PFAS and Diabetes: Association Without Causation

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Given ongoing concerns surrounding exposure to per- and polyfluoroalkyl substances (PFAS) and potential adverse human health effects, researchers recently examined whether PFAS exposure could contribute to an increased risk of developing type 2 diabetes (T2D). Relying on electronic health records of more than 65,000 patients from Mount Sinai Hospital, investigators designed a “nested case-control study” to examine whether an association exists between diagnosis of T2D and exposure to PFAS. Plasma samples were collected at baseline and at the time of study to quantify and detect certain PFAS variants, specifically PFOS (perfluorooctane sulfonate), PFHxS (perfluorohexane sulfonic acid), PFNA (perfluorononanoic acid), PFOA (perfluorooctanoic acid), PFHpA (perfluoroheptanoic acid), PFDA (perfluorodecanoic acid) and PFHpS (perfluoroheptane sulfonic acid).

Although the study suggests there may be evidence that higher PFAS exposures could increase risk for T2D, a close reading highlights several noteworthy limitations. Critically, the authors agree the study does not establish temporality—a fundamental tenet of causality—between PFAS exposure and subsequent diagnosis of T2D. In other words, there is no evidence that exposure to PFAS causes T2D. The authors further note the limited sample size impacted the study's statistical power (i.e., the likelihood of observing a statistically significant effect when one truly exists) and that certain interactions and potential confounding variables unaccounted for, such as dietary intake patterns, could affect their findings.

While significant time and resources went into designing, developing and implementing this investigation and its findings will contribute to the growing body of data surrounding PFAS, this study nonetheless cannot—and does not—establish that exposure to PFAS leads to the development of T2D.