Abs. 466, 205th Meeting, © 2004 The Electrochemical Society, Inc.

HYDRATED C_{60} FULLERENES PREVENT PROCESS OF EXPERIMENTAL ATHEROSCLEROSIS DEVELOPMENT AND FACILITATE THE ATHEROSCLEROTIC LESIONS REVERSION.

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Gistological and histochemical methods were used for investigations of hydrated C_{60} fullerenes (**HyFn**) aqueous solutions [1, 2] on the process of experimental atherosclerosis development in animals.

Experimental Procedures.
During 5 months, four groups of animals (40 males of "Shinshilla" rabbits) were investigated, namely:

(A) intact ones;
(B) rabbits, which received oral cholesterol load (200 mg/kg/day) during 5 months in accordance with the scheme: 5 days of cholesterol diet, 2 days of rest;
(C) rabbits, which received HyFn only (6 i/v injections for 3 months; total dose of C_{60} = 0.6 mg/kg of body weight);
(D) rabbits, which received oral cholesterol load during 5 months, and from the beginning of the fourth month of experiment, have been treated with HyFn (4 i/v injections for 2 months; Total dose of C_{60} = 0.4 mg/kg of body weight).

Results.
1.- On the fourth month of cholesterol diet (*group **B***), in aorta intima, in heart, liver and kidneys tissues of animals the multiple lesions, typical for experimental atherosclerosis, were observed.

2.- Intravenous injections of HyFn to the rabbits (*group **C***), which were on the standard diet, haven't resulted in any morphological and ultrastructural alterations in cardiac vessels and another organs.

3.- Beginning from the fourth month of experimental atherosclerosis development (*group **D***), treatment with HyFn stopped the progression of it and promoted the reversion of atherosclerotic lesions. This became apparently morphologically, because the hydrated fullerenes:

- Relax the endothelial cells, which makes interendothelial contacts more tight.
- Prevent adhesion and transendothelial migration of mononuclear cells to aorta intima.

Thus, the hydrated fullerenes not only prevent the development of pathological alterations, but also they promote the regeneration of disturbed tissues and result in regression of experimental atherosclerosis.

Main mechanisms of prophylactic and therapeutic action of HyFn are discussed from the point of view both "wise" antioxidative properties of HyFn [3, 4] and their ability to protect cell membranes from damaging factors.

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[2] G.V. Andrievsky, et. al., Chem. Phys. Letters, 364 (2002) 8.
[3] A.D. Roslyakov, et. al.,. Zh. Akad. Med. Nauk Ukrainy, 5 (1999) 338 (in Russ).
[4] G.V. Andrievsky, et. al.,. The 6-th Biennial International Workshop in Russia "Fullernes and Atomic Clusters", IWFAC'03, June 30 – July 4, 2003, St. Petersburg, Russia, Book of Abstracts, p.236 (http://www.ioffe.rssi.ru/IWFAC).