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Experimental models of insomnia and alcoholism: cognitive abilities and oxidative stress levels

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Background

Clinical studies demonstrate that insomnia and alcoholism are significantly associated [1]. We have previously shown that experimental insomnia causes oxidative stress in rats [2]. Ethanol is known to induce xanthine oxidase (XO) activity leading to excessive free radicals generation. High free radical levels are associated to cognitive decline [3]. We investigated the effects of the simultaneous application of experimental alcoholism and insomnia on the oxidative status and cognitive functions of male rats.

Materials and methods

Wistar rats were divided into four groups: I-control; II-Alcohol (10% ethanol, ad libitum for six weeks); III-Insomnia (constant light for six weeks); IV- Alcohol+Insomnia.

After sacrifice malondialdehyde (MDA) levels and endogenous XO activity were evaluated in blood plasma. Cognitive functions were assessed in active avoidance "shuttle box".

Results

Shuttle-box: cognitive abilities of rats in all experimental groups were significantly decreased compared to the same day controls; the "alcohol+insomnia" group showed shorter latency time than the "alcohol" group and longer time than the "insomnia" group; "insomnia" rats demonstrated increased number of escapes compared to the same day "alcohol" rats.

Blood plasma MDA levels decreased in the order: (Insomnia+Alcohol) > (Alcohol) > (Insomnia) >(Control). Relative differences in the XO activity were observed.

Conclusions

Relative differences in the XO activities suggested that the oxidative damage is not a result of XO-generated superoxide radicals only. Cognitive decline was correlative to blood plasma XO activity in all stress models. The correlation between cognitive deficits and oxidative stress markers indicated different adaptive abilities of the animals to the investigated stress models.

References

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