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# Baseline of Rubidium isotopic composition in mice organs

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Rubidium shares similar geochemical properties with Potassium, and is also involved in biological cycles where it usually substitutes to it. In the context of Alzheimer's disease (AD), there is a decrease in Rb concentration in serum of AD patient, while no effect is observed for K, suggesting an unusual decoupling between these elements [1]. This decrease is thought to be associated with dysfunctional Na+/K+-ATPase activity, and is a hallmark pathology of AD that results in an energy crisis preceding the formation of proteinaceous inclusions and neuron loss in the disease.

Recent studies have demonstrated the potential of isotopic measurements for detecting change in metal homeostasis associated with Alzheimer's disease (AD) [2]. For example, the Cu isotopic composition in the brain of a AD patient is lighter than for healthy ones, due to the formation of isotopically light Amyloid beta plaques in AD brains.

To gain insight into the change in Rb concentration observed in AD patient and explore the possibility of using Rb isotopes as a diagnostic tool for AD, we developed a method to analyze Rb stable isotopes in biological samples. The first step is to establish the Rb isotopic composition of various organs in a healthy state. Here we present the Rb isotopic composition of several organs in three-month-old mice by including brain, liver, and kidney using a Nu Sapphire MCICPMS. Additional data on other organs and body fluids will be presented at the conference.

Our initial findings indicate that the brain is enriched in the light isotope of Rb compared to the kidney and the liver, supporting the potential of Rb isotopes as a new tool to assess biological cycles of elements. Future investigations will involve testing the baseline in various organs and assessing the effect of aging before exploring Rb isotopes in mice and humans affected by AD. These findings hold promise for advancing our understanding of Rb in biological systems and its potential applications in AD diagnosis.

References:

[1] Roberts et al. (2016) *Acta Neuropathologica Communications,* 4:119

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