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THE ROLE OF DIET IN TREATMENT OF LIPID METABOLISM DISORDERS

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Abstract. Apart from arterial hypertension the most frequent risk factor for cardiovascular diseases is connected with lipid metabolism disorders, which according to studies conducted to date in Poland affect over half of the adult population. Diet plays a significant role in the regulation of lipid metabolism in the organism. Thanks to the rationalization of diet we may control lipid metabolism disorders, in many cases with no need to apply more invasive methods, such as pharmacological treatment or the application of cardiosurgery. An appropriate diet as well as a change in lifestyle (giving up smoking, avoiding stress, taking more exercise) make it possible to reduce total cholesterol concentration in blood serum, improve lipid balance and stop the progress or cause partial regression of atherosclerotic changes in coronary vessels, which in many cases may save lives. In the paper the current dietary recommendations in treatment of lipid metabolism disorders are presented.

Key words: lipid metabolism, cholesterol, dietary recommendations, atherosclerosis, fat

INTRODUCTION

Apart from arterial hypertension the most frequent risk factor for cardiovascular diseases is connected with lipid metabolism disorders, which according to studies conducted to date in Poland, i.e. Pol-MONICA, Pol-MONICA Bis and NATPOL III, affect over half adult population [Podolec et al. 2006]. According to Pająk et al. [2005], in over 50% adults the primary lipid metabolism disorder is hypercholesterolemia. In another national survey LIPIDOGRAM 2004 it was shown that hypercholesterolemia affects 69.2% surveyed population and next to arterial hypertension and obesity constitutes the most frequent risk factor for diseases of the circulatory system [Szczygieł et al. 2008]. Identical data were reported by Gnacińska et al. [2004], who additionally stressed that in 1996 the standardized death rate due to cardiovascular diseases in Poland

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was 2 times higher than e.g. it is in Holland, Luxembourg or Sweden [Gnacińska et al. 2004, Pająk et al. 2005, Drożdż et al. 2007]. This problem might even be more serious at present. This is suggested by the results of the Multi-centre National Population Health Survey (WOBASZ 2002-2006), evaluating among other things the frequency of dyslipidemia in men and women aged 20-74 years. These studies showed that approx. 70% adult men and women in Poland suffer from dyslipidemia, while among them approx. 90% have elevated cholesterol concentrations [Drożdż et al. 2007]. Gnacińska et al. [2004] reported that only in 6.8% individuals aged 50 years did not suffer from lipid disorders or arterial hypertension.

Hyperlipidemias are disorders of lipid metabolism characterised by increased serum concentrations of one or several lipoprotein fractions. Elevated concentrations of individual lipoprotein fractions in some cases are not connected with pathological disorders, since after a meal (particularly rich in fats) and following the consumption of alcohol reference values for individual lipoproteins in blood serum are exceeded [Imiela and Grabowski 2007]. The determination of frequency of dyslipidemia is crucial for public health, since this information is required for planning control strategies for these disorders and allocation of adequate resources for this purpose as well as monitoring of effectiveness of undertaken actions [Patel and Thompson 2006].

Lipid disorders manifested in an elevated concentration of LDL-cholesterol or a low concentration of HDL-cholesterol, and/or high concentration of triglycerides are considered independent risk factors for cardiovascular diseases, irrespective of the type of dyslipidemia one suffers from [Kłosiewicz-Latoszek and Cybulska 2006]. Depending on which lipid fraction level is elevated, we may distinguish 3 forms of hyperlipidemia, i.e. hypercholesterolemia – an elevated level of total and LDL-cholesterol, hypertriglyceridemia – an elevated level of triglycerides and VLDL-cholesterol, and mixed hyperlipidemia – elevated concentrations of cholesterol, triglycerides and LDL- and VLDL--cholesterol.

In healthy individuals lipoproteins daily transport approx. 100 g triglycerides, cholesterol and phospholipids. However, there are certain diseases in which excessive production of a specific type of lipoproteins is observed. This state is referred to as hyperlipoproteinemia [Rydén et al. 2007].

There are many studies [Podolec et al. 2006, Imiela and Grabowski 2007, Drożdż et al. 2007], which aimed at the determination of threshold values, the exceeding of which would indicate lipid metabolism disorders. In a study conducted by Drożdż et al. [2007] on a rural population hypercholesterolemia was diagnosed in case of total cholesterol concentration of $\geq$ 190 mg/dL, while serious hypercholesterolemia at $\geq$ 320 mg/DL. Low concentrations of HDL-cholesterol were recorded when the concentration of the HDL-cholesterol fraction in men amounted to < 40 mg/dL, while in women to < 46 mg/dL. In turn, hypertriglyceridemia was diagnosed when triglyceride concentration was > 150 mg/dL. Identical reference values in the diagnostics of dyslipidemia were adopted in the NATPOL PLUS survey, which was conducted in 2002; however, the concentration of LDL-cholesterol was also included here. The concentration of $\geq$ 115 mg/dL was assumed to be abnormal.

In turn, in studies by Gnacińska et al. [2004] and Ciborowska and Rudnicka [2006] desirable levels were defined as total cholesterol concentration < 200 mg/dL, LDL--cholesterol concentration < 130 mg/dL, HDL-cholesterol concentration > 40 mg/dL, while that of triglycerides < 150 mg/dL, respectively.

Treatment of increased blood concentrations of LDL-cholesterol begins with a change in lifestyle, first of all the diet. The administration of medication is not connected with a return to the previous eating habits, as a specific diet has to be followed from then on. Therapy of lipid metabolism disorders should begin always with non-pharmacological treatment, which primary goal in patients with hypertriglyceridemia is to ensure normal body weight, to follow an appropriate diet, to provide exercise and absolutely refrain from drinking alcohol [Dobrowolski and Kosiński 2008].

THE ROLE OF FAT AND FATTY ACIDS IN THE DIET OF PATIENTS WITH LIPID METABOLISM DISORDERS

Reduced consumption of saturated fatty acids (SFA) should be the primary goal of dietotherapy of hyperlipidemia [Imiela and Grabowski 2007]. The reason why it is necessary to reduce the consumption of SFAs is the fact that they inhibit catabolism of the most atherogenic fraction of lipoproteins, i.e. LDL, thus contributing to an increase in its blood concentration [Cybulska and Kłosiewicz-Latoszek 2005].

A similar position in the role of SFAs is presented by the American Heart Association – AHA, which stressed that among substances found in food the strongest action increasing the concentration of LDL-cholesterol is exhibited by saturated fatty acids [Lichtenstein et al. 2006]. Among SFAs the highest increase in the concentration of LDL-cholesterol is caused by miristic acid (C 14:0) and palmitic acid (C 16:0). Ellegard et al. [2007] reported that such properties are also found for lauric acid (C 12:0). Although saturated fatty acids also cause an increase in HDL-cholesterol level, an increase in the LDL concentration is much higher than that of HDL. As a consequence, the LDL-cholesterol to HDL-cholesterol ratio increases, which is an advantageous phenomenon. This ratio is considered the so-called atherogenic index [Cybulska 2007]. Its normal value should range from 2 to 4.

The American Heart Association recommends the consumption of SFAs below 7% total energy consumption [Lichtenstein et al. 2006]. Based on the results of the WOBASZ national program it was found that SFA consumption in Poland considerably exceeds the recommended values; the proportion of SFAs in the daily feed ration in men is on average 13.6%, while in women it amounts to 13.1% [Cybulska 2007].

In Italy and Greece, where animal fats to a considerably degree have been replaced in the human diet by olive oil, the ischaemic heart disease is found very rarely. A relatively low incidence of this disease was also reported in Japan. In this way it was concluded that a good method to prevent the ischaemic heart disease is to reduce the consumption of animal fats, which may be replaced by vegetable oils [Cichocka 2005].

The American Heart Association supports the recommendations of the Institute of Medicine and NCEP (National Cholesterol Education Program) concerning fat consumption, which should amount to 25-35% total daily energy [Lichtenstein et al. 2006].

Other important nutrients contributing to an increased concentration of LDL--cholesterol are trans fatty acids. This is practically an elaidic acid, formed from oleic acid in the course of a process called hydrogenation, applied in order to harden vegetable oils. Thus the main sources of trans isomers are hydrogenated margarines and shortenings [Cybulska and Kłosiewicz-Latoszek 2005]. A change in the configuration during this process is connected with a loss of biological activity of acids. Similarly as satu-

rated fatty acids, the trans forms of acids contribute to an elevation of total cholesterol content in blood plasma, including also the LDL fraction, at the same time reducing the level of HDL cholesterol. However, it needs to be stressed that they exhibit a stronger action in comparison to saturated fatty acids. The American Heart Association recommends that in the daily food ration the consumption of trans forms of fatty acids need not exceed 1% total energy [Lichtenstein et al. 2006].

Another component contributing to an increase in LDL-cholesterol is dietary cholesterol. According to the recommendations of American experts, in order to ensure an appropriate blood cholesterol level it is necessary to consume less than 300 mg daily with food [Lichtenstein et al. 2006]. Cybulska [2007] presents these recommendations in a slightly different manner. According to that author dietary cholesterol content may not exceed 300 mg a day in case of prophylactic diet, whereas in case of dietotherapy it is necessary to reduce the dietary cholesterol dose to 200 mg/day. In turn, Harman et al. [2008] showed that high consumption of dietary cholesterol does not cause an increase in serum LDL-cholesterol level when a high cholesterol consumption is accompanied by a moderate body weight loss. Such conclusions were drawn based on a slight reduction of LDL-cholesterol concentration in case of a group of experiment participants consuming 2 eggs a day as a rich source of cholesterol (containing approx. 600 mg cholesterol) for 12 weeks of the study and a group which did not consume any eggs; at the same time we need to stress here that in both groups as a result of the applied low energy diet a simultaneous loss of body weight was also found.

The human organism is capable of forming all fatty acids essential for metabolism, apart from two, i.e. linolic acid (LA) from the omega-6 group, and alfa-linolenic acid (ALA) from the omega-3 group, being a substrate for the formation of other fatty acids (e.g. arachidonic acid (AA) formed from LA). However, their conversion to such acids of the omega-3 lines as eicosapentaenoic (EPA) and docosahexaenic acids (DHA), is limited. Thus it is recommended to supply these acids directly with food and they are referred to as polyunsaturated fatty acids (PUFA) [Holman 1998].

It has been shown in many studies that EPA and DHA exhibit hypolipidemic action reducing the concentration of triglycerides in blood plasma by inhibiting their resynthesis in the intestinal wall and in the liver, as well as increasing catabolism in the process of beta oxidation. Depending on the dose of omega-3 acids the level of triglycerides may be reduced by 30% (assuming approx. 2 g fish oil daily), while in individuals with hypertriglyceridemia even up to 80% [Simopoulos 2002, Strauss et al. 2005].

Kris-Etherton et al. [2002] showed that men who consumed fish at least once a week died due to cardiovascular diseases less frequently than men who did not eat fish at all. In another study with the participation of men, which investigated the effect of omega-3 on the cardiovascular system, it was shown that in men consuming at least 35 gram fish, relative death risk rates due to coronary disease were much lower in comparison to men, who did not eat fish at all [Block and Pearson 2006]. Similar observations were made for women. Recent investigations showed an opposite correlation between the amount of consumed fish and omega-3 fatty acids, and death caused by diseases of the cardiovascular system [Kris-Etherton et al. 2002].

Block and Pearson [2006] reported that the concentration of triglycerides on empty stomach and after a meal may be reduced by 20-35%. This opinion is confirmed by studies conducted by Riediger et al. [2008], who carried out investigations on mice fed a diet enriched with oils of different origin with varying n-6:n-3 oil ratios (linseed oil

and fish oil). In each of three groups (also the control), researchers observed a decrease in the serum level of triglycerides by over 50%, which occurred thanks to the presence of omega-3 acids, and as they assume – thanks to the presence of monounsaturated acids, in case of the control. However, not all studies proved benefits resulting from the consumption of acids from the omega-3 family. An example of such a study was a cohort Health Professionals Follow-up Study. Based on data collected in the course of this study the authors did not find a relationship between saltwater fish consumption or the consumption of omega-3 acids, and the risk of cardiovascular diseases [Ascherio et al. 1995]. Similar conclusions were drawn by the authors of another American study, i.e. the Physicians' Health Study [Albert et al. 1998]. A lack of unambiguous results of clinical trials with PUFA n-3 suggests the need of further studies [Cybulska 2007].

It is believed that an advantageous effect of omega-3 acids on the circulation system results first of all from their anti-atherogenic and anti-inflammatory action, while the scope of this action depends on their endogenous changes to eicosanoids and prostaglandins [DeFilippis and Sperling 2006]. In turn, Block and Pearson [2006] stated that the anti-inflammatory effect of acids from the n-3 family on the cardiovascular system is controversial. This is evidenced by the data from the Diet and N-3 Intervention Trial (DOIT), which showed that in 70-year old men, who suffered from hyperlipidemia the application of a 3-year diet and/or its supplementation with omega-3 acids did not significantly improve inflammatory markers (soluble vascular adhesion factor and the von Willebrand factor). Despite that fact, the World Health Organization (WHO) recommends the consumption of α-linolenic acid at 0.8-1.1 g/day, while eicosapentaenoic acid EPA + docosahexaenoic acid DHA at 0.3-0.5 g/day. In case of α-linolenic acid these values correspond to 0.6-1.2% total energy, when daily diet supplies 2000 kcal [Lichtenstein et al. 2006].

Apart from omega-3 acids, including α-linolenic acid (C 18:2), eicosapentaenoic acid (C 20:5) and docosahexaenoic acid (C 22:6), the beneficial action, consisting in the capacity to reduce LDL-cholesterol, is also found for a monounsaturated fatty acid, i.e. oleic acid (C 18:1). Studies with the participation of patients after ileostomy showed that a diet rich in mono- and polyunsaturated acids increases the secretion of sterols (cholesterol and/or bile acids) from the intestine, such as a diet containing small amounts of saturated acids [Ellegard et al. 2007].

It is recommended to maintain appropriate proportions of n-6 and n-3 acids, which ratio should be 2-4:1 [Riediger et al. 2008]. A disturbed ratio of n-6 to n-3 acids (too high), caused by the consumption of excessive amounts of arachidonic acid (acid from the n-6 family) results in the formation of thromboxane TXA_2, which exhibits very strong proaggregation action and leukotriene LTB_4 with strong proinflammatory properties. Thus it should be attempted to have the PUFA n-6:n-3 ratio relatively low [Cybulska 2007]. It needs to be stressed that in Greece it is less than 2:1, while in countries of northern Europe more than 10:1. Some experts suggest that this proportion is not so crucial – we should rather be interested in the absolute level of consumption. A report from a working meeting of experts on that issue stated that an increased supply of ALA, EPA and DHA in the diet would make it possible to reach the desirable increase in the levels of these fatty acids in tissues, whereas a reduction of LA and AA consumption is not required [Stanley et al. 2007].

Monounsaturated fatty acids (MUFA) and polyunsaturated n-6 fatty acids, by replacing carbohydrates in the diet (in the isocalorie amount), contribute to a reduction of

the amount of LDL-cholesterol, whereas they increase the amount of the HDL fraction. Cybulska [2007] reported that in the therapeutic diet PUFA n-6 should supply from 4 to 10% energy.

THE ROLE OF CARBOHYDRATES IN DIET OF PATIENTS WITH LIPID METABOLISM DISORDERS

As a result of a limitation of fat intake, most of the daily energy requirement should be covered by carbohydrates, first of all complex, minimally processed, contained e.g. in seeds [Imiela and Grabowski 2007]. It was shown that dietary fiber, particularly its soluble fraction, i.e. pectins, β-glucans, mucilages, guar dietary fiber, reduces the blood serum level of LDL-cholesterol, does not contribute to a decrease in the HDL-cholesterol level and does not affect the level of triglycerides [Cybulska and Kłosiewicz-Latoszek 2005, Imiela and Grabowski 2007].

The mechanism of hypocholesterolemic action of dietary fiber has not been completely clarified. Some researchers are of the opinion that soluble fiber binds bile acids and cholesterol inside the intestinal lumen. Another mechanism refers to the inhibition of hepatic synthesis of fatty acids by fermentation products of intestinal microflora, including short-chain fatty acids: acetic and propionic [Brown et al. 1999]. The most popularly accepted theory claims that soluble dietary fiber enhances the rate at which bile acids, formed in the body from cholesterol through the process of synthesis, are excreted with feces. Binding and excretion of bile acids caused by the presence of dietary fiber results in an increasing amount of cholesterol accumulated in the organism being directed to their synthesis, thus reducing its concentration in the serum [Florkowska and Krygier 2004]. It seems that an indisputable fact is the role played by dietary fiber in changes of viscosity of chyme, forming viscous β-glucan gels, which increase may result in a reduction of absorption of cholesterol and binding of bile acids. Increased viscosity of chyme caused by the presence of β-glucans results in a limited return of bile acids to the liver or even their total retention. The generated deficit of bile acids results in the liver, enhancing the transformation of cholesterol towards their synthesis. β-glucans are found in all kernels of cereals, being basic components of cell walls. They are also contained in bamboo, mushrooms – mainly oyster mushrooms, as well as yeasts and certain grasses [Finkelman et al. 2006, Ishur et al. 2002, Odabasi et al. 2006, Rout et al. 2005].

Based on meta-analysis of many studies [Podolec et al. 2006] it was found that the consumption of dietary fiber is inversely correlated with the risk of cardiovascular diseases both in women and men. Based on statistical analysis it was concluded that with an increased consumption of dietary fiber by 10 g/day, a considerable improvement is observed in the lipid profile, as a result of which the risk of death connected with ischaemic heart disease is reduced [Pereira et al. 2004]. In case of dietary fiber we do not speak of physiological requirement, but rather recommended consumption, which should amount to 27-40 g/day (16-24 g/day non-starch polysaccharides – apart from lignins).

In patients with endogenous hypertriglyceridemia carbohydrates enhance the production of triglycerides in hepatic cells, which as a consequence contributes to an increase in the level of this component in blood serum. This suggests that these com-

pounds also cause a reduced level of HDL-cholesterol. Thus consumption of this macronutrient should be limited. Cybulska [2007] suggested that it should amount to 50% total energy.

THE ROLE OF VITAMINS AND FLAVONOIDS IN THE DIET OF PATIENTS WITH LIPID METABOLISM DISORDERS

The theory of oxidative changes in atherogenesis facilitated the advances in studies on antioxidant properties of vitamins in the prevention of initiation and development of atherosclerotic lesions. Epidemiological studies suggested that supplementation of diet with different nutrients exhibiting antioxidant properties makes it possible to inhibit the formation of atheromas [Witana et al. 2006].

Observations in the USA and Finland confirmed the advantageous effect of a diet rich in vitamin C, E, β-carotene and selenium. In a study by Bellizzi et al. [1994], taking into consideration data from 24 countries, the mean contents of vitamins A, C and E in the diet were negatively correlated with mortality caused by ischaemic heart disease, while in a study by Artaud-Wild et al. [1993] covering 40 countries, lower death rates were recorded only in regions with higher mean contents of vitamin E in the diet, although it did not refer to vitamins A or C.

Vitamin E is closely connected with LDL lipoproteins transporting it to target cells, thus its role in the protection of these molecules against oxidation and in the prevention of atherosclerosis is so important [Duda et al. 2002]. It does not reduce blood cholesterol concentration, but changes the structure of lipoproteins, preventing their oxidation and thus limiting atherogenic character [Imiela and Grabowski 2007]. Antioxidant properties of vitamin E depend both on the type of their homologous form and its concentration as well as the type of fatty acids dominating in the molecule [Duda et al. 2002]. Some researchers warn against long-term intake of high doses of vitamin E, since this may lead to adverse effects. In vitro studies show that high doses of this vitamin may have a pro-oxidant action on LDL, consisting in the formation of α-tocopheroxy radical, which inhibits the antioxidant action of vitamin C, which in turn synergistically cooperates with vitamin E. As a strong reducer it regenerates vitamin E from the oxidized radical form. This statement refers only to high doses of this vitamin, as well as other antioxidant vitamins (C and β-carotene), thus the protective role of antioxidants supplied at optimal amounts should not be undermined [Cybulska 2007].

A lack of success in the inhibition of the atherosclerotic process after administration of high doses of vitamin E to humans was also reported by Ganong [2007]. Failure in studies on the application of high doses of these vitamins in humans may result from the fact that these were synthetic compounds. The amount of antioxidant substances available with supplements may be much higher than that consumed with food and this high concentration may exhibit a toxic action. Randomized clinical trials (with a double blind trial) did not supply certain evidence supporting the protective action of antioxidant vitamins, and in case of some of these compounds supplementation proved to be even harmful [Ciborowska and Rudnicka 2006]. Antioxidant vitamins are nutritive substances found mainly in vegetables and fruit, which apart from these components contain also high amounts of dietary fiber, flavonoids, while low amounts of saturated fatty acids and cholesterol. It is such a combination of components that plays a signifi-

cant role in the prevention and treatment lipid disorders. Thus at the present state of knowledge such a diet should be recommended so that nutrients coming from food fully cover the requirement of this organism for antioxidants. The vitamin E requirement is 9-10 mg, that of vitamin C is 70 mg, while vitamin A is 800-1000 μg, respectively [Ciborowska and Rudnicka 2006].

Some authors attribute a significant role in the treatment of lipid disorders to vitamins from the B family [Gąsiorowska et al. 2008]. Vitamins B6 and B12 as well as folic acid participate in the process of homocysteine metabolism, removing it from the organism through transformation to methionine and cysteine [Sierakowska-Fijałek et al. 2008]. Studies conducted on hyperhomocysteinemia showed that the mechanism of atherogenic action in case of homocysteine is complex and not fully clarified. Many authors attribute the capacity to trigger oxidative stress to homocysteine. Homocysteine damages arterial endothelium, facilitating penetration of LDL, increases the aggregation capacity of platelets and causes modification of LDL.

In numerous randomized trails, which results have been published in recent years, no significant reduction of risk was found for the development of cardiovascular diseases, mainly cardiac infarct, due to the administration of folic acid and vitamins B (although such treatment reduced homocysteine concentration) [Toole et al. 2004, Bonaa et al. 2006, Lonn et al. 2006, Zoungas et al. 2006]. Thus, according to Lichtenstein et al. [2006] intake of these vitamins is not recommended in order to prevent cardiovascular diseases. However, some authors suggested the intake of folates at 400 μg daily, while vitamin B12 at 3 μg and vitamin B6 at 2 mg to prevent hyperhomocysteinemia [Kozłowska-Wojciechowska 2005, Gąsiorowska et al. 2008].

Recently an essential role in the prevention of many diseases has been attributed to flavonoids (polyphenols). The effect of the advantageous action of flavonoids consists in the removal from blood of already formed reactive oxygen species and prevention of their formation. This is possible thanks to the inhibition of enzymes connected with generation of free radicals: lipooxygenase, cyclooxygenase and xanthin oxidase by flavonoids. By reducing the quantity of free oxygen radicals, flavonoids protect against oxidation different fractions of cholesterol, particularly LDL, they activate the synthesis of prostacyclins and exhibit anti-aggregation action. Also the chelating action of many flavonoids prevents oxidation of plasma lipoproteins. The antioxidant action of flavonoids in the organism consists also in the inhibition of oxidation of endogenous antioxidants (vitamin E, ascorbic acid, glutathione and other compounds) in oxidation processes. Flavonoids, such as quercetin, rutin, hyperoside and vitexin play the role of antioxidants in relation to vitamin C. It was shown that they delay the transformation of ascorbate to dehydroascorbinin and protect against the action of free radicals. In turn, ascorbic acid inhibits oxidative changes of flavonoids and extends their protective action [Miller et al. 2008].

Flavonoids are classified as phytochemical substances, as they are found mainly in plants. Rich sources of flavonoids include vegetables, fruit, seeds of different plants, certain cereals, as well as wine, particularly red wine, tea, coffee, cocoa, fruit juices and many spices.

PLANT STEROLS AND STANOLS IN DIET OF PATIENTS WITH LIPID METABOLISM DISORDERS

Recently an increasing interest in plant sterols and stanols (phytosterols and phytostanols) has been observed as substances with properties reducing LDL-cholesterol [Cybulska and Kłosiewicz-Latoszek 2005]. Sterols are permanent components of cell membranes both in the animal and plant kingdom. Sterols found in plants are also called phytosterols. Phytosterols include sterols – unsaturated compounds, and stanols – saturated compounds, not having a double bond in their structure. The biggest amounts of naturally occurring sterols are contained in vegetable oils, nuts, legumes, sesame, sunflower and other seeds.

Grundy [2005] claimed that it is plant sterols contained in daily diet that have the capacity to most significantly reduce the level of LDL-cholesterol, by competition of intestinal absorption with cholesterol; however, they do not have an effect on cholesterol in the HDL fraction and triglycerides. The most effective compounds in the long-term reduction of cholesterol level are esters of sitostanol, which in a 12-month observation period in 153 individuals with the so-called environmental hypercholesterolemia caused a decrease in LDL-cholesterol level on average by 14.1% [Ellegard et al. 2007]. A typical Western diet contains 200-500 mg cholesterol, and 200-400 mg stanols and sterols. However, it needs to be remembered that absorption of plant sterols and stanols is approx. 10 times lower than that of dietary cholesterol [Patel and Thompson 2006].

Ellegard et al. [2007] showed that a higher secretion of cholesterol in the intestine is correlated with higher contents of plant sterols in the diet. One of the first studies concerning hypolipemic properties of sterols proved that sterols at a dose of 18 g/day caused a reduction of serum cholesterol level to 20%. However, in other studies it was stated that a lower dose of sterols (approx. 3 g/day) caused a reduction of cholesterol level to an identical degree as high sterol doses [Grundy 2004]. These conclusions were confirmed by Patel and Thompson [2006], who observed that a dose of sterols and stanols at 0.8-4 g/day reduced the concentration of LDL-cholesterol by 10-15%. It should be added here that the effectiveness of the action of stanols is maintained throughout the period they are administered. O'Neill et al. [2004] stated a similar effectiveness in the reduction of concentration of LDL fraction cholesterol already after a month of the administration of stanols and sterols; however, after two months the intake of stanols made it possible to maintain the effect of reduced cholesterol concentration, while in individuals taking sterols this effect was weakened. In a review study O'Neill et al. [2005] indicated that the administration of stanols for 6-13 weeks is as effective as in the first 3-4 weeks of their application. They also suggested that stanols need to be supplied in the long-term therapy of hypercholesterolemia.

The report of the Adult Treatment Panel III of 2001 recommended the therapeutic consumption of these compounds in the amount of 2-3 g/day [Cybulska and Kłosiewicz-Latoszek 2005, Grundy 2005]. Katan et al. [2003] claimed that the optimal amount of stanol esters should be approx. 2 g/day. This is consistent with the position of the American Heart Association, published in the recommendations of secondary prevention in patients with cardiovascular diseases [Superko 2000]. Ellegard et al. [2007] suggested that plant sterols may be more important in lowering the concentration of LDL-cholesterol than soluble dietary fiber.

THE ROLE OF ALCOHOL IN DIET OF PATIENTS OF LIPID METABOLISM DISORDERS

It results from observation studies that the relationship between the consumption of alcohol and total deaths takes the shape of letter J [Castelnuovo et al. 2006]. Moderate consumption of alcohol is connected with a lower total mortality, while higher consumption – with high mortality. The mechanism of this action of ethanol is connected among other things with metabolism of lipids and lipoproteids. Consumption of alcohol in moderate amounts increases the concentration of anti-atherogenic apo- and lipoproteins, such as apolipoprotein AI (apo AI) and high density lipoproteins (HDL), while it reduces the concentration of apolipoprotein B (apo B), low density lipoprotein (LDL) and lipoprotein (a) – Lp(a), which exhibit atherogenic action [Vasisht et al. 1992, Lecomte et al. 1996]. According to Rimm et al. [1999], a daily dose of 30 g ethanol causes an increase in HDL concentration on average by 3.99 mg/dl, while the concentration of apolipoprotein AI by 8.82 mg/dl. Ethyl alcohol also causes an increased activity of triglyceride lipases and reduced removal of HDL from the bloodstream [Krawiec et al. 2008]. Increased production of extrahepatic lipoprotein lipases is a response to an elevation of triglyceride concentration. Lipolysis of molecules rich in triglycerides increases the flow of cholesterol to HDL molecules from circulating remnants of very low density lipoproteids (VLDL) and increases total HDL concentration [Mukamal et al. 2003]. In the period of abstinence changes in the concentrations of apo- and lipoproteins occur in the reverse direction [Gueguen et al. 2002, Lamisse et al. 1994]. Quantitative and qualitative changes of lipids and lipoproteins are consequences of disturbed synthesis and degradation of lipoproteins under the influence of alcohol, which in turn is connected with a defect of glycosilation of proteins structurally related with lipoproteins [Lakshman et al. 1999]. It refers to the inhibition of final sialization (the attachement of sialic acid) of apolipoproteins E and J (proteins of HDL cholesterol), apolipoprotein (a) and cholesterol ester transfer protein (CETP) [Marmillot et al. 1999, Gosh et al. 2001, Liinamaa et al. 2006]. It was shown that apolipoprotein E (devoid of sialic acid) desialized as a result of excessive consumption of alcohol, has lower affinity to HDL molecules, which reduces its capacity to transfer cholesterol esters from peripheral tissues to the liver [Lamisse et al. 1994]. As a result the reverse transport of cholesterol from peripheral cells to the liver is inhibited.

Moderate consumption most frequently refers to consumption of alcohol at max. 30 g pure ethanol/day for men (which corresponds to 80 ml vodka or 300 ml wine or 700 ml beer) and max. 15 g/day for women. Its consumption is absolutely prohibited for patients with hypertriglyceridemia. In the chylomicronemia syndrome a meal high in fats, combined with alcohol, may cause acute pancreatitis. It is a typical complication for this lipid metabolism disorder. It also needs to be remembered that consequences of excessive consumption of alcohol include increased risk of certain cancers, diseases of the liver, pancreas, disorders of the central nervous system, pathological lesions in the nervous system (polyneuropathy, brain atrophies), deficiencies of many nutrients (vitamin A, folic acid, thiamine, pyridoxine, zinc), as well as the risk of addiction and resulting health and social problems.

CONCLUSIONS

Diet plays a significant role in the regulation of lipid metabolism in the organism. Thanks to the rationalization of diet we may control lipid metabolism disorders, in many cases with no need to apply more invasive methods, such as pharmacological treatment or the application of cardiosurgery.

The application of a diet to reduce blood serum concentration of cholesterol is by some researchers defined as relatively ineffective, due to the reduction of serum cholesterol level by 4% to 13%, while the application of statins makes it possible to reduce the concentration of LDL-cholesterol on average by 28%, or even by 35%. Jenkins et al. [2003] conducted studies which made it possible to compare hypocholesterolemic diets directly with identical properties of statins. The application of a low-fat diet poor in saturated fatty acids, based on wholemeal products, and a low fat diet supplemented with lovastatin and the same diet enriched with phytosterols, soybean protein, soluble dietary fiber and almonds, made it possible to reduce LDL cholesterol by 30.9% and 28.6%, respectively. These studies showed that there were no significant differences in the effectiveness between pharmacological treatment using statins and dietotherapy.

Recommendations concerning consumption of fat and saturated fatty acids may be followed by choosing lean meat or vegetables. Moreover, fat-free or and low-fat dairy products containing less than 1% fat should be chosen (Table 1). It is known that not only fat from food modifies the blood serum cholesterol content. A certain role in also played by the consumption of cholesterol with food. The main sources of dietary cholesterol include egg yolks, followed by offal processed meat products (liver, pate, headcheese). Thus in order to control the consumption of cholesterol it is first of all required to reduce the consumption of eggs (not more than 2-3 eggs/week).

Table 1. Diet and lifestyle recommendations for lipid disorders

• Limit your intake of saturated fat to < 7% of energy, trans fat to < 1% of energy, PUFA 6-10% of energy, omega 6 – 5-8% of energy, omega 3 – 1-2% of energy and cholesterol to < 300 mg per day by
– choosing lean meats and vegetable alternatives
– selecting fat-free (skim), 1%-fat, and low-fat dairy products
– minimizing intake of partially hydrogenated fats
• Consume a diet rich in vegetables and fruits ≥ 400 g/day
• Consume plant sterols and stanols 2-3 g
• Choose whole-grain, high-fiber foods. Consume soluble fiber such as psyllium 10-25 g
• Minimize your intake of beverages and foods with added sugars
• Choose and prepare foods with little or no salt
• Balance calorie intake and physical activity to achieve or maintain a healthy body weight

The main sources of α-linolenic acid (ALA) are vegetable oils, primarily canola oil and soybean oil. Other rich sources of this acid are also linseed oil and walnut oil from walnuts grown in California [Kris-Etherton et al. 2002]. The biggest natural sources of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are first of all saltwater

fish [Riediger et al. 2008]. Fish which contain relatively high amounts of EPA, DHA and ALA include mackerel, salmon (with the highest amounts of these acids being found in fish from fish farms) as well as tuna [DeFilippis and Sperling 2006]. In order to provide an adequate supply of omega-3 acids experts of the American Heart Association recommend the consumption of two servings of oily fish a week [Cybulska 2007]. Moreover, we may not forget of the fact that freshwater fish, particularly predatory fish species living in the wild, may also be rich sources of essential unsaturated fatty acids, particularly those from the family of n-3 polyenic fatty acids PUFA. Bienkiewicz et al. [2008] reported that among freshwater fish the dominant proportion of polyunsaturated fatty acids PUFA in the total fat content is found in fish living in the wild: perch 48.51%, pike 40.20% and pike perch 38.50%, respectively. The highest percentage content of monounsaturated fatty acids is found in carp 68.62%, bream 57.86% and grass carp 57.66%. In contrast, a relatively undesirable composition of fatty acids is found in panga and Nile perch. In the total fat content in these fish species a high percentage is found for saturated fatty acids, i.e. 42.99% in panga and 42.47% in Nile perch. At the same time we need to stress the fact that predatory freshwater fish, in relation to other freshwater fish species and saltwater fish, contain lower amounts of fat and thus also a lower calorie content. Pike has five times less DHA in relation to eel, but the amount of fat is more than 10 times lower.

In view of the significant role of dietary fiber in the regulation of lipid metabolism disorders, it is recommended to eat more vegetables, fruit (preferably raw) and legumes (beans, soybeans), due to their content of soluble fraction of dietary fiber, which reduces the concentration of cholesterol [Kłosiewicz-Latoszek and Cybulska 2006]. Consumption of dietary fiber may also by increased by diversification of the diet sing cereal grains (barley, oats) and cereal products (e.g. oat bran) with a bigger use of wholemeal products [Cichoń and Wądołowska 2006]. These products are also good sources of sterols; however, the content of these compounds is highly varied. For example, broccoli are an excellent source of sterols, while green peas contains much lower amounts of phytosterols [Ellegard et al. 2007]. According to Patel and Thompson [2006], the essential source of plant sterols are plants rich in oil and their processed products, including oils, as well as fruits and nuts. Sterols and stanols are usually added to margarines or yoghurts (functional food), which facilitates their use in dietotheraphy. Moreover, they are available in the form of capsules [Cybulska 2007]. In a Danish study it was proved that in patients using a diet which contained considerable amounts of olive oil the level of LDL cholesterol was slightly lower than in patients which diet contained large amounts of rapeseed or sunflower oil. This may result from the fact that olive oil contains much lower levels of plant sterols than rapeseed oil [Ellegard et al. 2007].

Table 1 lists general recommendations (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults 2001, Diet and Lifestyle Recommendations Revision 2006), which should be followed by patients with diagnosed lipid metabolism disorders. An appropriate diet as well as a change in lifestyle (giving up smoking, avoiding stress, taking more exercise) make it possible to reduce total cholesterol concentration in blood serum, improve lipid balance and stop the progress or cause partial regression of atherosclerotic changes in coronary vessels, which in many cases may save lives.

REFERENCES

Albert C.M., Hennekens C.H., O'Donnell C.J., Ajani U.A., Carey V.J., Willett W.C., Ruskin J.N., Manson J.E., 1998. Fish consumption and risk of sudden cardiac death. JAMA 279, 23-28.

Artaud-Wild S.M., Connor S.L., Sexton G., Connor W.E., 1993. Differences in coronary mortality can be explained by differences in cholesterol and saturated fat intakes in 40 countries but not in France and Finland. A paradox. Circulation 88, 2771-2779.

Ascherio A., Rimm E.B., Stampfer M.J., Giovannucci E.L., Willett W.C., 1995. Dietary intake of marine n-3 fatty acids, fish intake, and the risk of coronary disease among men. New Eng. J. Med. 332, 977-982.

Bellizzi M.C., Franklin M.F., Duthie G.G., James W.P., 1994. Vitamin E and coronary heart disease: the European paradox. Eur. J. Clin. Nutr. 48, 822-831.

Bienkiewicz G., Domiszewski Z., Kuszyński T., 2008. Ryby słodkowodne jako źródło niezbędnych nienasyconych kwasów tłuszczowych NNKT [Fishes as the source of PUFA polyunsaturated fatty acids]. Mag. Przem. Ryb. 3, 63, 58-59 [in Polish].

Block R.C., Pearson T.A., 2006. Wpływ kwasów tłuszczowych omega-3 na układ sercowo-naczyniowy [Influence of fatty acids omega-3 on the cardiovascular system Folia]. Cardiol. Exc. 1, 362-376 [in Polish].

Bonaa K.H., Njalstad I., Ueland P.M., Schirmer H., Tverdal A., Steigen T., Wang H., Nordrehaug J.E., Arnesen E., Rasmussen K., 2006. Homocysteine lowering and cardiovascular events after myocardial infarction. New Eng. J. Med. 345, 1578-1588.

Brown L., Rosner B., Willett W.W., Sacks F.M., 1999. Cholesterol-lowering effects of dietary fiber: a meta-analysis. Am. J. Clin. Nutr. 69, 30-42.

Castelnuovo A., Costanzo S., Bognardi V., Donati M.B., Iacoviello L., de Gaetano G., 2006. Alcohol dosing and total mortality in men and women. An updated meta-analysis of 34 prospective studies. Arch. Int. Med. 166, 2437-2445.

Ciborowska H., Rudnicka A., 2006. Dietetyka – żywienie zdrowego i chorego człowieka [Dietetics – feeding the healthy and ill man]. PZWL Warszawa [in Polish].

Cichocka A., 2005. Dieta śródziemnomorska w profilaktyce pierwotnej choroby niedokrwiennej serca [Mediterranean diet in coronary heart disease primary prevention]. Endokryn. Otył. Zab. Przem. Mat. 1, 30-39 [in Polish].

Cichoń R., Wądołowska L., 2006. Węglowodany. Żywienie człowieka. Podstawy nauki o żywieniu [Carbohydrates. Feeding the man. Bases of the theory about feeding]. Eds J. Gawęcki, L. Hryniewiecki. PZWL Warszawa [in Polish].

Cybulska B., 2007. Zdrowe żywienie dla zdrowego serca [Healthy diet for a healthy heart]. Przew. Lek. 2, 49-54 [in Polish].

Cybulska B., Kłosiewicz-Latoszek L., 2005. Diagnostyka i leczenie hipercholesterolemii [Diagnostics and treatment the hypercholesterolemia]. Żyw. Człow. Metab. 32, 74-89 [in Polish].

DeFilippis A.P., Sperling L.S., 2006. Understanding omega-3's. Am. Heart J. 151, 564-570.

Dobrowolski P., Kosiński P., 2008. Leczenie hipolipemizujące i hipoglikemizujące u pacjentów z nadciśnieniem tętniczym [Lipid-lowering drugs and glycaemic control in patients with hypertension]. Kardiologia na co Dzień 3, 20-24 [in Polish].

Drożdż K., Gaweł W., Gać P., Łukasik M., Seniuta J., Kolman K., Cedzyński Ł., Roma R., Doroszko A., Chachaj A., Poręba R., Derkacz A., Andrzejak R., Szuba A., 2007. Zaburzenia lipidowe u osób zdrowych i osób z chorobami układu sercowo-naczyniowego w populacji wiejskiej [Dyslipidemias in healthy individuals and patients with cardiovascular diseases in rural population]. Art. Hypert. 11, 515-521 [in Polish].

Duda G., Maruszewska M., Jóźwiak A., Chmielewski Z., Korybalska K., Wieczorowska-Tobis K., 2002. Analiza wpływu płci na wybrane składowe układu antyoksydacyjnego i gospodarki lipidowej osób w wieku podeszłym z chorobą niedokrwienną mięśnia sercowego [Analysis of the inluence of sex on selected components of the antioxidant system and lipid profile of elderly people with myocardial ischaemic disease]. Nowiny Lekar. 1, 25-19 [in Polish].

Ellegard L.H., Andersson S.W., Normén A.L., Andersson H.A., 2007. Dietary plant sterols and cholesterol metabolism. Nutr. Rev. 65, 39-43.

Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) 2001. JAMA 285, 2486-2497.

Finkelman M.A., Lempitski S.J., Slater J.E., 2006. Beta glucans in standardized allergen extracts. J. End. Res. 4 (12), 241-245.

Florkowska A., Krygier K., 2004. Zastosowanie nietrawionych oligosacharydów w produktach spożywczych [Use not-digested oligosaccharides in food products]. Przem. Spoż. 5, 44-46 [in Polish].

Ganong W.F., 2007. Fizjologia [Physiology]. PZWL Warszawa [in Polish].

Gąsiorowska D., Korzeniowska K., Jabłecka A., 2008. Homocysteine. Farm. Współ. 1, 169-175.

Gnacińska M., Zdrojewski T., Wierucki Ł., Kędzierski M., Rutkowski R., Bandosz P., Szpakowski P., Karpiński R., Szpajer M., Winiarska A., Wyrzykowski B., 2004. Współwystępowanie zaburzeń lipidowych i nadciśnienia tętniczego w populacji osób w wieku 50 lat [Coexistence of lipid disorders and arterial hypertension in the population of 50 year old subjects]. Art. Hypert. 2, 97-102 [in Polish].

Gosh P., Hale E.A., Lakshman M.R., 2001. Plasma sialic acid index of apolipoprotein J (SIJ): a new alcohol intake marker. Alcohol 25, 173-179.

Grundy S.M., 2005. Stanols esters as a component of maximal dietary therapy in the National Cholesterol Education Program Adult Treatment Panel III report. Am. J. Cardiol. 96, 27-50.

Gueguen S., Herbeth B., Pirollet P., Paille F., Siest G., Visvikis S., 2002. Changes in serum apolipoprotein and lipoprotein profile after alcohol withdrawal: effect of apolipoproteine E polymorphism. Alcohol. Clin. Exp. Res. 26, 501-508.

Harman N.L., Leeds A.R., Griffin B.A., 2008. Increased dietary cholesterol does not increase plasma low density lipoprotein when accompanied by an energy-restricted diet and weigh loss. Eur. J. Nutr. 47, 287-293.

Holman R.T., 1998. The slow discovery of the importance of omega3 essential fatty acids in human health. J. Nutr. 128, 427-433.

Imiela T., Grabowski M., 2007. Treatment of dyslipidemia. Cardiovas. Forum 12, 1-2, 23-31.

Ishur O., Sun C., Xiao P., Ashour A., Pan Y., 2002. A neutral beta-glucan from dates of the date palm, *Phoenix dactylifera* L. Carbohydr. Res. 14 (337), 1325-1328.

Jenkins D.J., Kendall C.W., Marchie A., Faulkner D.A., Wong J.M., de Souza R., Emam A., Parker T.L., Vidgen E., Lapsley K.G., Trautwein E.A., Josse R.G., Leiter L.A., Connelly P.W., 2003. Effects of a dietary portfolio of cholesterol-lowering foods vs lovastatin on serum lipids and C-reactive protein. J. Am. Med. Assoc. 290, 1-9.

Katan M.B., Grundy S.M., Jones P., Law M., Miettinen T., Paoletti R., Stresa Workshop Participants, 2003. Efficacy and safety of plant stanols and sterols in the management of blood cholesterol levels. Mayo Clin. Proceedings 78, 965-978.

Kłosiewicz-Latoszek L., Cybulska B., 2006. Rodzinna hipercholesterolemia – patogeneza, klinika i postępowanie [Familial hipercholesterolemia – pathogenesis, clinic and management]. Przew. Lekarz. 3, 80-86 [in Polish].

Kozłowska-Wojciechowska M., 2005. Jak zapobiegać hiperhomocysteinemii? Naturalne źródła folianów i witamin grupy B w polskiej diecie [How to prevent hyperhomocysteinemia? Natural sources folates and of vitamins of the B group in the Polish diet]. Czyn. Ryz. Supl. 11, 25-26 [in Polish].

Krawiec A., Cylwik B., Chrostek L., Supronowicz Z., Szmitkowski M., 2008. Wpływ przewlekłego spożywania alkoholu na stężenie lipidów, lipoprotein i apolipoprotein we krwi [The effect of chronic alcohol abuse on the lipids, lipoproteins and apolipoproteins concentrations in the sera]. Pol. Merk. Lek. 24, 144, 521 [in Polish].

Kris-Etherton P., Harris W.S., Appel L.J., 2002. Fish consumption, fish oil, omega-3 fatty acids and cardiovascular disease. Circulation 106, 2747-2757.

Lakshman M.R., Rao M.N., Marmillot P., 1999. Alcohol and molecular regulation of protein glycosylation and function. Alcohol 19, 239-247.

Lamisse F., Schellenberg F., Bouyou E., Delarue J., Benard J.Y., Couet C., 1994. Plasma lipids and alcohol consumption in alcoholic men: effect of withdrawal. Alcohol Alcohol. 29, 25-30.

Lecomte E., Herbeth B., Paille F., Steinmetz J., Artur Y., Siest G., 1996. Changes in serum apolipoprotein and lipoprotein profile induced by chronic alcohol consumption and withdrawal: determinant effect on heart disease? Clin. Chem. 42, 1666-1675.

Lichtenstein A.H., Appel L.J., Brands M., Carnethon M., Daniels S., Franch H.A., Franklin B., Kris-Etherton P., Harris W.S., Howard B., Karanja N., Lefevre M., Rudel L., Sacks F., Van Horn L., Winston M., Wylie-Rosett J., American Heart Association Nutrition Committee, 2006. Diet and lifestyle recommendations revision 2006. A Scientific Statement from the American Heart Association Nutrition Committee. Circulation. 114, 82-96.

Liinamaa M.J., Hannuksela M.L., Rämet M.E., Savolainen M.J., 2006. Defective glycosylation of cholesteryl ester transfer protein in plasma from alcohol abusers. Alcohol Alcohol. 41, 18-23.

Lonn E., Yusuf S., Arnold M.J., Sheridan P., Pogue J., Micks M., McQueen M.J., Probstfield J., Fodor G., Held C., Genest J., Heart Outcomes Prevention Evaluation (HOPE) 2 Investigators., 2006. Homocysteine lowering with folic acid and B vitamins in vascular disease. New Engl. J. Med. 354, 1567-1577.

Marmillot P., Rao M.N., Liu Q.H., 1999. Desialylation of human apolipoprotein E decrease its binding to human high- density lipoprotein and its ability to deliver estrified cholesterol to the liver. Metabolism 48, 1184-1192.

Miller E., Malinowska K., Gałęcka E., Mrowiska M., Kędziora J., 2008. Rola flawonoidów jako przeciwutleniaczy w organizmie człowieka [Role of flavonoids as antioxidants in human organism]. Pol. Merk. Lek. 24, 144, 556 [in Polish].

Mukamal K.J., Conigrave K.M., Mittleman M.A., Camargo C.A., Stampfer M.J., Willett W.C., Rimm E.B., 2003. Roles of drinking pattern and type of alcohol consumed in coronary heart disease in men. New Engl. J. Med. 348, 2, 109-118.

O'Neill F.H., Brynes A., Mandeno R., Rendell N., Taylor G., Seed M., Thompson G.R., 2004. Comparison of the effects of dietary plant sterol and stanol on lipid metabolism. Nutr. Metab. Cardiovasc. Dis. 14, 133-142.

O'Neill F.H., Sanders T.A., Thompson G.R., 2005. Comparison of efficacy on plant stanol ester and sterol ester: short term and long term studies. Am. J. Cardiol. 96 (supl.), 29-36.

Odabasi Z., Paetznick V.L., Chen E., Rodriguez J.R., McGinnis M.R., Ostrosky-Zeichner L., 2006. Differences in beta-glucan levels in culture supernatants of a variety of fungi. Med. Mycol. 3, 44, 267-272.

Pająk A., Wiercińska E., Polakowska M., Kozakiewicz K., Kaczmarczyk-Chałaś K., Tykarski A., Gaździk D., Zdrojewski T., 2005. Rozpowszechnienie dyslipidemii u mężczyzn i kobiet w wieku 20-74 lat w Polsce. Wyniki programu WOBASZ [Prevalence of dyslipidemia in men and women aged 20-74 in Poland. Results of program WOBASZ]. Kardiol. Pol. 63, 6 (supl. 4), 1-6 [in Polish].

Patel M.D., Thompson P.D., 2006. Phytosterols and vascular disease. Atherosclerosis 186, 12-19.

Pereira M.A., O'Reilly E., Augustsson K., Fraser G.E., Goldbourt U., Heitmann B.L., Hallmans G., Knekt P., Liu S., Pietinen P., Spiegelman D., Stevens J., Virtamo J., Willett W.C., Ascherio A., 2004. Dietary fiber and risk of coronary heart disease: a pooled analysis of cohort studies. Arch. Intern. Med. 165, 370-376.

Podolec P., Karch I., Pająk A., Kopeć G., Broda G., Drygas W., Rynkiewicz A., Zdrojewski T., Cieśliński A., 2006. Epidemiologia i prewencja. Przegląd polskich badań epidemiologicznych w kardiologii [Epidemiology and prevention]. Kardiol. Pol. 64, 1031-1037 [in Polish].

Riediger N.D., Othman R., Fitz E., Pierce G.N., Suh M., Moghadasian M.H., 2008. Low n-6:n-3 acid ratio, with fish- or flaxseed oil, in a high fat diet improves plasma lipids and beneficially alters tissue fatty acid composition in mice. Eur. J. Nutr. 47, 153-160.

Rimm E.B., Williams P., Fosher K., Criqui M., Stampfer M.J., 1999. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. BMJ 319, 1523-1528.

Rout D., Mondal S., Chakraborty I., Pramanik M., Syed S., 2005. Chemical analysis of a new (1-3), (1-6) branched glucan from an edible mushroom, Pleurotus florida. Carbohydr. Res. 16 (340), 2533-2539.

Rydén L., Standl E., Bartnik M., Van den Berghe G., Betteridge J. et. al., 2007. Guidelines on diabetes, pre-diabetes, and cardiovascular diseases: executive summary. The task force on diabetes and cardiovascular diseases of the European Society of Cardiology (ESC) and of the European Association for the Study of Diabetes (EASD). Eur. Heart J. 28, 88-136.

Sierakowska-Fijałek A., Baj Z., Kaczmarek P., Stępień M., Rysz J., 2008. Ocena zależności pomiędzy stężeniem homocysteiny a stężeniem wybranych parametrów przemiany lipidowej i cząsteczek adhezyjnych u dzieci z czynnikami ryzyka miażdżycy [Estimation of relation between homocysteine concentration and selected lipid parameters and adhesion molecules concentration in children with atherosclerosis risk factors]. Pol. Merk. Lek. 25, 148, 356 [in Polish].

Simopoulos A.P., 2002. The importance of the ratio of omega-6/omega-3 essential fatty acids. Biomed. Pharmacother. 56, 365-379.

Simopoulos A., 2008. The importance of the omega-6/omega-3 fatty acid ratio in cardiovascular disease and other chronic diseases. Exp. Biol. Med. 233, 6, 674-688.

Stanley J.C., Elsom R.L., Calder P.C., Griffin B.A., Harris W.S., Jebb S.A., Lovegrove J.A., Moore C.S., Riemersma R.A., Sanders T.A., 2007. UK Food Standards Agency Workshop Report: the effects of the dietary n-6:n-3 fatty acid ratio on cardiovascular health. Brit. J. Nutr. 98, 1305-1310.

Strauss M.H., Dorian P., Verma S., 2005 Fish oil supplementation and arrhythmias. JAMA 294, 2165-2171.

Superko R., 2000. Hipercholesterolemia and dyslipidemia. Current Treat. Opt. Cardiovasc. Med. 2, 173-187.

Szczygieł B., Boniecka I., Ukleja A., 2008. Metody oceny stanu odżywienia. Zapotrzebowanie na składniki odżywcze u chorych z otyłością [Methods of estimation of the nutrition status. Nutritional intake of nutrients in obesity]. Żyw. Człow. Metab. 35, 24-35 [in Polish].

Toole J.F., Malinow M.R., Chambless L.E., Spence J.D., Pettigrew L.C., Howard V.J., Sides E.G., Wang C.H., Stampfer M., 2004. Lowering homocysteine in patients with ischemic stroke to prevent recurrent stroke, myocardial infarction, and death: the Vitamin Intervention for Stroke Prevention (VISP) randomized controlled trial. JAMA 291, 565-575.

Vasisht S., Paut M.C., Sirvastaval L.M., 1992. Effect of alcohol on serum lipids and lipoproteins in male drinkers. Indian. J. Med. Res. 96, 333-337.

Witana K., Nowak R.J., Szpak A., Genowska A., 2006, The influence of the dietary habit on lipoprotein density in blood serum of men from Podlasie region. Rocz. Akad. Med. Białymst. 50, 82-86.

Zoungas S., McGrath B.P., Branley P., Kerr P.G., Muske C., Wolfe R., Atkins R.C., Nicholls K., Fraenkel M., Hutchison B.G., Walker R., McNeil J.J., 2006. Cardiovascular morbidity and mortality in the Atherosclerosis and Folic Acid Supplementation Trial (ASFAST) in chronic renal failure. J. Am. Coll. Cardiol. 47, 1108-1116.

ROLA ŻYWIENIA W LECZENIU ZABURZEŃ GOSPODARKI LIPIDOWEJ ORGANIZMU

Streszczenie. Najczęstszym, oprócz nadciśnienia tętniczego, czynnikiem ryzyka chorób sercowo-naczyniowych są zaburzenia lipidowe, które według dotychczasowych przeprowadzonych badań w Polsce dotyczą ponad połowy dorosłych osób. Żywienie odgrywa istotną rolę w regulacji gospodarki lipidowej organizmu. Dzięki racjonalizacji sposobu żywienia można kontrolować zaburzenia lipidowe organizmu, bez konieczności sięgania w wielu przypadkach po metody bardziej inwazyjne takie, jak leczenie środkami farmakologicznymi czy też stosowanie zabiegów kardiochirurgicznych. Odpowiednie postępowanie dietetyczne, jak również zmiana stylu życia (rezygnacja z palenia tytoniu, unikanie stresu, większa aktywność fizyczna) pozwala zmniejszyć stężenie cholesterolu całkowitego w surowicy krwi, polepszyć gospodarkę lipidową i zahamować progresję bądź wywołać częściową regresję zmian miażdżycowych w naczyniach wieńcowych, co wielokrotnie może uratować życie. W pracy przedstawiono aktualne zalecenia żywieniowe w leczeniu zaburzeń gospodarki lipidowej organizmu.

Słowa kluczowe: gospodarka lipidowa, cholesterol, zalecenia żywieniowe, miażdżyca, tłuszcz

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