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## The protective effect of astaxanthin on fetal alcohol spectrum disorder in mice

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**Background:** Astaxanthin (AST), known as a carotenoid pigment, is a strong antioxidant which protects membranous phospholipids and other lipids against peroxidation. Evidences showed that astaxanthin had up to several-fold stronger free radical antioxidant activity than vitamin E and carotene. In double-blind, randomized controlled trials, astaxanthin was found to lower oxidative stress in several human health conditions. Moreover, it is known that ROS up-regulate proinflammatory cytokines such as tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin-1 (IL-1), and IL-6. High levels of these cytokines are associated with neurotoxicity. Whereas, astaxanthin was found to reduce makers of inflammation e.g., TNF- $\alpha$ . Thus, astaxanthin has been deemed to be safe and has potential as a therapeutic antioxidant and anti-inflammation agent for further testing in human diseases.

**Objective:** To explore the protective effect of astaxanthin on fetal alcohol spectrum disorder in mice, and to investigate the underlying mechanisms.

**Methods:** The morphology, expression of neural marker genes, oxidative stress indexes, and inflammatory factors in mice model of fetal alcohol spectrum disorder with or without astaxanthin pretreatment were detected.

**Results:** Results showed that astaxanthin blocked maternal ethanol induced retardation of embryonic growth, and the down-regulation of neural marker genes, Otx1 and Sox2. Moreover, astaxanthin also reversed the increase of MDA, H<sub>2</sub>O<sub>2</sub>, and the decrease of GPx in fetal alcohol spectrum disorder. In addition, maternal ethanol induced up-regulation of TLR4, and the down-streaming MyD88, NF- $\kappa$ B, TNF- $\alpha$ , and IL-1 $\beta$  in embryos, and this was inhibited by astaxanthin pretreatment.

**Conclusions:** Results demonstrated a protective effect of astaxanthin on fetal alcohol spectrum disorder, and suggested that oxidative stress and toll-like receptor signaling associated inflammatory reaction were involved in this process.

### Biography

Ying Peng has completed his PhD at the age of 31 years from SunYat-Sen University School of Medicine, Guangzhou, China and Postdoctoral studies from National Cancer Institute-at Frederick, NIH, MD, USA. He is the Director and Professor of Department of Neurology, Sun Yat-Sen Memorial Hospital, SunYat-Sen University. As a clinical neurologist, he has published more than 75 papers in reputed journals and is serving as an Associate- Editor-in-Chief of "Chinese Journal of Nervous and Mental Disease" and Deputy- Chief of Neuro-pharmacological Association of Guangdong province, China.

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