFAS concentration by 100 ppt, which is approximately half of the median concentration predicted for the estimation sample's wells (*Materials and Methods*). We estimate that being served by a public well predicted to have more PFAS significantly increases the chances of infant mortality ($P = 0.012$) and also significantly increases the chance of a preterm ($P < 0.001$) or low-weight ($P < 0.001$) birth. As in Fig. 2, effects are especially large for the worst health outcomes.

Second, we impute PFAS concentrations around each contaminated site in 11 U.S. states with PFAS testing programs by combining topographical data with data on PFAS contamination (46) (*Materials and Methods*). We combine those imputed PFAS levels with data on births close to the contaminated sites and with our estimated effects of PFAS from Fig. 3*A* in order to predict the change in a year's birth outcomes around each contaminated site. The blue bars in Fig. 3*B* (*Left* axis) tally these effects across the 11 states. We estimate around 400 additional deaths, over 1,000 additional preterm births (of which around 600 are extremely preterm), and over 1,000 additional low-weight births (of which around 850 are extremely low-weight).

Finally, we combine these estimates with the social cost per reproductive outcome (*Materials and Methods*). The lifetime social costs from a preterm birth include additional medical costs for the child, additional delivery costs at birth, additional educational services, and lost labor productivity. These costs increase sharply in the degree of prematurity (40). The lifetime social costs from a low-weight birth include increased mortality in the first year, increased medical costs and disabilities, lost adult income, and increased long-term mortality risk. *SI Appendix*, section I details the calculations, which are based on several sources (39, 47, 48). We do not have social cost estimates per moderately low-weight birth. Our calculations are conservative in that we assume that the extra children born in one category would have otherwise been born in the next-most severe category. For the social cost of infant mortality, we conservatively assume that, had they survived through birth, these babies' life expectancies, quality of life, and medical outcomes would have been comparable to babies with extremely low birthweight, from ref. 49. The red hatched bars in Fig. 3*B* (*Right* axis) show that social costs in the 11 states are dominated by infant mortality and extremely low-weight births. Fig. 3*C* maps the lifetime social costs imposed by additional low-weight births on each year's birth cohort.

Fig. 3*D* aggregates social costs across the categories of preterm births and across the categories of low-weight births. It adds the social costs of infant mortality to the social costs of preterm births because the latter do not account for mortality risk. The purple bars plot the costs imposed on each year's birth cohort in the 11 states, which amount to $1.9 billion for preterm births and infant mortality and $2.7 billion for low-weight births. Likely sources of PFAS contamination are found throughout the United States (50). The hatched green bars scale our estimates to the whole U.S. by assuming that PFAS testing was comprehensive in those 11 states and that PFAS exposure in those 11 states

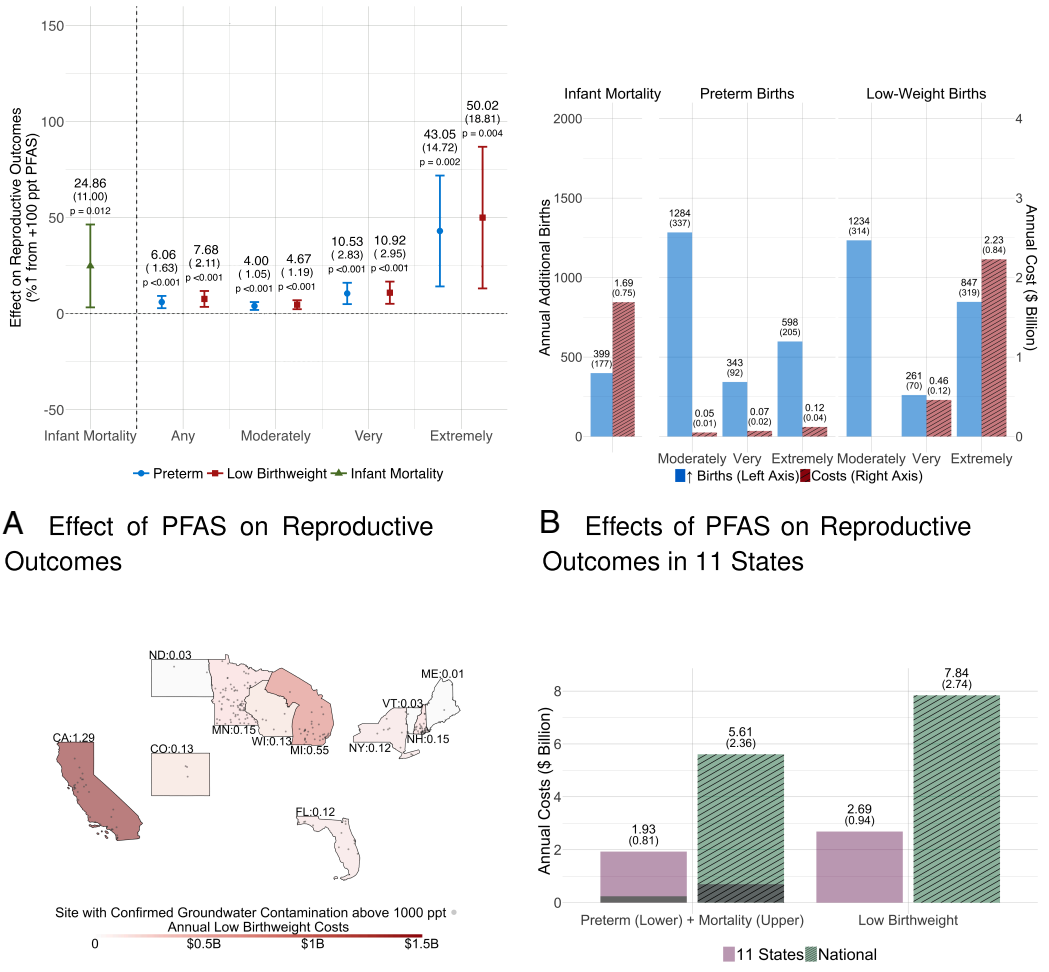

Fig. 3. (*A*) Estimated effect of a 100 ppt increase in PFAS at the median PFAS level, with 95% CIs. Labels give the point estimate, the SE (in parentheses), and the one-sided *P*-value for a null of a weakly negative effect. (*B*) Estimated change in annual rates of infant mortality, of preterm births, and of low-weight births (in blue). Social costs (hatched red bars) are lifetime social costs for an annual birth cohort. SEs in parentheses. (*C*) Map of lifetime social costs from each year's additional low-weight births, by state. (*D*) Social costs from infant mortality and preterm births (*Left*) and from low-weight births (*Right*) in the 11 states with data on contaminated sites (purple) and extrapolated to the contiguous U.S. (green, hatched).

is representative of exposure in the other states (*Materials and Methods*). We estimate costs per annual birth cohort of $5.6 billion from preterm births and infant mortality and of $7.8 billion from low-weight births. *SI Appendix*, section G shows that PFAS exposure increases infant mortality and extremely preterm births even after controlling for birthweight. The lifetime social costs across all three types of birth outcomes are therefore greater than the $7.8 billion we estimate from low-weight births alone.

Discussion

We have shown that PFAS exposure causes worse birth outcomes across the population of the U.S. state of New Hampshire. Extrapolating to the population of the United States, we estimate that PFAS generate at least $2.7 billion in annual costs in just the 11 states with a high level of testing—and at least $7.8 billion in annual costs nationwide. Either cost estimate far exceeds the estimated $1.5 billion in annual health benefits generated by the new U.S. PFAS rule (51). The national cost estimate also far exceeds even high-end estimates (of around $3.8 billion) of the cost of cleaning up PFAS to achieve this rule (52).

Our estimates are likely to be a lower bound on the true effects of PFAS on reproductive outcomes. Our assignment of mothers to treated and control groups is likely to be noisy: Both our matching of mothers to public wells and our calculated groundwater gradients are imperfect. In particular, some of the wells assigned as downgradient (or otherwise predicted to have high PFAS) may not be highly contaminated, whereas others assigned as upgradient or off to the side (or otherwise predicted to have low PFAS) may in fact be contaminated. Moreover, mothers are likely to drink water from other sources, including when out of the house, which reduces the strength of our treatment. All such complications should work to attenuate our estimates toward zero, which makes the strength and robustness of our estimated effects both more notable and more socially concerning.

Our estimated social costs are also likely to be lower bounds. In particular, our social costs per reproductive outcome assume that additional births would have otherwise come from the next-most severe category. However, some of the additional extremely preterm or extremely low-weight births are likely from babies who would have otherwise been above 31 wk or 1,500 g, respectively. Similarly, it is likely that some of the babies who died within

their first year of life would have experienced better life outcomes, measured by their quality of life and overall life expectancy, than babies born in the extremely low-weight category. By ignoring the potential movement of birth outcomes across multiple categories, we conservatively underestimate social costs.

Our estimated social costs are also a lower bound on the total social cost of PFAS because they include only the monetized portion of reproductive harms due to PFAS. Concurrent work suggests that early PFAS exposure reduces earnings and college completion (53). Some of these long-run consequences may be due to the effects on birthweight or gestational age that we estimate here, but to the extent that these consequences are additional to our estimates, their costs should be added to our social costs. In addition, PFAS are widely believed to cause a range of medical (2), ecological (3), and developmental (54) harms. Future work should seek causal estimates of these other effects from effectively random real-world exposure.

One limitation of the current study is that we do not observe the history of exposure. As a result, we do not know how the effects of PFAS vary with the duration of exposure or how rapidly cleaning up PFAS may provide benefits. We do find that outcomes are broadly similar among short- and long-term residents, which suggests that benefits from cleaning up PFAS could accrue rapidly. Future work should seek to distinguish short- and long-term impacts.

Another limitation of the current study is that our extrapolation uses only PFOA and PFOS, which are two of the more prominent species of PFAS. These two chemicals may be correlated with other pollutants, including other species of PFAS (*Materials and Methods*). Our estimated health impacts in New Hampshire then capture the broader costs of unregulated pollution associated with PFAS sources. Our estimated social costs are still well-defined even if other pollutants contribute to them: They define the benefits of installing PFAS-removal technologies such as granular activated carbon filtration as long as these technologies clean up correlated pollutants (*Materials and Methods* for why this is likely to be the case). When we extrapolate health impacts to other states, we implicitly hold correlations with other pollutants constant over space. It is not obvious if these correlations do in fact vary substantially over space or how such variation would affect our extrapolation.

Finally, we use data from New Hampshire because that is the only state for which we were able to obtain data on PFAS-contaminated sites, groundwater wells, and individual births. Supplemental analyses (in *SI Appendix*, sections D and H) show that our estimated effects appear to be similar for more- and less-contaminated sites and are approximately constant in the level of PFAS we predict for a mother's well. These results suggest that other states' PFAS-contaminated sites could have similar effects as in New Hampshire. On the other hand, New Hampshire has limited variation in mothers' demographics, which could affect how PFAS exposure translates into infant health. As more data become available, future work will have more power to assess how sensitivity to PFAS may vary with mothers' characteristics.

Materials and Methods

Data. Our analysis uses five primary sources of data, detailed in *SI Appendix*, section A. First, we use the universe of birth records for infants born to New Hampshire residents from 2010–2019, obtained from the New Hampshire Department of Health and Human Services. We match these birth records with the universe of death records for New Hampshire residents who die within their first year of life. The University of Arizona Institutional Review Board determined that our study was exempt. Second, we obtain locations served by public water systems and locations of groundwater-sourced public wells from the New Hampshire Department of Environmental Services. These data, particularly the locations of public wells, are not publicly available due to homeland security concerns. To receive access, one must apply directly to the New Hampshire Department of Environmental Services. Third, our set of contaminated sites comes from the Northeastern University PFAS Lab's set of sites with known PFOA/PFOS releases to nearby groundwater (46). Fourth, we use groundwater PFAS measurements from tests undertaken by the New Hampshire Department of Environmental Services. Fifth, we obtain a high-resolution digital elevation model of the state (55). Sixth, we obtain data on wind direction, air pollution, temperature, demographics, catchment area boundaries, and toxic contaminated sites from various sources.

Sample Construction. We use land surface topography from the high-resolution digital elevation model as the determinant of groundwater flow. The strong correlation between land surface topography and the direction of groundwater flow was first shown theoretically by ref. 43 and has since been widely validated and accepted in the field of hydrology (44, 45). We provide direct evidence of this correlation in New Hampshire at 13 sites for which detailed data on the groundwater table are available (*SI Appendix*, section L). For each PFAS-contaminated site and public well, we calculate its catchment area as the area from which water would flow to the given location, using the `Watershed` tool in Whitebox Tools.

In our base specification, we restrict attention to contaminated sites with concentrations above 1,000 ppt and define nearby contaminated sites as those within 5 km of a well (*SI Appendix*, section D assesses robustness to the 1,000 ppt and 5 km thresholds). If there exists at least one nearby contaminated site within the catchment area of a well, then we classify the well as downgradient and match it to the nearest such site. Otherwise, if a well is within the catchment areas of all nearby contaminated sites, then we classify it as upgradient and match it to the nearest such site. The wells that are classified as to the side are those that do not have any nearby contaminated site within their catchment areas but that also have some nearby contaminated site that is not clearly downstream of the well. We match these wells to the nearest contaminated site. If a well does not have any contaminated site within 5 km, then we drop the well from the sample. *SI Appendix*, section K provides more details of groundwater calculations and well classification. Our classification of wells as upgradient, downgradient, or to the side is conservative in that we place all wells that are plausibly treated by a nearby contaminated site into the downgradient group and we reserve the upgradient group only for wells that are clearly not treated by any nearby contaminated site (i.e., that are clearly a good control population). *SI Appendix*, section G shows that downgradient classification is positively correlated with the probability of PFAS at a well from ref. 56 and that upgradient classification is negatively correlated with the probability of PFAS at a well from ref. 56.

To assign mothers to public wells, we overlay a map of public water system (PWS) service areas onto the set of points representing the location of maternal residences, as found in the birth record data. We remove all individuals who reside in the service area of a system that receives its water solely from surface water sources or imports its water from nonlocal sources. We also discard all births to mothers who reside outside of the service area for any PWS. With the PWS of each mother determined, we attribute a mother's water source to the well within that PWS that is nearest to her residence. *SI Appendix*, section G assesses robustness to this assignment.

Our final sample drops individuals with missing data for any of the covariates used in our regression framework. (*SI Appendix*, section G shows that including these covariates does not appreciably alter our results. In addition, Fig. 2 includes specifications in which we drop medical and demographic controls and do not drop individuals missing the dropped covariates. These specifications again show that missing covariates do not drive our results.) Our final sample contains 11,539 births. *SI Appendix*, section C details how many births survive each of our sample restrictions and contains balance tables.

Regression Model: Effects of Being Downgradient. Our main analysis takes advantage of a spatial discontinuity: The groundwater beneath some drinking water wells contains PFAS from a contaminated site, whereas the groundwater beneath other drinking water wells does not contain PFAS from a contaminated site. Spatial boundaries have been used to identify causal effects in other contexts

(57–59). In order for this analysis to capture the causal impact of PFAS on infant health outcomes, it must be the case that mothers receiving water from wells that are near PFAS-contaminated sites but upgradient of those sites would, but for PFAS, have similar infant health outcomes as mothers receiving water from wells that are near PFAS-contaminated sites but downgradient of some contaminated site, conditional on the control variables. *SI Appendix*, section C shows that mothers receiving water from wells that are near PFAS-contaminated sites but downgradient of a contaminated site are, if anything, of higher socioeconomic status than mothers receiving water from wells that are near PFAS-contaminated sites but upgradient of those contaminated sites. *SI Appendix*, section E confirms that these differences imply that, if not for PFAS exposure, infant health outcomes would be broadly similar among these mothers and, if anything, would tend to be better among downgradient mothers.

In our main analysis, we estimate equations of the following form:

$$H_{ict} = \gamma Up\_or\_Down_{ict} + \beta Down_{ict} + \Gamma X_{ict} + \eta_c + \nu_t + \epsilon_{ict} \quad [1]$$

for birth i in county c and month t. In each equation, H_{ict} is the health outcome of interest, which can be an indicator for mortality within one year of birth, for a preterm birth, for a low-weight birth, or for a subcategory thereof. $Up\_or\_Down_{ict}$ is an indicator that is equal to 1 if the mother in birth i receives water from a well that is classified as either upgradient or downgradient of a contaminated site and is equal to 0 if the well is classified as to the side, relative to groundwater flow. $Down_{ict}$ is an indicator variable that is equal to 1 if the mother in birth i receives water from a well that is classified as downgradient and is equal to 0 if the well is not. The coefficient γ gives the effect of a well being upgradient of a nearby contaminated site relative to being to the side. The coefficient of interest is β, which gives the effect of a well being downgradient of some nearby contaminated site relative to being upgradient of all nearby contaminated sites. X_{ict} is a vector that contains an extensive set of demographic, environmental, medical, house, and well control variables, detailed in *SI Appendix*, section B. η_c and ν_t are county and year-by-month fixed effects, respectively.

In the main text, we report results in terms of the percentage change in the base rate: Letting $\hat{\beta}$ indicate the estimated coefficient and $\bar{H}$ indicate the average value of H_{ict} in the estimation sample, we report $100 \cdot \hat{\beta}/\bar{H}$. We hold $\bar{H}$ fixed across robustness checks. We estimate SEs that are robust to two-way clustering at the contaminated site level (in order to account for possible unobserved correlation among mothers near a given contaminated site) and at the year-by-month level (to account for possible unobserved correlation among mothers within a time period).

Including fixed effects at the county level absorbs the average effect of being in one county or another. Our estimates therefore only use the variation in outcomes across upgradient mothers and downgradient mothers within the same county, not across counties (60). Analogously, our year-month fixed effects mean that our estimates use the variation in outcomes across upgradient mothers and downgradient mothers who gave birth within the same month of a given year. And our vector of control variables means that our estimates implicitly compare upgradient mothers to downgradient mothers who have similar houses, wells, demographics, etc. This means that, while we use data on individual births across different counties, months, and maternal characteristics, our estimates implicitly compare upgradient mothers and downgradient mothers who gave birth within the same month and the same county and who have similar houses, wells, demographics, etc.

Other pollutants besides PFAS could differ between upgradient and downgradient mothers. Indeed, some other pollutants are known to co-occur with PFAS. To the extent that other pollutants do covary with PFAS around contaminated sites, our estimates capture the health impact of PFAS and associated pollutants. The primary water pollutants found to covary with PFAS are 1,4-dioxane and chromium-6 (61). However, both were regulated in New Hampshire prior to our study period, and are therefore likely to be removed by existing water system technologies (62, 63). Moreover, 1,4-dioxane is not known to affect reproductive or developmental outcomes (64), and water systems that violate chromium standards must warn customers only of the possibility of allergic dermatitis (65). The co-occurring pollutants that would affect our health estimates are unregulated pollutants such as microplastics, pharmaceuticals, and species of PFAS beyond PFOA and PFOS, provided that they are present in groundwater during our study period. Below we discuss the implications of these emerging pollutants for our social cost estimates.

Regression Model: Effects of Predicted PFAS. In our secondary analysis, we use a two-sample instrumental variables (IV) model (60), in which we first predict PFAS at the well level (a form of exposure estimation) and then relate predicted well-level PFAS to birth outcomes. This analysis allows us to understand how different concentrations of PFAS at contaminated sites–and wells–affect infant health, which is useful for moving to a national analysis of the potential impact of PFAS on infant health. We take this two-sample approach because we do not observe PFAS contamination at all of the wells in our sample: In a first-stage regression, we predict well-level PFAS contamination given the spatial relationship between the contaminated site and the well, soil characteristics, and local control variables, and in a second stage, we regress infant health outcomes on predicted PFAS contamination from the first stage. This IV approach can be used to handle issues of measurement error (here, missing well-level contamination data) under two assumptions. First, the instruments (downgradient indicator, distance from the contaminated site to the well, and soil characteristics) must be correlated with well-level PFAS contamination but otherwise uncorrelated with infant health outcomes conditional on the control variables. Second, the wells where we observe PFAS contamination must be similar to the wells where we do not observe PFAS contamination.

In the first stage, we use data on wells w in county c tested for PFAS at month t to estimate PFAS concentrations as a function of observable characteristics of a well w and its associated contaminated site j:

$$\begin{aligned}
&\operatorname{asinh}(PFAS_{wct}) \\
=&\lambda_1 Up_or_Down_{wjc} + \lambda_2 Down_{wjc} + \lambda_3 SP_{jc} + \lambda_4 AWC_{wc} \\
&+ \lambda_5 Clay_{wc} + \lambda_6 Sand_{wc} + \lambda_7 Silt_{wc} \\
&+ \lambda_8 \ln(dist_{wjc}) + \lambda_9 Down_{wjc} SP_{wc} + \lambda_{10} Down_{wjc} AWC_{wc} \\
&+ \lambda_{11} Down_{wjc} Clay_{wc} + \lambda_{12} Down_{wjc} Sand_{wc} \\
&+ \lambda_{13} Down_{wjc} Silt_{wc} + \lambda_{14} Down_{wjc} \ln(dist_{wjc}) \\
&+ \lambda_{15} \operatorname{asinh}(SitePFAS_{jc}) + \Gamma \tilde{X}_{wc} + \gamma t + \epsilon_{wct}. \quad [2]
\end{aligned}$$

Here, $PFAS_{wct}$ is the measured level of PFAS in well w, which we transform with the inverse hyperbolic sine (*asinh*) because our data include a large number of zeros. $Up\_or\_Down_{wc}$ and $Down_{wc}$ are assigned as in the primary specification. In addition to whether a well is downgradient of a contaminated site, we use a series of instruments for well-level PFAS contamination, including the measured soil porosity (mm/m) SP_{wc} [which affects the speed at which PFAS migrate horizontally and to the groundwater table (44)]; well w's available water capacity (mm/m) AWC_{wc}; the fractions of clay, sand, and silt particles in the soil ($Clay_{wc}$, $Sand_{wc}$, and $Silt_{wc}$, respectively), which control how water and PFAS move through the soil to reach groundwater (34, 66); and the distance from well w to contaminated site j, $dist_{wjc}$. We record $SitePFAS_{jc}$ as the PFAS attributed to a matched contaminated site by the Northeastern University PFAS Lab (46), which does not vary over time. The vector $\tilde{X}_{wc}$ of well-level control variables includes an indicator for whether a well is a residential well; Census tract-level median house sales price, median income, number of housing units, and percent of the workforce employed in manufacturing; Census block group-level temperature and particulate matter (PM2.5) pollution; the number of Toxics Release Inventory Program sites within 5km of the well; wind transport of PFAS (*SI Appendix*, section A.6); and the surface elevation of the well. We allow for a linear time trend, γt, in order to capture the migration of PFOA and PFOS through soils and into groundwater over time. *SI Appendix*, section J reports the results of estimating regression Eq. **2**. It also shows that predicted PFAS correlates with our downgradient classification.

Once we have estimated regression Eq. **2**, we use the estimated coefficients to predict $\widehat{asinh(PFAS_{ict})}$, which is a transformation of PFAS at the well used to supply water to the mother in birth i. We then estimate:

$$\begin{aligned}
H_{ict} =& \beta \operatorname{asinh}(\widehat{PFAS_{ict}}) + \lambda \operatorname{asinh}(SitePFAS_{jc}) \\
&+ \Gamma \tilde{X}_{ict} + \eta_c + \nu_t + \epsilon_{ict}. \quad [3]
\end{aligned}$$

H_{ict} and the fixed effects η_c and ν_t are as in regression Eq. **1**. $\tilde{X}_{ict}$ is the same as X_{ict} in Eq. **1** except without site-level PFAS (here pulled out separately) and without distance from the well to the contaminated site (here used an instrument). For those households with wells within 5 km of a PFAS-contaminated site, the coefficient β identifies the effect of receiving water from a well with marginally greater PFAS relative to receiving water from a well with marginally less PFAS. We bootstrap the SEs (*SI Appendix*, section J). *SI Appendix*, section G reports the estimated infant health effects of being downgradient implied by this two-sample IV model, obtained by multiplying the estimated $\hat{\beta}$ from regression Eq. **3** by the difference in predicted PFAS for downgradient and upgradient wells. It shows that these estimates are similar to the results of our primary analysis. *SI Appendix*, section H tests for nonlinear effects of PFAS exposure. It shows that the effects of PFAS are approximately constant in the domain of the data.

Calculating Effects Across the United States. In addition to the data sources used in the primary analysis, we obtain county-level data on the number of births in 2010 from the Centers for Disease Control Wonder service and population data from the 2010 Census at the block group (CBG) level. We use these data to impute CBG-level births:

$$\text{births}_g = \frac{\text{population}_g}{\text{population}_c}\text{births}_c, \qquad [4]$$

where g indexes CBGs and c is the county that contains the CBG.

To attribute exposure to PFAS at the CBG level, we calculate the catchment area for each contaminated site throughout the United States for which we have PFAS data from ref. 46 and for each CBG centroid that is within 5 km of a contaminated site. We classify each CBG as upgradient, downgradient, or to the side based on the relation of its centroid to nearby contaminated sites, following the procedure used for wells.

To attribute PFAS exposure to the residents within a given CBG, we fit the following equation to our New Hampshire data:

$$\begin{aligned}&\text{asinh}(PFAS_{wct})\\ =&\lambda_0 + \lambda_1 Up\_or\_Down_{wjc} + \lambda_2 Down_{wjc} + \lambda_3 SP_{jc} + \lambda_4 AWC_{wc}\\ &+ \lambda_5 Clay_{wc} + \lambda_6 Sand_{wc} + \lambda_7 Silt_{wc} + \lambda_8 \ln(dist_{wjc})\\ &+ \lambda_9 Down_{wjc} SP_{jc} + \lambda_{10} Down_{wjc} AWC_{wc}\\ &+ \lambda_{11} Down_{wjc} Clay_{wc} + \lambda_{12} Down_{wjc} Sand_{wc}\\ &+ \lambda_{13} Down_{wjc} Silt_{wc} + \lambda_{14} Down_{wjc} \ln(dist_{wjc})\\ &+ \lambda_{15} \text{asinh}(SitePFAS_{jc}) + \varepsilon_{wct}.\end{aligned}$$

This regression modifies regression Eq. **2** to include only explanatory variables that we observe nationwide. We use the fitted equation to predict the inverse hyperbolic sine of PFAS exposure at the CBG level from contaminated sites in the 11 U.S. states with widespread testing. Multiplying $\widehat{PFAS}_{ict}$ by the estimated $\hat{\beta}$ from regression Eq. **3** and births_g from Eq. **4** gives us the predicted change in reproductive outcomes within CBG g.

Summing across the CBGs within 5 km of a contaminated site for which we have data yields the predicted change in births in these 11 states. (Within these 11 states, more than 87% of the population lives in a CBG for which we have data from the Wonder service, so we ignore the possibility of health impacts in CBGs for which we lack data.) We extrapolate to the 48 states of the contiguous U.S. by multiplying the total effect in these 11 states by the ratio of the contiguous U.S. population to the population of these 11 states. That ratio is 2.91. This extrapolation assumes both that we have comprehensively tallied the effects of PFAS in those 11 states (which would not be true if there are untested contaminated sites) and that contamination in these 11 states is representative of contamination in the other 37 states (which would not be true if states that chose to test did so because of well-founded special concern about PFAS or if states that chose not to test did so because of well-founded special concern about high liability for PFAS).

Social Costs of Health Outcomes. From ref. 40, the social cost of each extremely preterm birth is $445,852; the social cost of each very preterm birth is $241,769; and the social cost of each moderately preterm birth is $36,728. Each of these figures incorporates differential costs borne by preterm individuals due to differentials in medical care, maternal delivery costs, early intervention services, special education services, assistive devices, and lost labor market productivity, all relative to a full-term birth. In order to make each cost relative to the next-most severe category, we difference the social cost of an extremely preterm birth from that of a very preterm birth to arrive at a social cost of $204,083 per extremely preterm birth. And we difference the social cost of a very preterm birth from that of a moderately preterm birth to arrive at a social cost of $205,041 per moderately preterm birth. The social cost of a moderately preterm birth is $36,728, as in ref. 40.

To calculate the social cost of the different severities of low-weight births, we closely follow Appendix F of ref. 39. We discuss these calculations in more depth in *SI Appendix*, section I. Most importantly, unlike the social costs used for preterm births, we directly account for differential mortality risks across the distribution of birthweight when calculating relative social costs for each severity group. When calculating the social costs of increased mortality risk, we conservatively use different values of life based on, among others, differential life expectancies for infants born in each low-weight bin (49). We obtain a social cost of $2,636,968 for each extremely low-weight birth (relative to a very low-weight birth) and of $1,767,021 for each very low-weight birth.

To calculate the social cost of infant mortality, we again use the results from ref. 49. In particular, we assume that if an infant who died within their first year of life had instead remained alive, then they would have had the quality of life of an infant who was born at an extremely low weight. This yields a social cost of $4,230,796 for each infant death.

We interpret our social cost estimates as reflecting the benefit of cleaning up PFAS. Yet, as discussed above, our health estimates could reflect contaminants that correlate with PFAS, especially unregulated contaminants. The primary technology for removing PFAS at the scale of a treatment plant is granular activated carbon filtration. This treatment captures many of the unregulated contaminants that may correlate with PFAS, including microplastics (67) and pharmaceuticals (68). The primary exceptions are short-chain PFAS, which are less effectively removed than long-chain PFAS such as PFOA and PFOS by granular activated carbon (69–72). However, short-chain PFAS have much smaller concentrations than PFOA and PFOS in groundwater systems (36, 37). Moreover, some of the PFAS-contaminated sites in our sample were closed before 2000 (46). Long-chain PFAS were the dominant forms produced prior to 2010, and it takes time for PFAS to migrate through soils to groundwater (33, 34). As a result, our estimated health impacts over 2010–2019 are likely to reflect exposure to long-chain PFAS more than short-chain PFAS, consistent with many groundwater PFAS concentration datasets that show much greater concentrations of PFOA and PFOS than short-chain PFAS (37).

Data, Materials, and Software Availability. All nonrestricted data and associated code are available on Figshare (73). Restricted data include (1) shapes of public water system service areas in New Hampshire, (2) the locations of public water wells, and (3) geocoded natality data for all births in New Hampshire. 1. Water and Sewer Line Distribution Areas (a. Department: New Hampshire Department of Environmental Services, Drinking Water and Groundwater Bureau, Email: dwgbinfo@des.nh.gov; b. Point of Contact: Kelsey Vaughn, Email: kelsey.vaughn@des.nh.gov; c. Instructions to obtain data: Email department/person of contact to apply for access). 2. Public Water Supplies (a. Department: New Hampshire Department of Environmental Services, Drinking Water and Groundwater Bureau, Email: dwgbinfo@des.nh.gov; b. Point of Contact: Pierce Rigrod, Email: pierce.laskey-rigrod@des.nh.gov; c. Instructions to obtain data: Email department/person of contact to apply for access). 3. New Hampshire Natality and Mortality Data (a. Department: New Hampshire Department of Health and Human Services, Email: HealthStatisticsMailFile@dhhs.nh.gov; b. Instructions to obtain data: https://www.dhhs.nh.gov/programs-services/population-health/health-statistics-informatics/vital-records-birth-death-data).

ACKNOWLEDGMENTS. Support to B.G., W.H., and D.L. was provided by the Technology Research Initiative Fund/Water, Environmental and Energy Solutions Initiative administered by the University of Arizona Office for Research, Innovation and Impact, funded under Proposition 301, the Arizona Sales Tax for Education Act, in 2000. Support to A.L. was provided by NSF Grant SES-2149335. This work used High Performance Computing resources supported by the University of Arizona Technology and Research Initiative Fund, University Information Technology Services, and Research, Innovation, and Impact and maintained by the UArizona Research Technologies department. Melina Lew provided graphical design assistance, supported by the Arizona Institute for Resilience. R.B. thanks the American Economic Association Mentoring Program for travel support. Portions of the paper were developed from the thesis of R.B.

Author affiliations: [a]Cornerstone Research, Los Angeles, CA 90013; [b]Department of Hydrology and Atmospheric Sciences, University of Arizona, Tucson, AZ 85719; [c]Private address, New Orleans, LA, 70117; [d]Department of Economics, University of Arizona, Tucson, AZ 85721; and [e]National Bureau of Economic Research, Cambridge, MA 02138

1. A. Cordner *et al*., The true cost of pfas and the benefits of acting now. *Environ. Sci. Technol.* **55**, 9630–9633 (2021).
2. S. E. Fenton *et al*., Per- and polyfluoroalkyl substance toxicity and human health review: Current state of knowledge and strategies for informing future research. *Environ. Toxicol. Chem.* **40**, 606–630 (2021).
3. M. G. Evich *et al*., Per- and polyfluoroalkyl substances in the environment. *Science* **375**, eabg9065 (2022).
4. I. T. Cousins, J. H. Johansson, M. E. Salter, B. Sha, M. Scheringer, Outside the safe operating space of a new planetary boundary for Per- and Polyfluoroalkyl Substances (PFAS). *Environ. Sci. Technol.* **56**, 11172–11179 (2022).
5. S. Kurwadkar *et al*., Per- and polyfluoroalkyl substances in water and wastewater: A critical review of their global occurrence and distribution. *Sci. Total Environ.* **809**, 151003 (2022).
6. H. A. Kaboré *et al*., Worldwide drinking water occurrence and levels of newly-identified perfluoroalkyl and polyfluoroalkyl substances. *Sci. Total Environ.* **616–617**, 1089–1100 (2018).
7. J. L. Domingo, M. Nadal, Human exposure to per- and polyfluoroalkyl substances (PFAS) through drinking water: A review of the recent scientific literature. *Environ. Res.* **177**, 108648 (2019).
8. K. L. Smalling *et al*., Per- and polyfluoroalkyl substances (PFAS) in United States tapwater: Comparison of underserved private-well and public-supply exposures and associated health implications. *Environ. Int.* **178**, 108033 (2023).
9. B. Xu *et al*., PFAS and their substitutes in groundwater: Occurrence, transformation and remediation. *J. Hazard. Mater.* **412**, 125159 (2021).
10. P. B. McMahon *et al*., Perfluoroalkyl and polyfluoroalkyl substances in groundwater used as a source of drinking water in the Eastern United States. *Environ. Sci. Technol.* **56**, 2279–2288 (2022).
11. A. M. Calafat, L. Y. Wong, Z. Kuklenyik, J. A. Reidy, L. L. Needham, Polyfluoroalkyl chemicals in the U.S. population: Data from the national health and nutrition examination survey (NHANES) 2003-2004 and comparisons with NHANES 1999-2000. *Environ. Health Perspect.* **115** 1596–1602 (2007).
12. K. Kato, L. Y. Wong, L. T. Jia, Z. Kuklenyik, A. M. Calafat, Trends in exposure to polyfluoroalkyl chemicals in the U.S. population: 1999–2008. *Environ. Sci. Technol.* **45**, 8037–8045 (2011).
13. N. M. Brennan, A. T. Evans, M. K. Fritz, S. A. Peak, H. E. von Holst, Trends in the regulation of per- and polyfluoroalkyl substances (PFAS): A scoping review. *Int. J. Environ. Res. Public Health* **18**, 10900 (2021).
14. S. R. Feingold, J. Esterkin, K. E. Deal, D. C. Jordan, Exploring the universe of PFAS regulation and litigation (2023). https://www.morganlewis.com/pubs/2023/04/exploring-the-universe-of-pfas-regulation-and-litigation.
15. X. Lim, Could the world go PFAS-free? Proposal to ban 'forever chemicals' fuels debate *Nature* **620**, 24–27 (2023).
16. Z. Abunada, M. Y. D. Alazaiza, M. J. K. Bashir, An overview of per- and polyfluoroalkyl substances (PFAS) in the environment: Source, fate, risk and regulations. *Water* **12**, 3590 (2020).
17. E. Koustas *et al*., The navigation guide–evidence-based medicine meets environmental health: Systematic review of nonhuman evidence for PFOA effects on fetal growth. *Environ. Health Perspect.* **122**, 1015–1027 (2014).
18. W. Austin, R. Mueller, Forever chemicals: Challenges and opportunities for researchers. *Rev. Environ. Econ. Policy* **19**, 118–130 (2025).
19. C. Care, A human health review of PFOS and PFOA (Tech. Rep. 42, Cooperative Research Centre for Contamination Assessment and Remediation of the Environment, 2016).
20. M. Kirk *et al*. The PFAS health study: Systematic literature review (Tech. Rep., The Australian National University, Canberra, 2018).
21. Z. Liew, H. Goudarzi, Y. Oulhote, Developmental exposures to perfluoroalkyl substances (PFASs): An update of associated health outcomes. *Curr. Environ. Health Rep.* **5**, 1–19 (2018).
22. National Science and Technology Council, Per- and polyfluoroalkyl substances (PFAS) report (Tech. Rep., 2023).
23. M. A. Verner *et al*., Associations of perfluoroalkyl substances (PFAS) with lower birth weight: An evaluation of potential confounding by glomerular filtration rate using a physiologically based pharmacokinetic model (PBPK). *Environ. Health Perspect.* **123**, 1317–1324 (2015).
24. K. Steenland, V. Barry, D. Savitz, Serum perfluorooctanoic acid and birthweight: An updated meta-analysis with bias analysis. *Epidemiology* **29**, 765 (2018).
25. R. Vestergren, I. T. Cousins, Tracking the pathways of human exposure to perfluorocarboxylates. *Environ. Sci. Technol.* **43**, 5565–5575 (2009).
26. S. Hurley *et al*., Preliminary associations between the detection of perfluoroalkyl acids (PFAAs) in drinking water and serum concentrations in a sample of California women. *Environ. Sci. Technol. Lett.* **3**, 264–269 (2016).
27. L. A. Nolan, J. M. Nolan, F. S. Shofer, N. V. Rodway, E. A. Emmett, The relationship between birth weight, gestational age and perfluorooctanoic acid (PFOA)-contaminated public drinking water. *Reproduct. Toxicol.* **27**, 231–238 (2009).
28. Y. Zhu, S. M. Bartell, Per- and polyfluoroalkyl substances in drinking water and birthweight in the US. *Environ. Epidemiol.* **4**, e0107 (2020).
29. G. Waterfield, M. Rogers, P. Grandjean, M. Auffhammer, D. Sunding, Reducing exposure to high levels of perfluorinated compounds in drinking water improves reproductive outcomes: Evidence from an intervention in Minnesota. *Environ. Health* **19**, 42 (2020).
30. J. M. Liddie, L. A. Schaider, E. M. Sunderland, Sociodemographic factors are associated with the abundance of PFAS sources and detection in U.S. community water systems. *Environ. Sci. Technol.* **57**, 7902–7912 (2023).
31. M. Marcus, R. Mueller, Unregulated contaminants in drinking water: Evidence from PFAS and housing prices. *J. Environ. Econ. Manag.* **125**, 102987 (2024).
32. J. G. Zivin, M. Neidell, W. Schlenker, Water quality violations and avoidance behavior: Evidence from bottled water consumption. *Am. Econ. Rev.* **101**, 448–453 (2011).
33. M. L. Brusseau, R. H. Anderson, B. Guo, PFAS concentrations in soils: Background levels versus contaminated sites. *Sci. Total Environ.* **740**, 140017 (2020).
34. B. Guo, J. Zeng, M. L. Brusseau, A mathematical model for the release, transport, and retention of per- and polyfluoroalkyl substances (PFAS) in the vadose zone. *Water Resour. Res.* **56**, e2019WR026667 (2020).
35. EPA, 2006 community water system survey. Volume II: Detailed tables and survey methodology (Tech. Rep. 815-R-09-002, Office of Water, United States Environmental Protection Agency, 2009).
36. X. C. Hu *et al*., Detection of poly- and perfluoroalkyl substances (PFASs) in U.S. drinking water linked to industrial sites, military fire training areas, and wastewater treatment plants. *Environ. Sci. Technol. Lett.* **3**, 344–350 (2016).
37. G. R. Johnson, M. L. Brusseau, K. C. Carroll, G. R. Tick, C. M. Duncan, Global distributions, source-type dependencies, and concentration ranges of per- and polyfluoroalkyl substances in groundwater. *Sci. Total Environ.* **841**, 156602 (2022).
38. J. Currie, J. Graff Zivin, K. Meckel, M. Neidell, W. Schlenker, Something in the water: Contaminated drinking water and infant health. *Can. J. Econ.* **46**, 791–810 (2013).
39. J. Currie, M. Mueller-Smith, M. Rossin-Slater, Violence while in utero: The impact of assaults during pregnancy on birth outcomes. *Rev. Econ. Stat.* **104**, 525–540 (2022).
40. N. J. Waitzman, A. Jalali, S. D. Grosse, Preterm birth lifetime costs in the United States in 2016: An update. *Sem. Perinatol.* **45**, 151390 (2021).
41. World Health Organization, Preterm birth (2023). https://www.who.int/news-room/fact-sheets/detail/preterm-birth.
42. United Nations Children's Fund and World Health Organization, Low birthweight: Country, regional, and global estimates, (Tech. Rep., UNICEF, New York, 2004).
43. J. Tóth, A theoretical analysis of groundwater flow in small drainage basins. *J. Geophys. Res.* **68** 4795–4812 (1963).
44. R. A. Freeze, J. A. Cherry, *Groundwater* (Prentice Hall, Englewood Cliffs, N.J, 1979).
45. G. F. Pinder, M. A. Celia, *Subsurface Hydrology* (Wiley-Interscience, Hoboken N.J., 2006).
46. PFAS Project Lab, Known PFAS contamination site database, Version: October 9, 2022 (2022). https://pfasproject.com/pfas-sites-and-community-resources/.
47. J. Rogowski, Cost-effectiveness of care for very low birth weight infants. *Pediatrics* **102**, 35–43 (1998).
48. T. J. Matthews, M. F. MacDorman, M. E. Thoma, Infant mortality statistics from the 2013 period linked birth/infant death data set. *Nat. Vital Stat. Rep.* **64**, 1–30 (2015).
49. D. M. Cutler, E. Meara, "The technology of birth: Is it worth it?" in *Frontiers in Health Policy Research, Volume 3*, A. M. Garber, Ed. (MIT Press, 2000), pp. 33–68.
50. D. Salvatore *et al*., Presumptive contamination: A new approach to PFAS contamination based on likely sources. *Environ. Sci. Technol. Lett.* **9**, 983–990 (2022).
51. E. P. Agency, PFAS national primary drinking water regulation. *Fed. Regist.* **89**, 32532–32757 (2024).
52. Black and Veatch, "PFAS National Cost Model Report" (Tech. Rep. WITAF 56, American Water Works Association, 2023).
53. I. Jacqz, T. Somuncu, J. Voorheis, *Fighting Fire with Fire(Fighting Foam): The Long Run Effects of PFAS Use at U.S. Military Installations* (Working paper CES 24-72, U.S. Census Bureau, Center for Economic Studies, 2024).
54. B. E. Blake, S. E. Fenton, Early life exposure to per- and polyfluoroalkyl substances (PFAS) and latent health outcomes: A review including the placenta as a target tissue and possible driver of peri- and postnatal effects. *Toxicology* **443**, 152565 (2020).
55. Earth System Research Center, University of New Hampshire, Lidar-derived Bare Earth DEM - NH (2022). https://granit.unh.edu/datasets/6b6e1b8af62d478396d6a8620ff45fcb/about.
56. A. K. Tokranov *et al*., Predictions of groundwater PFAS occurrence at drinking water supply depths in the United States. *Science* **386**, 748–755 (2024).
57. Y. Chen, A. Ebenstein, M. Greenstone, H. Li, Evidence on the impact of sustained exposure to air pollution on life expectancy from China's Huai River policy. *Proc. Natl. Acad. Sci. U.S.A.* **110**, 12936–12941 (2013).
58. K. Ito, Do consumers respond to marginal or average price? Evidence from nonlinear electricity pricing. *Am. Econ. Rev.* **104**, 537–563 (2014).
59. L. Muehlenbachs, E. Spiller, C. Timmins, The housing market impacts of shale gas development. *Am. Econ. Rev.* **105**, 3633–3659 (2015).
60. J. D. Angrist, J. S. Pischke, *Mostly Harmless Econometrics: An Empiricist's Companion* (Princeton University Press, Princeton, 2009).
61. J. L. Guelfo, D. T. Adamson, Evaluation of a national data set for insights into sources, composition, and concentrations of per- and polyfluoroalkyl substances (PFASs) in U.S. drinking water. *Environ. Pollut.* **236**, 505–513 (2018).

62. New Hampshire Department of Environmental Services, 1,4-Dioxane and Drinking Water, Environmental Fact Sheet DWGB-3-24 (2020). https://www.des.nh.gov/sites/g/files/ehbemt341/files/documents/2020-01/dwgb-3-24.pdf.
63. State of New Hampshire, Water Quality Standards, Monitoring, Treatment, Compliance, and Reporting, Code of Administrative Rules Env-Dw 700 (2020). https://www.des.nh.gov/sites/g/files/ehbemt341/files/documents/env-dw-700.pdf.
64. J. K. Anderson, W. B. Kappleman, "Review of the human health toxicology and ecotoxicology of 1,4-dioxane" in *Environmental Investigation and Remediation: 1,4-Dioxane and other Solvent Stabilizers*, T. K. Mohr, W. DiGuiseppi, J. Hatton, J. Anderson, Eds. (CRC Press, ed. 2, 2020), pp. 255–289.
65. State of New Hampshire, Public Notification by Water Systems, Code of Administrative Rules Env-Dw 800 (2019). https://www.des.nh.gov/sites/g/files/ehbemt341/files/documents/2020-01/Env-Dw%20800.pdf.
66. B. Guo, J. Zeng, M. L. Brusseau, Y. Zhang, A screening model for quantifying PFAS leaching in the vadose zone and mass discharge to groundwater. *Adv. Water Resour.* **160**, 104102 (2022).
67. N. A. Anuwa-Amarh, M. Dizbay-Onat, K. Venkiteshwaran, S. Wu, Carbon-based adsorbents for microplastic removal from wastewater. *Materials* **17**, 5428 (2024).
68. P. R. Rout, T. C. Zhang, P. Bhunia, R. Y. Surampalli, Treatment technologies for emerging contaminants in wastewater treatment plants: A review. *Sci. Total Environ.* **753**, 141990 (2021).
69. Z. R. Hopkins, M. Sun, J. C. DeWitt, D. R. Knappe, Recently detected drinking water contaminants: GenX and other per- and polyfluoroalkyl ether acids. *J. AWWA* **110**, 13–28 (2018).
70. M. Ateia, A. Maroli, N. Tharayil, T. Karanfil, The overlooked short- and ultrashort-chain poly- and perfluorinated substances: A review. *Chemosphere* **220**, 866–882 (2019).
71. D. M. Kempisty *et al*., Granular activated carbon adsorption of perfluoroalkyl acids from ground and surface water. *AWWA Water Sci.* **4**, e1269 (2022).
72. H. Smaili, C. Ng, Adsorption as a remediation technology for short-chain per- and polyfluoroalkyl substances (PFAS) from water–a critical review. *Environ. Sci. Water Res. Technol.* **9**, 344–362 (2023).
73. R. Baluja, B. Guo, W. Howden, A. Langer, D. Lemoine, PFAS-contaminated drinking water harms infants. Figshare. https://figshare.com/articles/dataset/PFAS-Contaminated_Drinking_Water_Harms_Infants/30601643. Deposited 13 November 2025.