

Impact of Astaxanthin Supplementation on Postprandial Oxidative Stress

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ABSTRACT

Astaxanthin is a naturally occurring carotenoid found in salmon and microalgae that has antioxidative properties. Previous research shows dietary supplementation may result in increased endogenous antioxidant status and reduced markers of oxidative stress (OS). Research suggests OS causes increased risk for cardiometabolic and other chronic diseases, making it important to find interventions that can decrease occurrence of OS. While astaxanthin has been shown to be antioxidative, most studies observing the impact of astaxanthin has on OS has involved rodent models, therefore human trials are needed.

PURPOSE: The purpose of this study was to assess whether four weeks of astaxanthin supplementation (6mg/day) can decrease blood markers of oxidative stress following the consumption of a high fat meal in healthy, young men. **METHODS:** Participants (n=13) were healthy, young men (mean \pm SD; Age: 23.7 \pm 2.7 yrs; Weight: 79.8 \pm 12.6 kg; Height: 171.7 \pm 5.9 cm) that completed a randomized, double blinded crossover counterbalanced study. All participants completed 4 weeks of 6 mg of astaxanthin supplementation in addition to 4 weeks of placebo supplementation with a one-week washout period between treatments.

After each supplementation period, participants consumed a high fat milkshake (milkshake; 1g fat/kg body weight, 1g carbohydrate/kg body weight, 0.25g protein/kg body weight) to induce postprandial OS. Blood samples were taken immediately before, as well as two, and four hours post milkshake consumption. The following were analyzed from each blood sample: triglycerides (TAG), glutathione (GSH), hydrogen peroxide (H₂O₂), malondialdehyde (MDA), and advanced oxidation protein products (AOPP).

RESULTS: Participant compliance on average was 98%. Regarding AOPP, there was no treatment x timepoint interaction or a significant main effect for treatment, while there was a main effect for time (F = 17.14, p < 0.01) with significantly higher levels of AOPP at two and four hours postprandial compared to pre ingestion (p < 0.01). In regard to MDA, there was no treatment x timepoint interaction or a significant main effect for treatment but a main effect for time was noted (F= 24.66, p < 0.01) with significantly higher (p < 0.01) MDA levels 2 and 4 hours post ingestion when compared to pre ingestion levels. In terms of GSH, while there was no treatment x timepoint interaction or main effect for time, the main effect for treatment did approach significance (F = 3.67, p = 0.06). There was no treatment x timepoint interaction for TAG nor a main effect for treatment, while there was a significant increase in TAG levels over time noted by a main effect (F = 15.80, p < 0.01) with 2 and 4 hours post ingestion being significantly higher than pre ingestion TAG levels (p < 0.01). Furthermore, with H₂O₂, a treatment x timepoint interaction (F = 5.94, p < 0.01) was noted. H₂O₂ levels were significantly lower (p < 0.05) 2 and 4 hours post ingestion following astaxanthin supplementation when compared to the placebo treatment.

CONCLUSION: The high fat meal induced significant OS, reflected by increased levels of MDA, AOPP, and H₂O₂. Despite postprandial hyperlipidemia, astaxanthin was able to significantly decrease H₂O₂, suggesting astaxanthin is a desirable exogenous antioxidant for mitigating OS.