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Non-targeted metabolomics by high resolution mass spectrometry in HPRT knockout mice

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Abstract

Aims:

Lesch-Nyhan disease (LND) is characterized by hyperuricemia as well as neurological and neuropsychiatric symptoms including repetitive self-injurious behavior. Symptoms are caused by a deficiency of the enzyme hypoxanthine-guanine phosphoribosyltransferase (HPRT) as a result of a mutation on the X chromosome. To elucidate the pathophysiology of LND, we performed a metabolite screening for brain and serum extracts from HPRT knockout mice as an animal model for LND.

Main methods:

Analyses were performed by high performance liquid chromatography (HPLC)-coupled quadrupole time-of-flight mass spectrometry (QTOF-MS).

Key findings:

In brain extracts, we found six metabolites with significantly different contents in wild-type and HPRT-deficient mice. Two compounds we could identify as 5-aminoimidazole-4-carboxamide ribotide (AICAR) and 1-methylimidazole-4-acetic acid (1-MI4AA). Whereas AICAR was accumulated in brains of HPRT knockout mice, 1-MI4AA was decreased in these mice.

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