Effect of Astaxanthin on Muscular Atrophy

Reference: Tateo Sugiura, Yoshiharu Iida, Hisashi Naito, Daijiro Ohmori, Katsumasa Goto, Toshitada Yoshioka. 2005 Japanese Journal of Physical Fitness and Sports Medicine. Vol. 54, No. 6, pg 466. December 2005. (Translated from Japanese)

Objective: Patients wearing casts or other devices that hinder mobility are reported to have muscular atrophy. It is commonly thought that the cause is from reactive oxygen species (ROS). The use of Vitamin E, along with other antioxidants, prevents ROS from causing muscular atrophy that arises from lack of movement; however there has been conflicting reports on its effectiveness, varying from some claiming that it works and others that it does not.

In this experiment, Astaxanthin (Ax), which is considered to be a more effective antioxidant than Vitamin E or beta-carotene, will be administered to subjects as food supplement to see its effect on muscular atrophy caused by lack of movement. It will also be tested if the amount of Ax intake will make a difference in its effectiveness.

Methods: 14-week old, Wister-type, male rats were used. Mice were all the same weight after growth for one week under controlled conditions. The rats were separated into three separate groups: Control group (n=7), Ax 0.04% group, and Ax 0.2% group.

15 days after the administration of Ax, each rat had his right leg contained with a cast in an extended position to decrease muscle mass in the triceps surae muscle group for 10 days. At the end of the experiment, the weights of the rats were measured and, along with the use of Nembutal (an anesthesia), euthanized. The plantaris muscle was extracted for analysis.

Results and Analysis: Groups that were administered Ax had significantly less muscle atrophy than those in the Control group ($p<0.05$). The level of Cu/Zn-SOD expressed was higher in the rats with casts than those without casts in the control group; however, in the Ax group, the level expressed was insignificantly different from those with casts and those without. In addition, the level expressed in the control group with casts was significantly higher than the Ax group with casts on. The level of calpain and ubiquitin expressed was higher in the control group with casts than those in the Ax group with casts, but the difference was insignificant. Also, significantly less (of calpain and ubiquitin) was expressed in the Ax 0.2% with casts compared to the control group with casts. The same pattern was seen with Capthesin L expression.

Presently, it is reported that muscular atrophy in patients who are immobile due to casts was caused by oxidative stress. The increase in oxidative stress accelerates the reaction of lipoperoxide, which causes distress in the cell membrane and sarcoendoplaxmic reticulum, leading to an increase in $Ca2+$ in the cytoplasm and concurrently causing a decrease in its discharge. An increase in $Ca2+$ concentration activates calpain along with cathepsin. In addition, the presence of lipoperoxide causes disruption in the cell membrane of the mitrocondria, causing iron ions and ROS to leak in the cytoplasm, which leads to ubiquitination (of proteins.) Ax is the same as beta-carotene in that they are both carotenoids. They both prevent lipoperoxides from disturbing the cell membrane in many biological organisms, but Ax is more active than other antioxidants. Based on this information, we believe Ax intake prevents muscular atrophy by protecting membranes; preventing oxidative stress which results in atrophy; preventing the

facilitation protease and ubiquitination. The effects due to the quantity of Ax uptake were not clear in this study.