REVIEW ARTICLE

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Exogenous dietary factors as important modulator of human lipid profile

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Abstract

Introduction. Estimation of lipid profile parameters in blood is an important element in the diagnosis of metabolic diseases, especially for evaluation of the whole state of health and identification of risk factors of civilization diseases. Exogenous dietary factors have great influence on laboratory lipid parameters, and improper nutrition habits or application of different diets can change the lipid profile. This is important in making clinical decisions by the doctor. Knowledge of associations between lipid profiles and exogenous factors derived from diets is still incomplete and underestimated.

Objective. The aim of this review is analysis of the role of the most popular diets, as an exogenous factor influencing the human lipid profile. Additionally, the role of the appropriate preparation of patients for laboratory examination of lipid profile is demonstrated.

State of knowledge. The most popular diets taken into account were high-fat, vegetarian, Mediterranean, and high-fibre, as well as diets based on low or high glycaemic index. The most negative effect on the all lipid profile parameters is connected with nutritional factors derived from the high glycaemic index diet. The most positive effect demonstrates an appropriate balanced diet, such the vegetarian and Mediterranean diets, which can significantly improve lipid profile parameters.

Conclusion. Awareness of the influence of exogenous factors derived from diet as an important modulator of human lipid profile is very important in medicine for undertaking an appropriate therapeutic decision. Adequate preparation of patients for laboratory examination and their education in this field is important and still needed.

Key words

lipid profile, laboratory tests, exogenous nutrition factors, diets

INTRODUCTION

Laboratory tests are procedures in which a sample of biological material (blood, urine and other body fluids) is examined to obtain information about the health status of the general population, and primarily to provide up-to date information about the actual health condition of individual persons. The tests supply precise and reliable data about specific health problems and constitute complementation of clinical and subjective examinations enabling correct diagnosis. Measurement of changes in the concentration of particular parameters reflect the course of a disease and therapy efficacy. They also allow prediction of recovery as well as survival time of patients [1, 2]. Estimation of the lipid profile in blood is an important element in the evaluation of the health state in the general population, identification of risk factors of metabolic disturbances and future diseases. Moreover, it is an essential element in diseases diagnostics and provides information about the best method of treatment.

Recent research has indicated that infusion of high-density lipoproteins (HDL) could be considered as a new anti-atherosclerotic method of treatment, beyond lipid profile improvement by conventional pharmacotherapy. This is extremely significant due to the epidemics of obesity, cardiovascular diseases, and risk of metabolic syndrome in developed countries [3, 4]. The primary aim of performing laboratory tests is to obtain reliable results which are adequate for the patient's actual clinical condition. There are many factors which may largely influence the results of laboratory tests, and contribute to the so-called pre-laboratory error. It is very important not only to reveal its source, but also awareness of the possibility of its occurrence, which should be considered both by the medical staff and patients. Patients should be informed about the recommendations for the performance of laboratory diagnostic tests because they can be the source of pre-laboratory error if they do not follow these recommendations [5, 6].

The composition of diets is a significant factor of lipid profile modulation; however, its role is frequently underestimated. Diet factors should not be considered as a strict source of a pre-laboratory error, but nutritional exogenous factors can substantially affect the results of lipid laboratory tests. Nevertheless, ignorance of this fact may cause false interpretation of the diagnostic test results; in this aspects it can therefore be considered as a source of pre-laboratory error. Moreover, an improperly balanced diet may constitute a risk factor of some diseases or intensify existing disorders [7]. Currently, in the view of slim figure culture, as well as increasing popularity of new diets (especially reducing the

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body mass), their role and influence on the lipid profile seems to be underestimated. On the other hand, the purpose of lipid profile examination may include assessment of the efficacy of the applied diet in the treatment of civilisation diseases. Moreover, the practical application of nutrition as a preventative or corrective treatment of disease (diet therapy) is also well known [7, 8, 9].

The review is based on literature research, using the PubMed database, taking into account papers published between 1992–2018 using the searching terms or their combination: "lipid profile", "dyslipidaemia", "laboratory parameters", "laboratory examination", "pre-laboratory errors", "total cholesterol", "high density lipoproteins cholesterol", "low density lipoproteins cholesterol", "triglycerides", "lipids recommendation", "diets", "nutrition way" "vegetarian diet", "Mediterranean diet", "high-fat diet", "high-fibre diet", "high-protein diet", "glycaemic index diet". Finally, 93 items of synthetic original papers and reviews were selected which seemed to be the most useful for this issue.

OBJECTIVE

The aim of this review is to analyze the role of the most popular diets as exogenous dietary factors which can influence the human lipid profile, especially such parameters as total cholesterol (T-CH), high density lipoproteins cholesterol (CH-HDL) and low density lipoproteins cholesterol (CH-LDL), as well as the concentration of triglycerides (TG). An additional aim was to show the role of the appropriate preparation of patients for laboratory examination of lipid profile to avoid false results, as well as current recommendations and cut-off values for the laboratory parameters established by different national scientific societies. Attention is also drawn on the various sources of pre-laboratory errors. Information about this is especially important for the correct assessment of the clinical state of the examined patients, and in making the appropriate clinical decisions by the doctor. Moreover, knowledge about the associations between lipid profile and dietary factors is still incomplete and underestimated.

STATE OF KNOWLEDGE

Recommendations concerning monitoring of lipid profile parameters in the general population and risk groups. The basic parameters of lipid profile routinely determined in medical diagnostic laboratories are: total cholesterol concentration, high density lipoproteins cholesterol and low density lipoproteins cholesterol as well as triglycerides concentration. Patient care issues are generally related to screening, diagnosis and treatment, and are based on the measurement results of serum lipids parameters. The reference values of laboratory parameters of lipid profile are established by scientific association experts on the basis of epidemiological and analysis of clinical survey results [10, 11, 12]. Generally recommended blood values of lipid laboratory parameters, based on recommendation of different societies for European, Japanese and American populations, are presented in Table 1 [13, 14, 15, 16, 17].

Table 1. Recommended values of lipid parameters in particular populations

Parameter	European Society of Cardiology and the European Atherosclerosis Society (ESC/EAS) [13,14] (European population)		Japan Atherosclerosis Society (JAS) [15] (Japanese population)		Third Report of the National Cholesterol Education Program – Adult Treatment Panel III (NCEP-ATPIII) [16,17] (American population)	
Total cholesterol mg/dL (mmol/L)	115–190 (3.0–5.0)		<220 (<5.7)		< 200 (< 5.1)	
Cholesterol LDL mg/dL (mmol/L)	<70 (<1.8)	Very high risk patients after heart infarct or stroke *SCORE ≥ 10%	< 120 (<3.1)	High risk ≥ 2%	<100 (<2.5)	CHD** or CHD risk equivalents*** 10-year risk > 20%
	<100 (<2.5)	High risk patients SCORE 5–10%	<140 (<3.6)	Intermediate risk SCORE ≥ 0.5–2%	<130 (<3.3)	Moderately high risk: 2+ risk factors**** (10-year risk 10% to 20%) Moderate risk: 2+ risk factor**** (10-year risk <10%)
	<115 (<3.0)	Moderate or small risk patients SCORE < 5%	<160 (<4.1)	Low risk SCORE < 0.5%	<160 (<4.1)	0 – 1 risk factor
Cholesterol HDL mg/dL (mmol/L)	Males≥40 (≥1.0) Females≥45 (≥1.2)		Males ≥40 (≥1.0) Females ≥40 (≥1.0)		Males ≥40 (≥1.0) Females ≥40 (≥1.0)	
Triglicerydes mg/dl (mmol/L)	≤ 150 (≤1.7)		≤ 150 (≤1.7)		≤ 150 (≤1.7)	

* SCORE (Systematic Coronary Risk Estimation) – estimates the 10-year risk of a first fatal atherosclerotic event – heart attack, stroke, or other occlusive arterial disease, including sudden cardiac death. Range is used to assess individual risk of death due to circulatory system diseases. Calculation is carried out using SCORE risk Tables.
** CHD includes history of myocardial infarction, unstable angina, stable angina, coronary artery procedures (angioplasty or bypass surgery), or evidence of clinically significant myocardial ischemia.
*** CHD risk equivalents include clinical manifestations of non-coronary forms of atherosclerotic disease (peripheral arterial disease, abdominal aortic aneurysm, and carotid artery disease [transient ischemic attacks or stroke of carotid origin or >50% obstruction of a carotid artery]), diabetes, and 2+ risk factors with 10-year risk for hard CHD >20%.
**** Risk factors include cigarette smoking, hypertension (BP 1≥40/90 mm Hg or antihypertensive medication), low HDL cholesterol (<40 mg/dL), family history of premature CHD (CHD in male first-degree relative <55 years of age; CHD in female first-degree relative <65 years of age), and age (men ≥45 years; women ≥55 years).

It is very important to know the limit values of the lipid profile recommended and accepted by appropriate scientific societies or organisations dedicated to particular populations. Some divergences may result in misinterpretation of laboratory test results and increased economic costs of patients treatment, as well as laboratory examination [18, 19, 20]. Due to this fact, there is a needs to harmonize the target values of lipid research results in medical diagnostic laboratories, not only in Poland but worldwide, which is a constant trend in international laboratory control systems [13, 14, 21].

In order to properly diagnose of dyslipidaemia and adequate interpretation of obtained results, relevant rules should be kept in all diagnostic laboratories. First of all, it is important to put on the laboratory sheets, reference values of lipid profile parameters recommended by experts from appropriate scientific societies. Secondly, the units of obtained results should be given, as well as methods applied if they differ from those routinely used. This is very important and should be mandatory, because the laboratory sheets sometimes present the results with values and units other than those recommended. This mainly concerns reference ranges for low density lipoproteins cholesterol [14, 15, 16, 17]. The consequence of such discrepancies are an inappropriate reflection of the clinical condition of the patient, and the possibility of problems in detecting a disease or predisposition to a disease. It is also related to the lack of implementation of appropriate clinical procedures as soon as possible. For example, in the NATPOL 2011 epidemiological study which assessed the risk of development of cardiovascular diseases in the Polish population aged 18–79, 65% of hypercholesterolaemia cases were observed to have been undiagnosed due to misinterpretation of the results [22, 23]. Providing unified values of lipid management in laboratory sheets, as recommended by appropriate scientific societies, will improve the efficacy of dyslipidaemia diagnosis and treatment. Moreover, this could provide family medicine and primary care doctors with appropriate diagnostic tools; it may also prevent the deliberate decision of patients to reduce drugs dosage or its cessation [14, 24]. It is very important to develop clinical practice guidelines (CPGs) relating to laboratory diagnostic testing and its continuous updating. Its aim is to standardize the practice and improve patient care based on the best available evidence [25, 26].

In the general population, lipid profile examination is recommended every five years in persons up to the age of twenty, every two years with an average risk of cardiovascular disease (CVD), and every two to six months for those at high risk. This mainly concerns males aged over 40 and females over 50, after the menopausal period. Examination of basic lipid parameters should be carried out in the fasting state, and interpretation of the final results performed on the basis of two or three designations. If the values for total cholesterol differ by more than 30 mg/dL, the examination should be repeated after one to eight weeks [27, 28]. According to recent reports, analysis of the lipid profile could be conducted not only in a fasting state, which is currently recommended by the European scientific societies, but also in a postprandial state. Interpretation of these results should be performed carefully, taking into account clinical and laboratory implications of the situation. It is especially crucial to determine the cut-off value for particular parameters of lipid profile, including flagging their desirable concentration [29]. Some authors suggest a greater use of postprandial lipid determination which would contribute to the establishment of an evidence-based approach to the implementation and evaluation of empirical interventions, the aim of which would be to improve the nutrition-related health in the population [30]. Lipid profile cannot be performed under psychological or physical stress, after recent heart infarct, stroke, during pregnancy, injury or body mass loss. In families with genetic-conditional hypercholesterolaemia and precocious cardiovascular disease, lipid profile assessment should be carried out at the age of two years [1, 31].

Principles of patient preparation for laboratory examination of lipid profile. First of all, the results of a laboratory test have to be reliable and should reveal the actual condition of the patient from the aspect of clinical diagnosis of disease. Sometimes, lack of correspondence in laboratory tests result with the patient's clinical condition may be caused by a mistake in the analytical phase (performance of the test). The priority of the laboratory should be the control of random and systematic mistakes by taking part in extra-laboratory evaluation programmes, as well as carrying out regular intra-laboratory controls in accordance with obligatory procedures.

One of the important elements which influence laboratory test results are nutritional factors derived from diet, or lack of an appropriate fasting period before the examination. An adequately balanced diet provides the organism with all indispensable nutrients as well as energy for homeostasis of the organism. However, an improperly composed diet disturbs this equilibrium and may also disturb many biochemical parameters. It can be considered as a source of some kind of factors which may result in a pre-laboratory error. From the aspect of the discussed issue, a diet rich in fats mostly influences lipid management parameters, and may affect measurement results of both total cholesterol, its fractions (HDL and LDL) and triglycerides. This is why, directly before the examination, the patient should be prepared properly [32, 33]. It is especially significant to inform the patient about the way of preparation for the laboratory examination. Detailed recommendations concerning preparations for laboratory tests are as follows:

1) before scheduled collection of material, the patients should not change their diet for at least three weeks, so that the test reveals the actual condition of the organism;
2) patients should not consume alcohol as it may cause a transient increase in the concentration of some parameters (triglycerides, free fatty acids and glycerol);
3) patients should fast for at least 12 hours before the test, because the meal may change the values of many parameters (especially glucose, triglycerides, cholesterol fractions, as well as phosphates);
4) blood samples should always be collected at the same time (in the morning), without intensive physical activity [34, 35, 36].

The scheme of relationship between laboratory test results and sources of potential errors connected with patients conditions is presented on Figure 1.

Lipid and carbohydrate metabolism parameters are mostly sensitive to nutritional factors. In order to eliminate pre-laboratory errors, the patient should be precisely informed about the rules of preparation for conducting laboratory

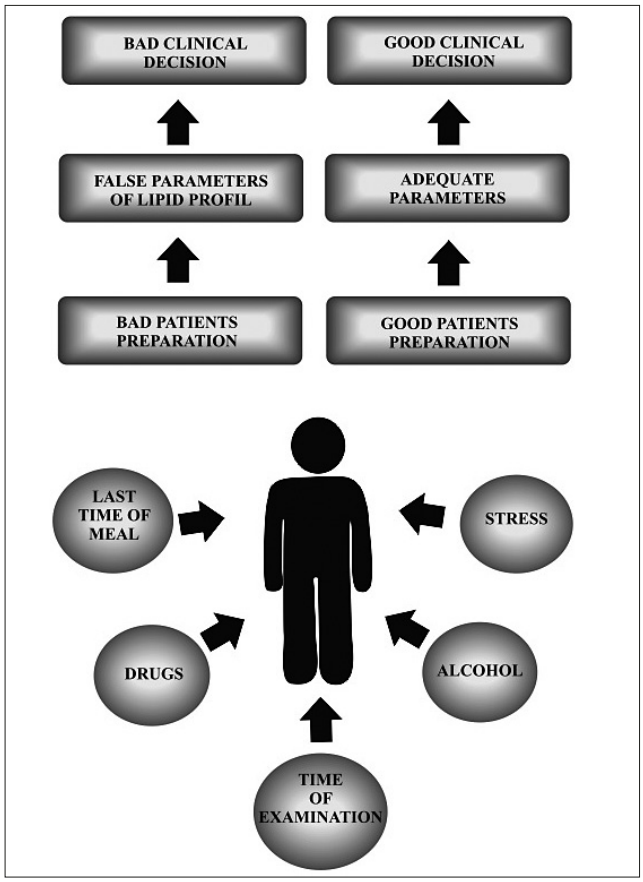

Figure 1. Relationship between laboratory test results and sources of potential errors

tests, because of the possibility of occurrence of a discrepancy between the result of the laboratory test and the clinical state of the patient [5, 37].

The most popular diets as sources of exogenous factors influencing lipid profile. The word "diet" comes from the Greek and literally means "way of life", including also the way of eating. The most preferable is well-balanced way of eating, composed of a relevant ratio of proteins (12–15%), fats (30%, including a minimum of 3% of unsaturated fatty acids) and carbohydrates (50–60%, including 10% of simple sugars, even up to 70% instead of fats). Such a diet provides the optimum amount of energy and supplies the organism with the essential nutrients necessary for growth, energy production and repair processes, and includes indispensable amounts of mineral components (micro- and macro-elements), vitamins and fibres [38, 39, 40]. Another type of diet is the therapeutic diet used in the prophylaxis and treatment of many diseases. It is a modification of a normal diet, being a special method of nutrition which differs from the rational method. It takes into account the quantity and quality of food and is matched to the actual and special needs of a patient, e.g. ability to ingest, digest and metabolize due to disease limitations.

Other ways of nutrition are considered as alternative diets in which some groups of products are specifically selected or deliberately omitted, or special ways of food preparation are applied [39, 40, 41]. Currently, the expertise of dietitians plays an important role in health guidance and improving the state of health: for example, it has been revealed that changes in body weight are significantly greater when patients were supported by dietitians during routine counseling than in the case of lack of such support. Additionally, the primary care outpatient clinic is an important source of information about healthy nutrition for patients, its influence on different biochemical laboratory parameters, especially on lipids and carbohydrate profile [42, 43, 44].

The way of nutrition is very important for maintaining good general health condition of the body; however, awareness of how nutritional factors can affect the human organism, including lipid profile parameters, is very low and often controversial, which is why it became the purpose of this review. Apart from a high-fat diet, which obviously affects the lipid profile, other diets are also mentioned in this review – vegetarian, Mediterranean and high-fibre diet, as well as diets based on a low or high glycaemic index.

Vegetarian diet. This diet is one of the most popular, best recognised and longest-known way of nutrition avoiding products of meat origin. Variants of the vegetarian diet are: semi-vegetarian diet, which excludes red meat; the lacto-ovo vegetarian diet, which excludes red meat, poultry and sea food; lacto vegetarian diet, which excludes all kinds of meat and eggs; ovo vegetarian diet, which excludes all kinds of meat and milk products; and the vegan diet, which excludes all kinds of meat, fish, milk products and eggs.

The vegetarian diet is regarded as a low fat diet, because it includes a limited amount of fats and saturated fatty acids, which are replaced by poly- and mono-unsaturated fatty acids. From this aspect, the vegetarian diet constitutes a significant factor contributing towards a lipid profile change in blood. After its application, a considerable decrease in total cholesterol and LDL cholesterol concentrations by about 25–50% are observed in blood, and some data report even 65% [45, 46, 47]. Some studies revealed the most significant role of oleic acid derived from rapeseed oil and olive rich in monounsaturated fatty acids in decreasing concentration of total cholesterol and LDL fraction, as well as increasing the HDL cholesterol fraction. In turn, omega-3 polyunsaturated fatty acids (α-linolenic acid) play the most important role in decreasing triglycerides and VLDL concentrations, as well as reducing post-meal lipaemia (activation of lipoprotein lipase, responsible for VLDL and chylomicrons catabolism). Omega-6 polyunsaturated fatty acids (derivatives from linoleic acid) mainly decrease total cholesterol concentration [48, 49, 50, 51].

In the vegetarian diet, replacing proteins of animal origin with the those of plant origin is of great significance. Literature data emphasise the biggest role of soya proteins in the regulation of the concentration in lipids. It causes a decrease in total cholesterol and triglycerides concentrations, as well as an increase, however small, of HDL cholesterol fraction. It is probable that the amino-acidic composition of this protein is responsible for this phenomenon. Lysine or methionine have hypercholesterolemic activity, whereas the greater part of arginine or glycine in amino-acids pool increases the normalisation of cholesterol concentration in blood [52, 53].

The vegetarian diet, due to a significant supply of fibre and phytosterols, may diminish the risk of cardiovascular diseases, neoplasms, diabetes or obesity, and it is recommended for persons belonging to these diseases risk groups. However, it is not a well-balanced diet, and vegetarians are especially

subject to deficiencies in some mineral components, as well as vitamin D, vitamin B_2 and proteins (depending on the diet variant) which definitely influences the health condition [45, 46].

Mediterranean diet. This diet is considered the healthiest and the safest way of nutrition which improves numerous biochemical and clinical parameters. It is characterized by large consumption of vegetal products rich in phytosterols, such as sitosterol and campesterol, having a great influence on lipid profile changes. These compounds significantly reduce cholesterol absorption in the intestine which decreases its blood concentration. With plant sterols consumption (2–3g daily), total cholesterol concentration decreases by about 14%. Extensive and long-term studies have shown that this way of nutrition is not only an important traditional diet within the Mediterranean areas, but also has multiple effects of the diet on public health nutrition, society, and the environment [54, 55].

The efficacy of the Mediterranean diet was proved in the limiting of the prevalence cardiovascular diseases, and by a significant decrease in total cholesterol, LDL and VLDL cholesterol fractions, and Apo B, as well as triglycerides concentrations with a simultaneous increase in the concentration of HDL cholesterol fraction. Enhancement of the antioxidant systems capacity of the organism is one of the mechanisms resulting from this type of diet application [56, 57].

Moreover, it is indicated that the Mediterranean diet positively influences the metabolic status, especially in metabolically healthy obese and metabolically obese normal weight phenotype young. The latter, the so-called MONW syndrome, constitutes an increasingly growing problem of late detection of an increasing risk of cardio-metabolic disease [58]. Results of the PREDIMED (PREvención con DIeta MEDiterránea) trial in long-term observations revealed its beneficial effect on cardiovascular disease prevention in the populations of the developed countries. Almost seven-and-a-half thousand people with high CVD risk were examined, and two variants of vegetarian diets examined, without energy restriction and no special intervention on physical activity. The Mediterranean diet supplemented with extra-virgin olive oil and diet supplemented with nuts, as well as a control diet (advice on a low-fat diet) were applied. The study revealed significant improvements in classical and emerging CVD risk factors, among others on the parameters of lipid profile and lipoprotein particles. It was revealed for both variants of Mediterranean diets, especially rich in unsaturated fat and polyphenols, which was also supported by other multicentre data [46, 59, 60].

High fat diet. Another type of diet which has evident influence on lipid profile is the high fat diet, elaborated by Jan Kwasniewski, MD (also known as the Atkins' Diet). This is not a high fat diet in the general sense of the word because the Atkin's diet is defined as a low carbohydrates diet with simultaneous increased fat intake, but this second aspect is the most popular and better known. Although this diet is often defined as an optimal diet, it is completely confusingly, because it does not have much in common with the optimal supply of energy and nutrients. The majority of literature data reveal that this diet is based on the ingestion of as much fatty foods as possible, with the simultaneous exclusion of carbohydrates (sugar, sweets, bread, fruit). It provides the organism with six times more cholesterol than recommended for daily ingestion [61, 62]. Application of this diet was observed to increase total cholesterol and LDL cholesterol fraction concentrations. The biggest changes were found in LDL cholesterol fraction. Other surveys revealed this tendency in lipid profile changes; however, they mainly concerned only reference values of the above parameters. Cholesterol concentration increase in blood was observed mainly at cholesterol ingestion in amounts of 200–300 mg daily, whereas a further increase in cholesterol intake resulted in a lower increase of its concentration in blood [63, 64, 65]. However, literature data are not univocal, and there are reports which do not indicate a negative influence of this type of diet on lipid profile. There are also some observations concerning both an increase and decrease of total cholesterol and LDL cholesterol fraction concentrations, as well as a significant drop in triglycerides concentration in persons on this diet. At the initial stage of this diet, HDL cholesterol fraction concentration increased but dropped after some time. This suggests that some divergences in elicited observational results may depend on the amount of weight loss, as well as the kind of ingested fat during diet application [48, 66, 67]. It is emphasised that a high-fat diet which provides the organism with large amounts of triglycerides promotes inhibition of their endogenous production in liver, and along with carbohydrates limitation, it activates metabolism of fats in tissue reserves. Besides, exogenous triglyceride enhances protein lipase activity which is responsible for their hydrolysis, resulting in postprandial lipaemia decrease. However, the significant influence of high-fat diets indicates a reasonable concern for the safety of its application, especially in the context of its effect on CVD development, increased risk of atherosclerosis, and postprandial hypercholesterolaemia [68, 69].

High protein diet. This is the recommended way of nutrition in conditions of extreme starvation, severe burns or injuries, and diseases accompanied by high temperature. This type of diet is based on providing the body with an adequate amount of protein of high nutritional value, mainly for anabolic purposes, such as cells and tissues composition and reconstruction, as well as antibodies, enzymes and hormones synthesis. In turn, the basic presumption of this type of diet when applied to reduce body mass (e.g. Dukan Diet) is initially the total elimination and subsequently significant limitation of carbohydrates and fats provided, and replacing them with an increased amount of proteins. It aims at using endogenous reserves of fats. The amount of consumed protein in this diet largely exceeds (twice or three times) the recommended daily intake of this component in adults (0.9 g/kg of body mass/daily) and amounts to 2–3 g/kg of body mass/daily [70, 71]. It is mainly rich in proteins of animal origin as well as of vegetal origin – mostly gluten. It should significantly reduce atherogenic lipids fraction (total cholesterol and triglycerides) and increase the HDL cholesterol fraction. In hyperlipidaemic patients, a high protein diet rich in gluten resulted in a decrease in LDL cholesterol fraction and triglycerides by 10% and 19%, respectively, but the HDL cholesterol fraction concentration did not change significantly. Other studies revealed a significant increase in total cholesterol and HDL cholesterol fraction without considerable changes in LDL cholesterol fraction, due to no gluten diet application [72, 73, 74].

However, this way of nutrition involves many threats, especially to persons with kidney or liver insufficiency and for whom high protein diet is therefore absolutely inadvisable. In the case of renal failure, this diet increases glomerular filtration which raises intraglomerular pressure and results in progressive renal injury. It is strictly related to water-electrolyte balance disturbances due to an increased amount of sodium (even above 2g) provided, especially with processed meat, poultry and fish. In turn, the risk for persons with hepatic failure is the inability to metabolize ammonia and mercaptans, produced by the processing of nitrogen compounds. The high protein diet was also found to contribute to arteriosclerosis, which seems to be the result of consumption of a lot of meat rich in saturated fatty acids. However, some authors indicate that the application of a high protein diet had no harmful effects on the values of blood lipids, nor on liver and kidney function [70, 75, 76].

High fibre diet. This type of diet is based on providing the organism with higher than traditional amounts of fibre – especially water-soluble. Also known as the "high residue diet", it is basically recommended in cases of constipation and prophylaxis of intestinal diseases. Moreover, it prevents development of arteriosclerosis, diabetes and obesity, which are directly associated with the properties of fibre contained in food. Fibre is a mixture of substances of vegetal origin. It is composed of water-soluble components, such as pectines, gums, some polysaccharides, including β-glucan, as well as non-soluble lignin, cellulose and haemicellulose. It is neither digested nor absorbed in the digestive tract (resistant to hydrolytic activity of digestive enzymes), and binds with cholesterol and forms non-absorbable complexes which increase its faecal excretion.

Another mechanism is the decrease in cholesterol concentration in blood and liver, which results in an increase of endogenous synthesis of cholesterol. Cholesterol is transformed into bile acids which accelerate its metabolism and results in its excretion, as well as reduction of its concentration in blood. Cholesterol LDL fraction also decreases [77, 78]. The highest efficacy of the high fibre diet was observed in hyperlipidaemic patients with considerably reduced total cholesterol and LDL cholesterol fraction concentrations. Pectines, guar gum and psyllium were indicated as factors responsible for these positive changes. However, significant changes in HDL and triglyceride levels were not observed. Other data indicate that a high-fibre diet regulates the concentration of these parameters [79, 80, 81].

Diets based on glyacemic index. Literature data reveal that diet based on the glycaemic index considerably affects lipid profile parameters. The glycaemic index (GI) is an average proportional increase in the concentration of blood glucose after ingesting a product containing 50g of absorbable carbohydrates, which was adopted as the basis for the GI scale (100%). It allows the classification of dietary products, based on their effect on glucose concentration in blood two or three hours after ingestion (post-meal glycaemia). For low GI products, it amounts to 55, whereas for high GI products it is > 70 [70, 82]. Diets based on high GI (e.g. Omish Diet, Diamonds' Diet) mostly affect the lipid profile parameters, they contribute especially to the increase in triglycerides concentration. It is connected with utilization of the provided high GI products to endogenous TG synthesis. All remaining parameters of lipid profile also change but to a different extent; however, the effect of a high glycaemic diet on cholesterol fractions is not clear [83, 84]. Some literature data do not confirm the influence of a high GI diet on concentrations of both total cholesterol and its LDL and HDL fractions. Nevertheless, the majority of studies confirms an increase in the concentration of LDL cholesterol fraction, together with a decrease in HDL cholesterol fraction concentration, with a significantly elevated TG concentration. This effect probably results from the co-existence of strictly connected physiological processes, e.g. postprandial glycaemia increase or hyperinsulinaemia, which results in increased hepatic triglycerides synthesis and increased VLDL synthesis [85, 86, 87]. Besides, replacement of saturated fatty acids with carbohydrates results in an even higher increase of their concentration than in the case of replacing them with mono-unsaturated fatty acids. It was proved that in the case of providing the organism with excessive amounts of carbohydrates of high GI and low fat contents, HDL cholesterol fraction concentration was reduced, but the ratio of total cholesterol/HDL cholesterol and triglycerides concentration increased. A low GI diet produces opposite results, especially in the case of triglycerides concentration which causes their significant reduction [88, 89, 90].

Information about the influence of nutritional factors derived from the most popular diets presented in this review on lipid profile parameters are given in Table 2.

Table 2. Influence of most popular diets on human lipid profile parameters.

Type of diet applied	T-CH	CH-HDL	CH-LDL	TG
Vegetarian	↓	↑	↓	↓
mediterranean	↓	↑	↓	↓
high-fat	↑	↓	↑	↓
high-protein	(-)	(-)	↓	↓
high-fibre	↓	↑	↓	(-)
based on low GI	(-)	↑	↓	↓
based on high GI	↑	↓	↑	↑

T-CH – total cholesterol; CH-HDL – high density lipoproteins cholesterol; CH-LDL – low density lipoproteins cholesterol; TG – triglycerides; GI – glycemic index.
↓ – lowering of level; ↑ – increasing of level; (-) – without significant changes.

As shown in Table 2, all presented diets influence the blood lipid profile, but some of them also trigger changes in other directions. Generally, the cardio-protective effect of the vegetarian, Mediterranean, low GI and high fibre diets was observed in relation to improvement in almost all profiles of lipid parameters. High fat and high GI diets showed an atherogenic effect, visible in the deterioration of most lipid parameters. Although diets based on a high-protein intake exerted a lowering effect on LDL and triglycerides concentration, it disturbed the function of vital organs, like kidneys and liver, and had a negative influence on many other metabolic pathways.

The rules of proper nutrition are recommended by appropriate scientific societies and published in the form of the Healthy Eating Pyramid in purpose to facilitate public understanding of the nutritional recommendations. The first was published in 1992 by the United States Department of Agriculture (USDA) and has been evolving ever since.

Currently in Poland, nutritional recommendations created by National Food and Nutrition Institute (NFNI) of 2009 are mandatory. According to this pyramid, oil, meat, fish and dry seeds of legumes are foods that people should eat occasionally. The base of the NFNI pyramid consists of cereal products, fruits and vegetables, but most importantly, physical activity is indicated [91, 92]. When discussing the problem of linking a diet with the lipid profile, it is necessary to just emphasize the importance of physical activity as a factor shaping the general health condition. Physical activity is one of the corners well-known in the dietetic conception of the Healthy Eating Pyramid and Healthy Eating Plate, which summarizes the best dietary information available today. According to the experts from the Harvard School of Public Health, composing a whole lifestyle, not only diet in line with the above guidelines, can lead to a lower risk of heart disease and premature death [93].

CONCLUSIONS

The analysed literature data clearly confirm the influence of nutritional exogenous factors derived from diets on lipid profile parameters. This influence is diverse and should be taken into account in interpreting the results of laboratory tests, and can be considered as a kind of pre-laboratory error. This should be known and recognised both by patients and medical staff (doctors and laboratory diagnosticians). Being aware of diet factors on the results of lipid profile examination may protect against unintentional falsification of laboratory results. This is of vital importance because the composition of different diets has not been fully examined, and there are no reliable results documenting their influence on the lipid profile parameters. The temporary popularity of some "miraculous diets" creates large possibilities for the occurrence of a difficult to identify pre-laboratory error, and can cause an incorrect diagnosis by clinicians. Medical staff should take preventive or educational actions, informing patients about the need for proper preparation before laboratory examination. Moreover, doctors should always interview the patients in terms of dietary issues and physical activity in order to establish the patient's actual condition in relation to the results of laboratory tests.

This review is an interesting approach on the effects of nutritional exogenous factors derived from some most popular diets as important modulators of human lipid profile from the aspect of appropriate patient care.

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REFERENCES

1. Kallner A. Measurement performance goals: How they can be estimated and a view to managing them. Scand J Clin Lab Invest Suppl. 2010; 242: 34–39. doi: 10.3109/00365513.2010.493364
2. Walz SE, Darcy YP. Patient safety & post-analytical error. Clin Lab Med. 2013; 33(1): 183–194. doi: 10.1016/j.cll.2012.10.001
3. Greenland P, Alpert JS, Beller GA, Benjamin EJ, Budoff MJ, Fayad ZA, et al. ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2010; 56(25): e50–103. doi: 10.1016/j.jacc.2010.09.001
4. Rosenson RS, Davidson MH, Hirsh BJ, Kathiresan S, Gaudet D. Genetics and causality of triglyceride-rich lipoproteins in atherosclerotic cardiovascular disease. J Am Coll Cardiol. 2014; 64(23): 2525–2540. doi: 10.1016/j.jacc.2014.09.042
5. Dikmen ZG, Pinar A, Akbiyik F. Specimen rejection in laboratory medicine: Necessary for patient safety? Biochem Med. 2015; 25(3): 377–385. doi: 10.11613/BM.2015.037. eCollection 2015
6. Simundic AM, Lippi G. Preanalytical phase--a continuous challenge for laboratory professionals. Biochem Med. (Zagreb) 2012; 22(2): 145–149.
7. Song S, Paik HY, Park M, Song Y. Dyslipidemia patterns are differentially associated with dietary factors. Clin Nutr. 2016; 35(4): 885–891. doi: 10.1016/j.clnu.2015.06.002
8. Heitmann BL, Lissner L. Can adverse effects of dietary fat intake be overestimated as a consequence of dietary fat underreporting? Public Health Nutr. 2005; 8(8): 1322–1327
9. Tangvik RJ, Tell GS, Eisman JA, Guttormsen AB, Henriksen A, Nilsen RM, et al. The nutritional strategy: four questions predict morbidity, mortality and health care costs. Clin Nutr. 2014; 33(4): 634–641. doi: 10.1016/j.clnu.2013.09.008
10. Brunzell JD, Davidson M, Furberg CD, Goldberg RB, Howard BV, Stein JH, et al. Lipoprotein management in patients with cardiometabolic risk: consensus conference report from the American Diabetes Association and the American College of Cardiology Foundation. J Am Coll Cardiol. 2008; 51(15): 1512–1524. doi: 10.1016/j.jacc.2008.02.034
11. Langsted A, Freiberg JJ, Nordestgaard BG. Fasting and nonfasting lipid levels: influence of normal food intake on lipids, lipoproteins, apolipoproteins, and cardiovascular risk prediction. Circulation 2008; 118(20): 2047–2056. doi: 10.1161/CIRCULATIONAHA.108.804146
12. Vesper HW, Wilson PW, Rifai N. A message from the laboratory community to the National Cholesterol Education Program Adult Treatment Panel IV. Clin Chem. 2012;58(3):523–527. doi: 10.1373/clinchem.2011.178202
13. Catapano AL, Graham I, De Backer, Wiklund O, Chapman MJ, Drexel H, et al. 2016 ESC/EAS Guidelines for the Management of Dyslipidaemias: The Task Force for the Management of Dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS) Developed with the special contribution of the European Assocciation for Cardiovascular Prevention & Rehabilitation (EACPR). Eur Heart J. 2016; 37(39): 2999–3058. doi: 10.1093/eurheartj/ehw272
14. Stępińska J, Solnica B, Kulpa J, Jankowski P, Kalarus Z, Opolski G, et al. The need to harmonize the target values of lipid research results in medical diagnostic laboratories in Poland. Diagn Lab. 2012; 48: 473–474.
15. Teramoto T, Sasaki J, Ishibashi S, Birou S, Daida H, et al. Executive summary of the Japan Atherosclerosis Society (JAS) guidelines for the diagnosis and prevention of atherosclerotic cardiovascular diseases in Japan – 2012 version. J Atheroscler Thromb. 2013; 20(6): 517–523.
16. Grundy SM, Cleeman JI, Merz CN, Brewer HB Jr, Clark LT, Hunninghake DB, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. Circulation 2004; 110(2): 227–239.
17. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): 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) final report. Circulation 2002; 106(25): 3143–3421.
18. An International Atherosclerosis Society Position Paper: Global recommendations for the management of dyslipidemia – full report. Expert Dyslipidemia Panel of the International Atherosclerosis Society Panel members. J Clin Lipidol. 2014; 8(1): 29–60. doi: 10.1016/j.jacl.2013.12.005
19. Bays HE, Jones PH, Orringer CE, Brown WV, Jacobson TA. National Lipid Association. National Lipid Association Annual Summary of Clinical Lipidology. J Clin Lipidol. 2016; 10(1 Suppl): S1–43. doi: 10.1016/j.jacl.2015.08.002
20. Lippi G, Banfi G, Church S, Cornes M, De Carli G, Grankvist K, et al. Preanalytical quality improvement. In pursuit of harmony, on behalf of European Federation for Clinical Chemistry and Laboratory Medicine (EFLM) Working group for Preanalytical Phase (WG-PRE). Clin Chem Lab Med. 2015; 53(3): 357–370. doi: 10.1515/cclm-2014–1051
21. Hoerger TJ, Wittenborn JS, Young W. A cost-benefit analysis of lipid standardization in the UnitedStates. Prev Chronic Dis. 2011; 8(6): A136.

22. Begum H, Li B, Shui G, Cazenave-Gassiot A, Soong R, Ong RT, et al. Discovering and validating between-subject variations in plasma lipids in healthy subjects. Sci Rep. 2016; 6: 19139. doi: 10.1038/srep19139
23. Zdrojewski T, Rutkowski M, Bandosz P, Gaciong Z, Jędrzejczyk T, Solnica B, et al. Prevalence and control of cardiovascular risk factors in Poland. Assumptions and objectives of the NATPOL 2011 Survey. Kardiol Pol. 2013; 71(4): 381–392. doi: 10.5603/KP.2013.0066
24. Craig SR, Amin RV, Russell DW, Paradise NF. Blood cholesterol screening influence of fasting state on cholesterol results and management decisions. J Gen Intern Med. 2000; 15(6): 395–399.
25. Descamps OS, Cooney MT, De Backer G, Graham I. A simple multiplier to calculate the impact of HDL cholesterol on cardiovascular risk estimation using SCORE. Atherosclerosis 2012; 222(2): 564–566. doi: 10.1016/j.atherosclerosis.2012.03.035
26. Misra S, Moberg-Aakre K, Langlois M, Watine J, Twomey PJ, Oosterhuis WP, et al. How Useful are Laboratory Practice Guidelines? EJIFCC 2015; 26(3): 190–196.
27. Flink L, Underberg JA, Newman JD, Gianos E. The recent national lipid association recommendations: how do they compare to other established dyslipidemia guidelines? Curr Atheroscler Rep. 2015; 17(4): 494. doi: 10.1007/s11883-015-0494-9
28. Sidhu D, Naugler C. Fasting time and lipid levels in a community-based population: a cross-sectional study. Arch Intern Med. 2012; 172(22): 1707–1710.
29. Nordestgaard BG, Langsted A, Mora S, Kolovou G, Baum H, et al. Fasting is not routinely required for determination of a lipid profile: clinical and laboratory implications including flagging at desirable concentration cut-points-a joint consensus statement from the European Atherosclerosis Society and European Federation of Clinical Chemistry and Laboratory Medicine. Eur Heart J. 2016; 37(25): 1944–1958. doi: 10.1093/eurheartj/ehw152
30. Bravo E, Napolitano M, Botham KM. Postprandial Lipid Metabolism: The Missing Link Between Life-Style Habits and the Increasing Incidence of Metabolic Diseases in Western Countries? The Open Translational Medicine Journal. 2010; 2: 1–13.
31. Cybulska B, Szostak WB, Podolec P, Kopeć G, Naruszewic M, Undas A, et al. Polish Forum for Prevention Guidelines on Dyslipidaemia. Kardiol Pol. 2008; 66(11): 1239–1242.
32. Lillo R, Salinas M, Lopez-Garrigos M, Naranjo-Santana Y, Gutiérrez M, Marín MD, et al. Reducing preanalytical laboratory sample errors through educational and technological interventions. Clin Lab. 2012; 58(9–10): 911–917.
33. Plebani M. Towards a new paradigm in laboratory medicine: the five rights. Clin Chem Lab Med. 2016; 54(12): 1881–1891. doi: 10.1515/cclm-2016-0848
34. Hata Y, Nakajima K. Life-style and serum lipids and lipoproteins. J Atheroscler Thromb. 2000; 7(4): 177–197.
35. Jaksz-Recmanik E, Bobiński R. Pre-analytical errors in nurse practice. Probl Pielęg. 2011; 19(3): 386–390.
36. Stańda-Nowakowska M, Zurzycka P, Repka I. Assessment of knowledge of patients responding to laboratory tests – preliminary results. Pielęg XXI w. 2014; 47(2): 11–15.
37. Cramb R, French J, Mackness M, Neely RD, Caslake M, MacKenzie F. Lipid external quality assessment: commutability between external quality assessment and clinical specimens. Ann Clin Biochem. 2008; 45(Pt 3): 260–265. doi: 10.1258/acb.2007.007120
38. Fatati G. Sustainable diet: history lessons. Recenti Prog Med. 2015; 106(11): 540–544. doi: 10.1701/2074.22486
39. Thiengwiboonwong S, Chongsuwat R, Temcharoen P, Pandii W, Pavadhgul P. Efficacy of dietary modification following the National Cholesterol Education Program (NCEP) recommendation on lipid profiles among hyperlipidemia subjects. J Med Assoc Thai. 2013; 96(10): 1257–1267.
40. Thompson RL, Summerbell CD, Hooper L, Higgins JPT, Little P, Talbot D, et al. Dietary advice given by a dietitian versus other health professional or self-help resources to reduce blood cholesterol. Cochrane Database Syst Rev. 2003; 3: CD001366.
41. Reid R, Fodor G, Lydon-Hassen K, McCrea J, Bowlby M, Difrancesco L, et al. Dietary counselling for dyslipidemia in primary care: results of a randomized trial. Can J Diet Pract Res. 2002; 63(4): 169–175.
42. Berry EM, Dernini S, Burlingame B, Meybeck A. Food security and sustainability: can one exist without the other? Public Health Nutr. 2015; 18(13): 2293–2302. doi: 10.1017/S136898001500021X. Epub 2015 Feb 16
43. Dudzińska M, Neć M, Zwolak A, Oszywa-Chabros A, Malicka J, Smoleń A, et al. The role of the primary care outpatient clinic in the promotion of healthy nutrition – preliminary reports. Fam Med Primary Care Rev. 2016; 18(3): 230–234.
44. Imanaka M, Ando M, Kitamura T, Kawamura T. Impact of Registered Dietitian Expertise in Health Guidance for Weight Loss. PLoS One 2016; 11(3): e0151456. doi: 10.1371/journal.pone.0151456. eCollection 2016
45. Nazarewicz R. The effect of vegetarian diet on selected biochemical and blood morphology parameters. Rocz Panstw Zakl Hig. 2007; 58(1): 23–27.
46. Sicińska P, Pytel E, Maćczak A, Koter-Michalak M. The use of various diet supplements in metabolic syndrome. Postepy Hig Med Dosw. 2015; 69: 25–33.
47. Winiarska-Mieczan A, Mazurek K. Comparison of the nutritional value of traditional, semivegan and vegan diets. Żywienie Człowieka i Metabolizm. 2005; 3: 203–213.
48. Bałasińska B, Mazur A. Oxidized dietary lipids may participate in the development of atherosclerosis. Postepy Hig Med Dosw. 2004; 58: 176–182.
49. Hodson L, Skeaff CM, Chisholm WA. The effect of replacing dietary saturated fat with polyunsaturated or monounsaturated fat on plasma lipids in free-living young adults. Eur J Clin Nutr. 2001; 55(10): 908–915.
50. Li Y, Hruby A, Bernstein AM, Ley SH, Wang DD, Chiuve SE, et al. Saturated Fats Compared With Unsaturated Fats and Sources of Carbohydrates in Relation to Risk of Coronary Heart Disease: A Prospective Cohort Study. J Am Coll Cardiol. 2015; 66(14): 1538–1548. doi: 10.1016/j.jacc.2015.07.055
51. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am J Clin Nutr. 2003; 77(5): 1146–1155.
52. Nowicka G, Panczenko-Kresowska G. Hypolipidemic action of protein of legume seeds. Żywienie Człowieka i Metabolizm. 2005; 32: 47–53.
53. Reynolds K, Chin A, Lees KA, Nguyen A, Bujnowski D, He J. A meta-analysis of the effect of soy protein supplementation on serum lipids. Am J Cardiol. 2006; 98(5): 633–640.
54. Donini LM, Dernini S, Lairon D, Serra-Majem L, Amiot MJ, Del Balzo V, et al. A Consensus Proposal for Nutritional Indicators to Assess the Sustainability of a Healthy Diet: The Mediterranean Diet as a Case Study. Front Nutr. 2016; 3: 37. doi: 10.3389/fnut.2016.00037. eCollection 2016
55. Wu T, Fu J, Yang Y, Zhang L, Han J. The effects of phytosterols/stanols on blood lipid profiles: a systematic review with meta-analysis. Asia Pac J Clin Nutr. 2009; 18(2): 179–186.
56. Mitjavila MT, Fandos M, Salas-Salvadó J, Covas MI, Borrego S, Estruch R, et al. The Mediterranean diet improves the systemic lipid and DNA oxidative damage in metabolic syndrome individuals. A randomized, controlled, trial. Clin Nutr. 2013; 32(2): 172–178. doi: 10.1016/j.clnu.2012.08.002
57. Richard C, Couture P, Desroches S, Charest A, Lamarche B. Effect of the Mediterranean diet with and without weight loss on cardiovascular risk factors in men with the metabolic syndrome. Nutr Metab Cardiovasc Dis. 2011; 21(9): 628–635. doi: 10.1016/j.numecd.2010.01.012
58. Park YM, Steck SE, Fung TT, Zhang J, Hazlett LJ, Han K, et al. Mediterranean diet, Dietary Approaches to Stop Hypertension (DASH) style diet, and metabolic health in U.S. adults. Clin Nutr. 2017; 36(5): 1301–1309. doi: 10.1016/j.clnu.2016.08.018
59. Martínez-González MA, Salas-Salvadó J, Estruch R, Corella D, Fitó M, Ros E, et al. Benefits of the Mediterranean Diet: Insights From the PREDIMED Study. Prog Cardiovasc Dis. 2015; 58(1): 50–60. doi: 10.1016/j.pcad.2015.04.003
60. Ros E. The PREDIMED study. Endocrinol Diabetes Nutr. 2017; 64(2): 63–66. doi: 10.1016/j.endinu.2016.11.003
61. Dashti HM, Al-Zaid NS, Mathew TC, Al-Mousawi M, Talib H, Asfar SK, et al. Long term effects of ketogenic diet in obese subjects with high cholesterol level. Mol Cell Biochem. 2006; 286(1–2): 1–9.
62. Wolfe BM, Piché LA. Replacement of carbohydrate by protein in a conventional-fat diet reduces cholesterol and triglyceride concentrations in healthy normolipidemic subjects. Clin Invest Med. 1999; 22(4): 140–148.
63. Archer WR, Lamarche B, St-Pierre AC, Mauger JF, Deriaz O, Landry N, et al. High carbohydrate and high monounsaturated fatty acid diets similarly affect LDL electrophoretic characteristics in men who are losing weight. J Nutr. 2003; 133(10): 3124–3129.
64. Grieb P, Kłapcińska B, Smól E, Pilis T, Pilis W, Sadowska-Krepa E, et al. Long-term consumption of a carbohydrate-restricted diet does not induce deleterious metabolic effects. Nutr Res. 2008; 28(12): 825–833. doi: 10.1016/j.nutres.2008.09.011
65. Harman NL, Leeds AR, Griffin BA. Increased dietary cholesterol does not increase plasma low density lipoprotein when accompanied by an energy-restricted diet and weight loss. Eur J Nutr. 2008; 47(6): 287–293. doi: 10.1007/s00394-008-0730-y.

66. Dattilo AM, Kris-Etherton PM. Effects of weight reduction on blood lipids and lipoproteins: a meta-analysis. Am J Clin Nutr. 1992; 56(2): 320–328.
67. Matthew J, Sharman MJ, Kraemer WJ, Love DM, Avery NG, Gómez AL, et al. A ketogenic diet favourably affects serum biomarkers for cardiovascular disease in normal-weight men. J Nutr. 2002; 132(7): 1879–1885.
68. Bolesławska I, Przysławski J, Szczepanik A, Chuchracki M, Żwirska J. Lipid profile and parameters of oxidative stress in women and men using/applaying the optimal model of nutrition. Bromat Chem Toksykol. 2010; 3: 276–280.
69. Winiarska-Mleczan A, Pyzik D. Evaluation of the nutritional value of menus, high-fat diet "optimal". Żyw Człow Matab. 2006; 33: 73–82.
70. Ciborowska H, Rudnicka A. Dietetics – Nutrition of healthy and sick individuals. Wyd. PZWL, Warszawa, 2014.
71. Pesta DH, Samuel VT. A high-protein diet for reducing body fat: mechanisms and possible caveats. Nutr Metab. 2014; 11(1): 53. doi: 10.1186/1743-7075-11-53. eCollection 2014
72. Brar P, Kwon GY, Holleran S, Bai D, Tall AR, Ramakrishnan R, et al. Change in lipid profile in celiac disease: beneficial effect of gluten-free diet. Am J Med. 2006; 119(9): 786–790.
73. Dittfeld A, Gwizdek K, Parol D, Michalski M. Glutenfree diet: Characteristics of target groups. Postepy Hig Med Dosw. 2018; 72: 227–239.
74. Jenkins DJ, Kendall CW, Vidgen E, Augustin LS, van Erk M, Geelen A, et al. High-protein diets in hyperlipidemia, effect of wheat gluten on serum lipids, uric acid and renal function. Am J Clin Nutr. 2001; 74(1): 57–63.
75. Antonio J, Ellerbroek A, Silver T, Vargas L, Tamayo A, Buehn R, et al. A High Protein Diet Has No Harmful Effects: A One-Year Crossover Study in Resistance-Trained Males. J Nutr Metab. 2016; 2016: 9104792.
76. Schwingshackl L, Hoffmann G. Comparison of high vs. normal/low protein diets on renal function in subjects without chronic kidney disease: a systematic review and meta-analysis. PLoS One 2014; 9(5): e97656. doi: 10.1371/journal.pone.0097656. eCollection 2014
77. Theuwissen E, Mensink R. Water-soluble dietary fibers and cardiovascular disease. Physiol Behav. 2008; 94(2): 285–292. doi: 10.1016/j.physbeh.2008.01.001
78. Veldhorst M, Smeets A, Soenen S, Hochstenbach-Waelen A, Hursel R, Diepvens K, et al. Protein-induced satiety: effects and mechanisms of different proteins. Physiol Behav. 2008; 94(2): 300–307. doi: 10.1016/j.physbeh.2008.01.003
79. Anderson JW, Allgood LD, Turner J, Oeltgen PR, Daggy BP. Effects of psyllium on glucose and serum lipid responses in men with type 2 diabetes and hypercholesterolemia. Am J Clin Nutr. 1999; 70(4): 466–473.
80. Brown L, Rosner B, Sacks FM. Cholesterol-lowering effects of dietary fiber: a meta-analysis. Am J Clin Nutr. 1999; 69(1): 30–42.
81. Wu H, Dwyer KM, Fan Z, Shircore A, Fan J, Dwyer JH. Dietary fiber and progression of atherosclerosis: The Los Angeles Atherosclerosis Study. Am J Clin Nutr. 2003; 78(6): 1085–1091.
82. Mikołajczak J, Bator E, Bronkowska M, Piotrowska E, Orzeł D, Wyka J, et al. The value of the glycemic indexes and loads of selected cereals consumed with milk. Rocz Panstw Zakl Hig. 2012; 63(4): 433–440.
83. Ciok J, Dolna A. The glycemic index, and lipid disorders. Żyw Człow Metab. 2005; 32: 38–45.
84. Dumesnil JG, Turgeon J, Tremblay A, Poirier P, Gilbert M, Gagnon L, et al. Effect of a low-glycaemic index-low-fat-high protein diet on the atherogenic metabolic risk profile of abdominally obese men. Br J Nutr. 2001; 86(5): 557–568.
85. Denova-Gitierrez E, Huitron-Bravo G, Talavera JO, Castañón S, Gallegos-Carrillo K, Flores Y, et al. Dietary glycemic index, dietary glycemic load, blood lipids and coronary heart disease. J Nutr Metab. 2010; 2010: 170680. doi: 10.1155/2010/170680
86. Fried SK, Frao SP. Sugars, hypertriglyceridemia and cardiovascular disease. Am J Clin Nutr. 2003; 78(4): 873S-880S.
87. Venn BJ, Green TJ. Glycemic index and glycemic load: measurement issues and their effect on diet-disease relationships. Eur J Clin Nutr. 2007; 61: S122–S131.
88. Adamska E, Górska M. Index and glycemic load. Przegl Kardiodiabetol. 2008; 3: 223–231.
89. Shikany JM, Phadke RP, Redden DT, Gower BA. Effects of low- and high-glycemic index/glycemic load diets on coronary heart disease risk factors in overweight/obese men. Metabolism 2009; 58(12): 1793–1801. doi: 10.1016/j.metabol.2009.06.006
90. Slyper A, Jurva J, Pleuss J, Hoffmann R, Gutterman D. Influence of glycemic load on HDL cholesterol in youth. Am J Clin Nutr. 2005; 81(2): 376–379.
91. The rules of proper nutrition. http://www.izz.waw.pl/pl?/option=comcontent-&view=article&id=7. (access: 2019.04.17)
92. Całyniuk B, Grochowska-Niedworok E, Białek A, Czech N, Kukielczak A. Food guide pyramid – its past and present. Probl Hig Epidemiol 2011; 92(1): 20–24.
93. The Nutrition Sources. Harvard T.H.N. School Of Public Health. www.hsph.harvard.edu/nutritionsource/healthy-eating-plate/. (access: 2019.04.17)