

1st International Immunonutrition Workshop, Valencia, 3–5 October 2007, Valencia, Spain

Moderate ingestion of beer reduces inflammatory and oxidative brain events induced by aluminium in mice

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Al is a highly neurotoxic element and has been suggested to participate in the degeneration of cells in the brain of human subjects and experimental animals. Although the exact mechanism by which the metal may influence degenerative brain processes is unknown, there is evidence that exposure to Al causes an increase in both oxidative stress and inflammatory events⁽¹⁾. On the other hand, Si intake affects the bioavailability of Al at the gastrointestinal level, and therefore the Al accessibility to the brain. The aim of the present study was to examine the effect of supplementing Si in the diet, as silicic acid or by drinking beer, to prevent inflammation and oxidative stress in brain of mice exposed to oral Al. Four groups of male adult NMRI mice with an initial body weight of 30 g were studied: (1) negative control administered with deionised water; (2) positive control receiving 450 mg Al(NO₃)₃/kg body weight per d; (3) positive treatment group with Si (diet 2 plus 50 mg silicic acid/l; (4) positive treatment group with beer (diet 2 plus beer, equivalent to 1 litre/d for human subjects). The Al and Si contents of beer and brain tissue were measured by inductively-coupled plasma MS and atomic emission spectrometry respectively following wet ashing of the organic matter. Whole right hemibrains were dissected, frozen, homogenized and analysed for mineral content and mRNA for superoxide dismutase (SOD), catalase, glutathione peroxidase (GPx) and TNF α .

Brain Al levels were significantly lower ($P < 0.01$) for the negative control animals than for those of groups 2–4. Si intake in the form of beer lowered, although not significantly (2.45 μ g/g v. 3.85 μ g/g), the brain Al levels of intoxicated mice (group 2). Quantitative RT-PCR showed that administration of Al significantly decreased levels of both SOD and catalase mRNA for group 2, which were normal for the brains of mice receiving silicic acid or beer. Furthermore, for group 2 TNF α and GPx mRNA levels were significantly increased ($P < 0.05$; Figure) suggesting that Al induced inflammation and brain damage. These levels were approximately normal for groups 3 and 4 after consuming Si as silicic acid and beer respectively ($P < 0.05$). There was a significant correlation between brain Si and Al levels and the expression of some brain enzymes.

The results suggest that moderate beer consumption, by means of its Si content, effectively protects against the neurotoxic effects of Al. However, consumption of strong beers should be avoided because of the well-known negative effect of alcohol on the brain.

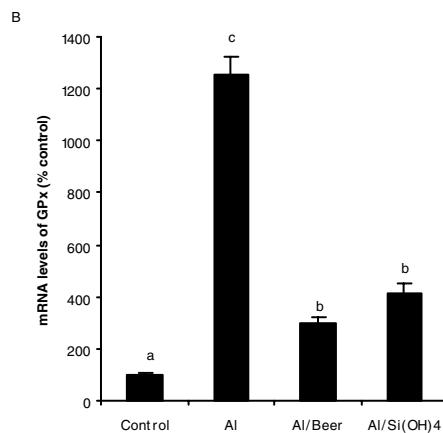


Figure. The GPx values, expressed in arbitrary units, are means with their standard errors represented by vertical bars for determinations for twelve mice. ^{a,b,c}Means with unlike superscript letters were significantly different ($P < 0.05$; Kruskal-Wallis and non-parametric multiple comparison test).

This work has been supported by the Asociación de Cerveceros Españoles and project AGL-2005-07204-C02-01.

1. Campbell A (2006) *J Alzheimers Dis* 10, 165–172.