Evidence of brain protecting action of carotenoids extracted from shrimp against the effects of ethanol: a possible role for astaxanthin

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Ethanol ingestion constitutes a frequent drug-abuse situation, which affects a number of organs, including the brain. Ethanol enhances reactive oxygen species (ROS) formation, which is potentially injurious to the brain tissue. The ROS-effects are counteracted by mechanisms involving antioxidant substances, like carotenoids. We have previously shown that ethanol facilitates the propagation of cortical spreading depression (CSD), an excitability-related neural phenomenon. This CSD-effect was attenuated by a shrimp (*Litopenaeus vannamei)* carotenoids extract. Since the main carotenoid found in shrimp is astaxanthin, here we investigated the effects of pure astaxanthin on CSD. Adult Wistar rats recived per gavage, during 18 days, 2.5, 10 or 90 µg/kg/d astaxanthin dissolved in ethanol (3 g/kg). CSD was recorded on the cortical surface 1-3d thereafter. Four groups, treated respectively with ethanol, distilled water and soybean oil with- and without astaxanthin were also studied for comparison. Ethanol-treated rats displayed higher ($P<0.05$) CSD-velocities, compared to the distilled water-group. Astaxanthin addition to ethanol lead to lower CSD-velocities in a dose-dependent manner, following an exponential decay model ($r^2 = 0.9998$; $y = 2.9048 + 1.1988e^{(-x/9.0217)}$, where the x-variable is astaxanthin dose in µg/kg/day). The velocities of the soybean oil groups were not different from the 10 µg/kg/day astaxanthin+ethanol and distilled water groups. Data demonstrated that astaxanthin antagonizes the ethanol effect on CSD in the rat brain, which is consistent with previous data suggesting that astaxanthin, the main shrimp carotenoid, could be responsible for the previously observed CSD-effects. Probably carotenoid antioxidant properties are involved in such effects.

Keywords: Astaxanthin; Cortical spreading depression; Ethanol.