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Uranium isotopic composition as novel biomarker of chronic uranium exposure

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Until recently, the extent of uranium (U) exposure to the general population of the United States via drinking water has been underrecognized. Nearly two-thirds of community water systems, serving about 320 million people, have detectable U, with ~2% exceeding 30 µg/L, the US EPA maximum contaminant level. This widespread U exposure is a major public health concern, particularly for the Native American communities in Northern Plains and Colorado Plateau due to naturally occurring U in aquifers and widespread U mining near these communities. Uranium is an established nephrotoxicant at high levels of exposure and ingested U from drinking water targets the kidneys. Although, urinary U levels are an established biomarker of exposure, there are no reliable biomarkers for the biological interaction of U with the kidney. Chronic low-dose U exposure may affect kidney health negatively and when undetected, outcomes may become progressively degenerative and irreversible. For an early diagnosis of U induced kidney effects, it is crucial to identify markers that reveal U accumulation levels in the kidney. Here, we develop urinary U isotope ratios ($^{238}U/^{235}U$ expressed as $\delta^{238}U$) as sensitive markers for U induced kidney effects.

We report results from a pilot exposure study in which mice were exposed to 50 µg/L U in drinking water for 2, 7 and 14 days. We measure U concentrations and $\delta^{238}U$ in organs including the gastrointestinal tract, kidneys, and femurs of exposed mice. We observe significant U accumulation in femurs (up to 1553 ng/g) and kidneys (up to 1040 ng/g). Our preliminary data show that kidneys and femurs are enriched in ^{235}U by up to 0.3‰ ($\delta^{238}U \sim -0.3$‰) relative to drinking water. The average $\delta^{238}U$ of the gastrointestinal tract (stomach, cecum, colon, small intestine) were significantly different from that of the kidneys. Our preliminary $\delta^{238}U$ of the urine show ^{238}U enrichment relative to the kidneys due to preferential uptake of ^{235}U by kidneys. Our results, for the first time, reveal that U accumulation in kidneys is accompanied by measurable U isotopic fractionation. Future research in U isotopes to quantify kidney accumulation may enable intervention before irreversible damage occurs.