In situ rat brain and liver spontaneous chemiluminescence after acute ethanol intake

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The influence of acute ethanol administration on the oxidative stress status of rat brain and liver was assessed by in situ spontaneous organ chemiluminescence (CL). Brain and liver CL was significantly increased after acute ethanol administration to fed rats, a response that is time-dependent and evidenced at doses higher than 1 g/kg. Ethanol-induced CL development is faster in liver compared with brain probably due to the greater ethanol metabolic capacity of the liver, whereas the net enhancement in brain light emission at 3 h after ethanol treatment is higher than that of the liver, which could reflect the greater susceptibility of brain to oxidative stress. The effect of ethanol on brain and liver CL seems to be mediated by acetaldehyde, due to its abolishment by the alcohol dehydrogenase inhibitor 4-methylpyrazole and exacerbation by the aldehyde dehydrogenase inhibitor disulfiram. In brain, these findings were observed in the absence of changes in the activity of superoxide dism