Role of carotenoids as antioxidants in the marine copepod, *Tigriopus californicus*

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The food, pharmaceutical, and cosmetic industries are directing increasing attention to compounds with antioxidant properties. This interest arises from antioxidants' capability to quench reactive oxygen species (ROS) such as H2O2 and O2-, which play a key role in aging and pathological conditions1. ROS attack bio-membranes and cause damage to lipids, proteins, and DNA2. Antioxidants prevent oxidative damage by reducing ROS and preventing the radical chain reactions that would otherwise lead to damage of cellular components. Animals internally synthesize many antioxidants, such as superoxide dismutase and glutathione, but can also obtain antioxidants from their diet.

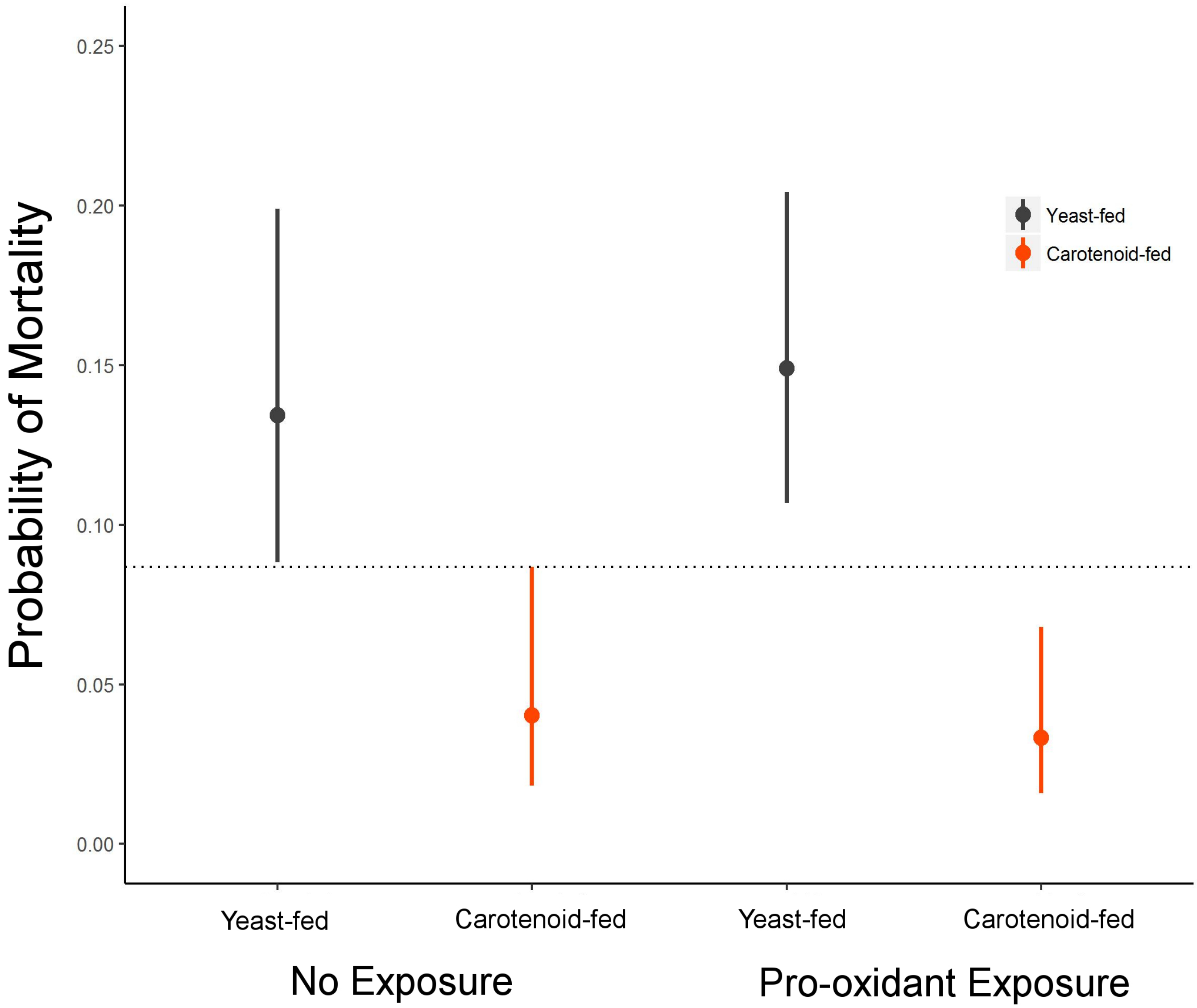

Figure 1: Mortality of carotenoid-deficient and carotenoid-supplemented copepods for both the pro-oxidant exposure and control treatments shown with 95% upper and lower confidence levels.

Carotenoids are organic pigments synthesized by plants that are the source of red, orange, and yellow colors in many plants and animals. Carotenoids have antioxidant properties and can quench ROS by dispersing the radical's energy into a solvent, as shown in many in vitro studies3. Increased dietary intake of carotenoids has been correlated with a variety of physiological benefits, such as increased immune function and increased pathogen resistance across multiple species4. Many scientists propose that the physiological benefits of increased carotenoid consumption arise from their antioxidant properties. However, evidence for carotenoids'in vivo antioxidant activity is contentious.

We used the marine copepod, Tigriopus californicus, to test the antioxidant properties of carotenoids in animal systems. In the wild, T. californicus obtains carotenoids by consuming algae. In the lab, T. californicus can be raised on a yeast diet to completely remove carotenoids from its system. Carotenoids can be reintroduced into the copepods by supplementing their yeast diet with powdered carotenoid. We fed 600 yeast-raised copepods either yeast or yeast with carotenoid supplement for 48 hours. We then placed half of them in a 100μM tert-butyl hydroperoxide (tBHP) solution to stimulate the production of ROS. After 24 hours of exposure, we counted copepods to assess mortality (Fig. 1).

The mortality data are promising and show that carotenoid-fed copepods had a lower mortality than carotenoid-deficient copepods in both the control and pro-oxidant exposure groups. However, there was no difference in mortality between the control groups and the pro-oxidant groups, a result that suggests our concentration of pro-oxidant had no effect on the copepods' mortality. We will repeat the experiment with a higher concentration of tBHP for a shorter amount of time to hopefully induce more oxidative damage in the exposure groups. We will also measure the amount of malondialdehyde using high performance lipid chromatography to quantify the level of lipid peroxidation in each group.

Statement of Research Advisor:

Philip worked as an independent investigator on the study of the role of carotenoids in protection from oxidative damage in copepods. He designed the study, confronted the numerous problems that arose in the execution of the research, and is now taking a leading role in the analysis and interpretation of data

–Geoffrey Hill, Biological Sciences

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