

PRESS RELEASE

For Immediate Release

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Astaxanthin Shows Promise to Inhibit NAFLD/Hepatic Steatosis

Burlington, NJ, July 23, 2015 - AstaReal® astaxanthin significantly improved NAFLD in patients with Werner syndrome, known as adult progeria, in a clinical study conducted by the Department of Clinical Cell Biology and Medicine, Graduate School of Medicine, Chiba University (Prof. Kotaro Yokote), which was published in the Journal of American Geriatrics Society (JAGS: June 2015, Vol. 63, No.6, 1271-1273).

■ What is Werner syndrome?

Werner syndrome, also called adult progeria, is a rare genetic disorder, clinically characterized by the juvenile onset of “symptoms generally regarded as an aging indicator.” Symptoms in typical Werner syndrome patients, for example, include premature graying of the hair or hair loss and presence of bilateral cataracts, begin to display in young adulthood, usually in the early 20s. Patients are also at increased risk of age-related diseases such as heart disease, type 2 diabetes, osteoporosis and arteriosclerosis. These diseases-develop one after another resulting in an average life expectancy of around 50 years.

According to genetic research, the cause is supposed to be a mutation of the WRN gene, located on Chromosome 8. When functioning normally, the gene encodes for the WRNp protein. Together, the gene and protein respond to DNA replication malfunctions by unwinding DNA, a crucial step in DNA repair and replication. A mutation in the gene causes unrepaired DNA accumulation due to an insufficiency of helicase, an enzyme contributing to DNA repair, or decreased particular tissue cells due to cell death accelerated by telomere shortening at an abnormally fast pace.

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■ Result from the administration of a dietary supplement “AstaReal®”

A Werner syndrome patient who visited the Chiba University Hospital was suffering from diabetes mellitus. Liver computed tomography (CT) revealed many lipid droplets in liver cells, indicating Nonalcoholic Fatty Liver Disease (NAFLD) with serious hepatic steatosis. After consultation with the patient, attending physicians Dr. Minoru Takemoto and Prof. Yokote suspected the liver might be under oxidative stress due to reactive oxygen species (ROS) and recommended the administration of astaxanthin to quench ROS. The patient was given 2 capsules of AstaReal® (containing 6mg natural astaxanthin extract per capsule) everyday (i.e. astaxanthin at a dose of 12 mg/day). AstaReal® was supplied by the AstaReal Co., Ltd. of the Fuji Chemical group (President: Mitsunori Nishida) as a “dietary supplement”. After 3 months of treatment, CT scan showed a significant decrease of lipid droplets in the liver. The treatment of 12mg/day astaxanthin was continued upon patient request for another 3 months, resulting in additional decrease of lipid droplets noted by liver CT.

Blood test confirmed that the patients’ liver function returned to normal, with improvement in hepatitis markers including aspartate aminotransferase (AST) and alanine aminotransferase (ALT). Such preferable outcomes from the 6-month treatment encouraged the patient to continue to take astaxanthin for nearly 2 years after that without any recurrence of NAFLD nor side effects due to the long-term treatment.

■ Discussion

Werner syndrome patients tend to be insulin resistant due to visceral fat accumulation. In addition, their mitochondrial dysfunction leads to the high incidence of ROS. Since the liver easily receives long-term oxidative stress due to these factors, the patient in this case possibly experienced NAFLD.

Meanwhile, many basic studies have demonstrated that the strong antioxidant effect of astaxanthin stabilizes the plasma membrane, improves mitochondrial function, and inhibits DNA damage and inflammatory response as well. In a study with mouse models of diabetes mellitus, astaxanthin actually mitigated endoplasmic reticulum (ER) stress and inflammation in the liver. Such multiple effects probably brought the therapeutic success in this case.

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Although the potential of astaxanthin treatment for nonalcoholic steatohepatitis (NASH) has already been expected from the result of a study with NASH mouse models conducted by Associate Prof. Tsuguhito Ota, et al, the Kanazawa University Brain/Liver Interface Medicine Research Center (a health column, the Sankei Shimbun, April 8 2014), this is the world's first report of a clinical study involving human subjects. The result mentioned above was obtained from 1 patient with a rare genetic disease, but it is a very valuable case report in that the patient had a clear etiopathogenesis and medical history.

For further data on this clinical study, we will present at the 62nd Kanto/Koshinetsu regional meeting of the Japan Geriatrics Society held on September 26 of this year.

PubMed

<http://www.ncbi.nlm.nih.gov/pubmed/26096415>

<Reference: About NAFLD>

Excessive drinking and hepatitis virus infection have been attributed so far in causing hepatic cirrhosis and hepatitis. In order to prevent or treat these diseases, the limitation of alcohol use and virus eradication with antiviral agents are considered to be effective, and in fact, they have provided expected efficacy. However, NAFLD, a symptom causing hepatitis in the absence of excessive drinking history and virus infection, is often seen in recent days, which is becoming a major issue to overcome.

NAFLD develops even in people with obesity, as well as metabolic syndrome, and may lead to hepatic steatosis. In Japan, there are many people with NAFLD, including 10 million carriers. In 10% to 20% of them (1 million to 2 million people), this condition progresses to NASH, resulting in hepatic cirrhosis and hepatitis. Diet modification to prevent NAFLD and the search for drugs or health foods to prevent/inhibit NASH are in urgent demand.

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