Hg stable isotopes as tracers for dietary Hg exposure and metabolic detoxification in humans

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Mercury (Hg) is a natural element whose global biogeochemical cycle has been profoundly changed by human activites. In its methylmercury (MeHg) form, Hg bioaccumulates along aquatic food chains and affects human and wildlife health. MeHg is neurotoxic to the developing foetus and young children and is thought to be linked to cardiovascular disease in adults. Humans are thought to mainly absorb toxic MeHg through dietary exposure. There is however substantial variation in dose-response among different populations. A better understanding of precise dietary exposure and metabolic breakdown of MeHg would help optimizing Hg risk management and environmental policy.

Hg stable isotope signatures show large variations across Earth surface reservoirs and within biological species. These variations result from the gradual fractionation of heavy/light and even/odd Hg isotopes during the multiple physicochemical processes that shuttle Hg across the Earth's surface. Two useful Hg isotope fingerprints, δ^{202} Hg and Δ^{199} Hg, characterize its source, or code for the transformations that Hg has undergone in its biogeochemical cycle.

Recent work on Hg isotopes in human hair has given insight into dietary Hg exposure. Hair Δ^{199} Hg, which has a photochemical origin and is not affected by Hg metabolism, appears to be a robust and conservative marker for the dietary source of MeHg. Amazonion river people have low Δ^{199} Hg that are identical to that of the fresh water fish they consume; similarly Europeans have higher Δ^{199} Hg corresponding to a mixed sea food diet, while North-Americans have elevated Δ^{199} Hg corresponding to the consumption of open ocean predator fish such as tuna.

In contrast, the δ^{202} Hg signature is affected by nearly all chemical transformations of Hg. Human hair δ^{202} Hg shows a systematic 2 ‰ enrichment in the heavier isotopes compared to the fish that was consumed across all three populations. We will show that the origin of this 2 ‰ enrichment potentially lies in the metabolic demethylation of MeHg, a detoxification mechanism that lowers MeHg exposure.