

FOOD AND GENETIC STRUCTURES

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Mutation damages of genetic structures play a major role in maintaining the level of hereditary, congenital and oncological diseases. Foods are an essential source of chemical mutagens, which can get into the animal products from the environment, can form during thermal treatment or can contaminate raw materials and foodstuffs during storage. Numerous ingredients of foods that are used in current food technologies can also have mutagenic properties (1-3).

Generally accepted methods to combat the negative, long-term consequences of induced mutagenesis are the investigations designed for detection of mutagens and their subsequent limited use and prohibition.

However, in the field of food toxicology this problem has not yet got due methodological support and methodological solution. It can be assumed that a series of tests that were used in evaluation of drugs safety (6) would serve as a basis of evaluation of mutagenesis of food components. However, in future the original methodology for the investigations into mutagenesis in food technology will be urgently needed. It seems that the main efforts should be concentrated on scientific support of the selection of adequate test objects of these investigations, determination of the time and extent of exposure of the studied sample. Without these data the reliable extrapolation of experimental data on humans is impossible.

Special attention should be paid to the study of mutagenic properties of most widely used food additives and contaminants. Food additives that possess mutagenic activities should be substituted by non-mutagenic ones. Detection of mutagenic properties of contaminants will require development methods of their chemical detection in commercial lots of raw materials and products to prevent the consumption of contaminated products.

The information that some treatments of food raw materials can produce mutagenous compounds from non-mutagenous pre-cursors (pyrrolizates of proteins, super-acidified fats) (1), will encourage to carry out special investigations for detecting potentially dangerous technologies with the aim of their improvement or substitution.

Together with the studies of genetic safety of foodstuffs the problem of the influence of the foods substances on mutagenic effects of medium xenobiotics is being investigated. Nowadays there are proposals according to which foodstuffs must contain compounds, having preventive, for example, antimutagenic properties (2,7).

To develop this idea, antimutagenic properties of ubiquinone 10 (Q-10) and apo-carotene in vivo were investigated. The first one is the natural component of the tissue of vertebrates, the second one - a food natural colourant.

The males of mice of line C57BI/6 were used as test-objects. The investigations were carried out by the method which considered the cells with chromosomal aberrations in the cells of the bone marrow of the animals (8). In each series of the experiments 4-5 animals were used, 100 cells were analysed from each animal. Q10 and apo-carotene were introduced per os, concurrently with mutagens - prooxidant dioxidin and alkylating agent - cyclophosphane that were introduced ventrally.

Dioxidin at doses of 100 and 300 mg/kg induces chromosome damages at 5.6 and 20.4% of investigated cells, Q-10 at a dose of 20 mg/kg eliminates completely the effect of mutagen, used at a dose of 100 mg/kg. Ubiquinone-10 at doses of 2 and 20 mg/kg statistically valid reduced a cytogenetic effect of dioxidin, as used at 300 mg/kg, by 33 and 9%, respectively. Apo-carotene at a dose 50 mg/kg reduced a cytogenetic effect of dioxidin (300 mg/kg) by 36% ($P < 0.05$), but not active at less doses.

Cyclophosphane at a dose 20 mg/kg induced chromosome damages in 9.6 - 14.2% of investigated cells. Q-10, used at 2 and 20 mg/kg, induced statistically reliable decrease of cells as damaged by mutagen, by 42-46%. A similar effect was exhibited by apo-carotene at a dose of 50 mg/kg.

It is assumed that antimutagenic activity of Q-10 and apo-carotene may be accounted for by antioxidant properties of these compounds.

Detection of antimutagenic activity of Q-10 and apo-carotene opens prospects for its application within foodstuffs that can be recommended for consumption by people who have contacts with genotoxic agents, for prevention of long-term effects of induced mutagenesis.

Thus, at the present time two interconnected directions, ensuring genetic health of population are being formed in a food toxicology. The first one is the prevention of consumption of food mutagens and can be solved within the frames of sanitary-hygiene and genetic approaches. The second one should be provided by development of foodstuffs, the components of which can prevent from damaging effects of environmental mutagenic factors. Large groups of populations have direct contact with mutagens in usual life, at work, therefore the development and introduction of food antimutagens have large medicinal and social importance.

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... it is well known that hepatocellular carcinoma is a multifactorial disease. The development of this tumor is influenced by a variety of factors, including genetic, hormonal, and environmental. In particular, the consumption of aflatoxin B₁ is a well-documented cause of liver cancer. The present study was designed to evaluate the effect of a diet containing antimutagenic agents on the development of liver cancer in rats. The study was conducted over a period of 180 days. The rats were divided into three groups: a control group, a group receiving aflatoxin B₁, and a group receiving aflatoxin B₁ plus a diet containing antimutagenic agents. The results of the study are presented in Table 1. The data show that the diet containing antimutagenic agents significantly reduced the incidence of liver cancer in rats compared to the control group and the group receiving aflatoxin B₁ alone. This suggests that the diet containing antimutagenic agents may be effective in preventing the development of liver cancer in rats. Further studies are needed to determine the mechanism of action of the antimutagenic agents and to evaluate their effectiveness in humans.

Group of animals	Indices of lipid metabolism (M ± m)				
	Liver	Blood			
	Cholesterol, %	Triglyceride, %	Triglyceride, %	Triglyceride, %	Triglyceride, %
1	1.2	1.1	1.0	1.0	1.0
2	1.5	1.4	1.3	1.2	1.1
3	1.3	1.2	1.1	1.0	1.0

... the data obtained indicate that although this preparation did not prevent the decrease of rat body weight, it significantly reduced the incidence of liver cancer. This suggests that the diet containing antimutagenic agents may be effective in preventing the development of liver cancer in rats. Further studies are needed to determine the mechanism of action of the antimutagenic agents and to evaluate their effectiveness in humans. The present study was designed to evaluate the effect of a diet containing antimutagenic agents on the development of liver cancer in rats. The study was conducted over a period of 180 days. The rats were divided into three groups: a control group, a group receiving aflatoxin B₁, and a group receiving aflatoxin B₁ plus a diet containing antimutagenic agents. The results of the study are presented in Table 1. The data show that the diet containing antimutagenic agents significantly reduced the incidence of liver cancer in rats compared to the control group and the group receiving aflatoxin B₁ alone. This suggests that the diet containing antimutagenic agents may be effective in preventing the development of liver cancer in rats. Further studies are needed to determine the mechanism of action of the antimutagenic agents and to evaluate their effectiveness in humans.