



The 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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INTRODUCTION

Aim: To study the effect of aluminium (Al) in the levels of chromium (Cr) and vanadium (V) in mouse brain.

Animals were divided into four groups (n = 12). A control group consisting of mice that received only deionised water (named **Cont neg**). The other three groups received Al(NO₃)₃, at the level of 450 µg/ml dissolved in their drinking water, for three months. One group consisted of intoxicated mice that received only aluminium nitrate (**Cont post**); the other group (**Al + Si**) received aluminium nitrate and a solution of silicic acid (50 mg/ml); and the last group (**Al + Beer**) received aluminium nitrate and an amount of commercial beer equivalent to moderate to high consumption in humans (1 l/day). Elements were monitored in faeces, urine, blood and brain tissue with ICP-OES following previous methods (González-Muñoz et al., 2008).

V was only detected in the faecal samples (Table 1), being significantly higher in the Al group (4.132 vs. 3.383, 3.100 and 3.315; for 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 also 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).

CONCLUSIONS

Consumption of beer and silicic acid appears to **partially block the negative effects of aluminium ingestion in the normal metabolism of chromium**. Further studies are needed to investigate the potential interaction of Al in the absorption/excretion of V as this can also induce neurotoxicity.

MATERIAL AND METHODS

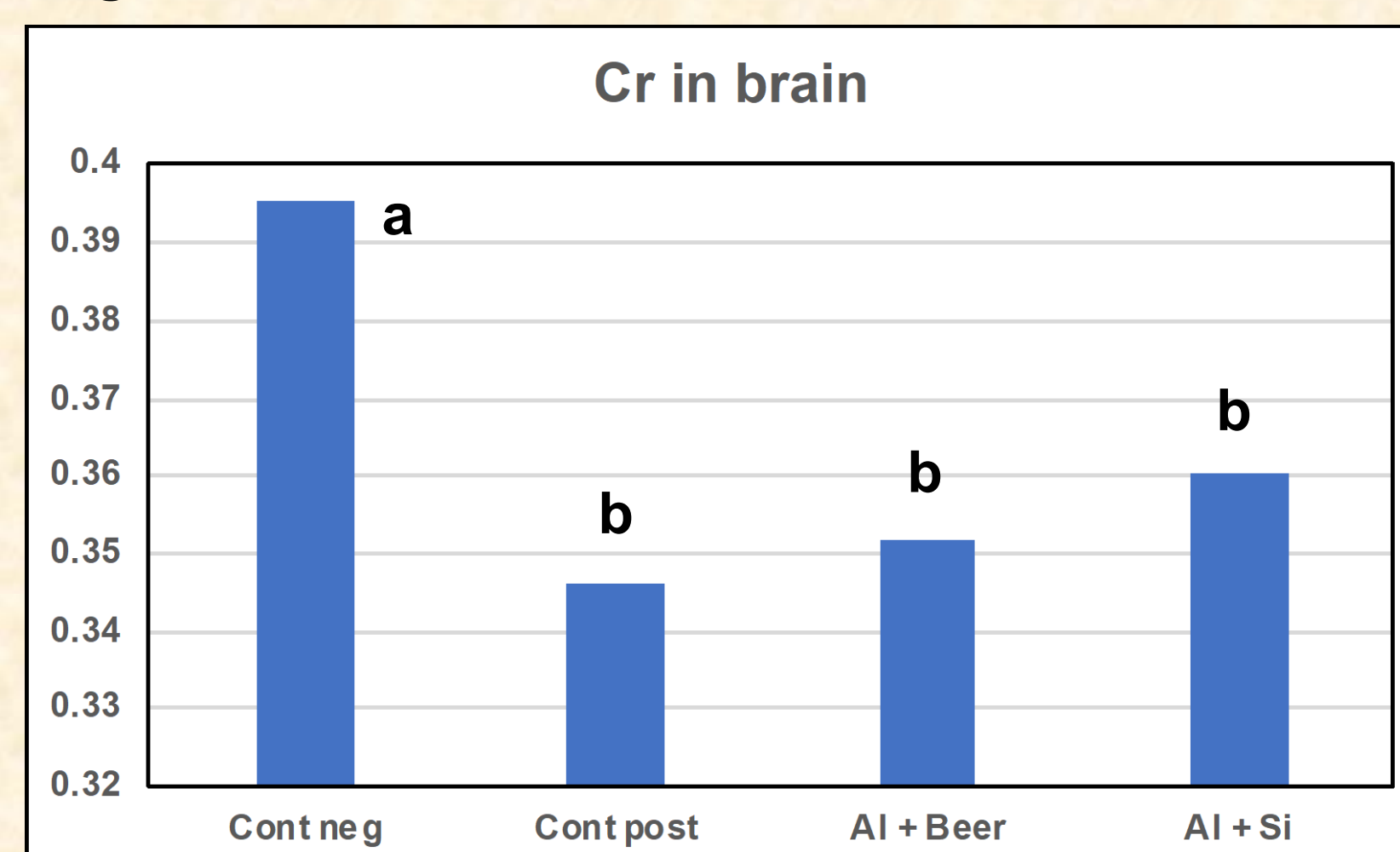
RESULTS & DISCUSSION

Table 1. Faecal, urine and blood Cr and V concentrations (µg/g) in the mice in the different experimental treatment groups

Sample	Control negative	Control positive	Al + Beer	Al + Si
Faeces (Cr)	3.15 ^a ± 2.18	2.87 ^b ± 0.60	2.55 ^b ± 0.91	2.27 ^b ± 0.77
Faeces (V)	3.38 ^a ± 0.83	4.13 ^b ± 0.69	3.31 ^a ± 0.89	3.10 ^a ± 1.22
Urine (Cr)	0.00069 ^a ± 0.00031	0.00047 ^b ± 0.00018	0.00065 ^a ± 0.00022	0.00060 ^a ± 0.00025
Blood (Cr)	0.158 ^a ± 0.017	0.187 ^b ± 0.048	0.211 ^b ± 0.053	0.197 ^b ± 0.037

Arithmetic mean results are presented as mean values S.D. Different letters in the same row indicate significantly different values.

Figure 1. Cr concentrations in brain of mice.



Different letters indicate significantly different values