

PFAS and Blood Pressure in Pregnancy: What the Puerto Rico PROTECT Cohort Tells Us

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A new study in *Environmental Pollution* (Varshavsky et al., 2026) adds to the growing body of research on exposure to per- and polyfluoroalkyl substances (PFAS) and the potential development of adverse human health effects, specifically blood pressure and hypertension among pregnant women.

Researchers followed over 500 pregnant women enrolled in the Puerto Rico PROTECT cohort from 2011–2022. PFAS were measured in blood serum once, at 16–20 weeks of pregnancy and were compared to blood pressure readings taken at that visit and again at 24–28 weeks of pregnancy. Four PFAS, perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorohexanesulfonic acid (PFHxS), and perfluorononanoic acid (PFNA), were examined individually, while perfluorodecanoic acid (PFDeA) and perfluoroundecanoic acid (PFUA) were simply deemed “detected or not.” Researchers also used a mixture method to estimate the combined effect of all six PFAS compounds. Statistical models were adjusted for age, pre-pregnancy body mass index and college education, and, in separate analyses of the individual PFAS, stratified by fetal sex and parity.

Across analyses, higher PFAS levels tended to line up with higher blood pressure, but individual blood pressure changes were small and statistically imprecise. The strongest signal was in the mixture analysis. A combined one-tertile increase across all six PFAS was associated with 2.14 odds ratio (OR; 95% CI: 1.32–3.46) of hypertension at 24–28 weeks of pregnancy. Stronger associations were also observed among first-time mothers and women carrying female fetuses.

Upon first blush, the 2.14 OR and mostly positive, though often imprecise, directions across many of the individual analyses seem to suggest the existence of a causal relationship between exposure to PFAS and the development of gestational hypertension. However, several limitations within the study, some acknowledged by the authors and others not, restrict such an interpretation.

Notably, “hypertension,” as defined and categorized by the study, does not mirror a clinical diagnosis of gestational hypertension. [The American College of Obstetricians and Gynecologists \(ACOG\)](#) requires elevated readings on two occasions, at least four hours apart, after 20 weeks of pregnancy, for a diagnosis of gestational hypertension to be rendered. However, the authors classify hypertension from a single reading at each visit, and the earlier readings (16–20 weeks) were taken at or before the 20-week benchmark; the only post-20-week reading was a single measurement at 24–28 weeks, which is where the 2.14 OR arises. Additionally, severity thresholds used in the classification of hypertension were drawn from general adult guidelines, not those from ACOG for gestational hypertension specifically, although the study attributes them to ACOG. Also, the authors did not study preeclampsia, nor did they have sufficient data on other potential sources of PFAS exposure within the cohort.

Moreover, pregnancy changes how PFAS shows up in blood. Blood volume expands substantially and the kidneys filter much faster. Both determinants shift serum PFAS concentrations in pregnant women independent of exposure levels. Because these adaptations differ between women and may vary in those who ultimately develop high blood pressure, they can influence the observed relationship in either direction. In particular, women who go on to develop high blood pressure may have blunted plasma volume expansion and reduced renal clearance, which would raise their serum PFAS and could produce a spurious positive association (i.e., reverse causation). This runs counter to the authors' suggestion that hemodilution would bias results toward an inverse association and thus strengthen their

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findings. In fact, the study did not report or adjust for kidney function (e.g., glomerular filtration rate) to test this phenomenon, which the field generally recognizes as a challenge.

Other factors, including obesity, maternal age, and nulliparity (no prior births), are also associated with elevated blood pressure. Age and BMI were adjusted for, but parity was not; the authors instead treated it as an effect modifier, even though nulliparity is also associated with higher serum PFAS (reflecting no prior lactational elimination), making it a likely confounder as well. Not every contributor could be measured or adjusted for, including diet, kidney function, and exposure to other chemicals, within the study population.

Despite its limitations, this is a thoughtful study that contributes to the growing body of research into PFAS exposure and the potential development of adverse health effects. For now, the study is likely best read as a hypothesis-generating signal rather than proof of causation, particularly as the 2.14 OR is one of the larger estimates among many examined without adjustment for multiple comparisons, and PFAS was measured only once, so temporality cannot be firmly established. Nonetheless, regulators, governmental agencies, businesses, and stakeholders should keep abreast of and closely evaluate medical and scientific developments as it relates to PFAS exposure pathways and development of human disease.