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Alcohol-related glucose-dependent functional system of error processing

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Background

Ridderinkhof and colleagues in 2002 [1] reported that moderate "acute" alcohol intake reduces the amplitude of the 'error-related negativity, a negative deflection in the electroencephalogram associated with error commission in speeded response time tasks. Their work is however, subject to a great deal of criticism. To consider arrow flank tasks responses generally, as cognitive functions as a whole is practically non-informative and to say that blood alcohol concentration of 1.0 per mile is a moderate alcohol dose, when legally it is already intoxication is not correct. Ridderinkhof and colleagues were rather careful in analyzing their results. Their study own our respect since it showed that error commission is related to the functions of the monitoring response system in the mediofrontal brain. The mechanisms for Ridderinkhof's and colleagues' work was defined by Holroyd et al [2].

Materials and methods

Ridderinkhof et al/Holroyd et al models, as well as the Peters et al. of blood-brain glucose metabolism were considered. Our data were critically analyzed: Wherefore, we have demonstrated that even episodic moderate intake of alcohol in insignificant doses (23 ml of absolute ethanol) after a significant period of time (average of 8 days) leads to increase in error commission under intensive mental activities, using a more complex and standard tests/tasks (Anfimov geometric tables - for active attention and visual productivity coefficient analyses; ten series of two-digit figures - for visual memory analysis; one-digit figure and letter on increasing row from 3-10 or any vowel letter - for auditory memory analysis and simple mathematical deduction - for operative memory analysis) as a measure of cognitive functions.

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Results

The percentage of error committed was dependent on the blood glucose concentration. To explain the results of our study, we proposed a model - "alcohol related glucose-dependent functional system of error processing", in which not only the Ridderinkhof et al model is incorporated, but also the fishbone model of blood-brain glucose metabolism (Peters et al. Neurosci & Biobehav Rev 28; 2004: 143-180), and in which leptin and insulin - are the main regulators. Neuronal functions depend on the blood-brain glucose proportionality i.e. selective firing mechanisms of nervous impulses is dependent on the blood-brain glucose level. Lowering of the blood glucose level (e.g. inadequate energy reserve) leads to loss of impulses. The response-monitoring system in the basal ganglia is then activated.

Conclusions

The error processing capacity of these processes depends on the mesencephalic dopamine system, anterior cingulate cortex activities, and the blood-brain glucose level. The major concepts of the "alcohol-related glucose dependent functional system of error processing" unravel basic knowledge about the effect of drugs and other psychotic substances on the nervous system functions.

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