Effect of silicic acid and alcoholic beer intake on the excretion of chromium and vanadium and their deposition in the brains of mice chronically exposed to aluminium nitrate.

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The effect of aluminium (Al) exposure and silicon (Si) intake on the levels of chromium (Cr) and vanadium (V) in mouse brain was studied. Six-week-old male NMRI mice were divided into four groups. Three groups received $Al(NO_3)_3$, at a dose of 450 μg/ml, for three months, meanwhile the fourth group only received deionised water. The first group received aluminium nitrate (Al group); the second group aluminium nitrate and silicic acid (50 mg/ml); and the third group aluminium nitrate and commercial beer. Metals were monitored by ICP-OES in the right hemibrain, faeces, urine and blood. V was only detected in the faecal samples, being significantly higher in the Al group (4.132 vs. 3.383, 3.100 and 3.315; groups 4, 2 and 3, respectively; all in μg/g; *p*-value=0.038). Conversely, lower and significantly lower levels of Cr were detected in the faeces (2.867 vs. 3.155, 2.270 and 2.550 μg/g; *p*-value=0.296) and blood (0.187 vs. 0.158, 0.197 and 0.211 μg/l; *p*-value=0.013) in the Al group, respectively, meanwhile were lower in urine (0.00047 vs. 0.00069, 0.00060, 0.00065 μg/μmol creatinine; *p*-value=0.311), suggesting a potential effect of Al intoxication in the metabolism of Cr. These unknown effects might explain the lower levels of Cr that were detected in the intoxicated animals' brain (0.346 μg/g). Thus, intoxicated animals that were provided with Si showed Cr-brain levels slightly higher than in the Al-group (0.360 and 0.352 vs. 0.346 μg/g; *p*-value=0.552). Consumption of beer/silicic acid appears to partially block the negative effects of aluminium ingestion on the normal metabolism of chromium.

Keywords: chromium, vanadium, aluminium intoxication, silicon, brain accumulation.