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Nutrition and Health Claims

The responsibility of nutrition claims in the achievement of a balanced diet and background material on data establishing nutrition and health claims.

Briefing Note

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EXECUTIVE SUMMARY

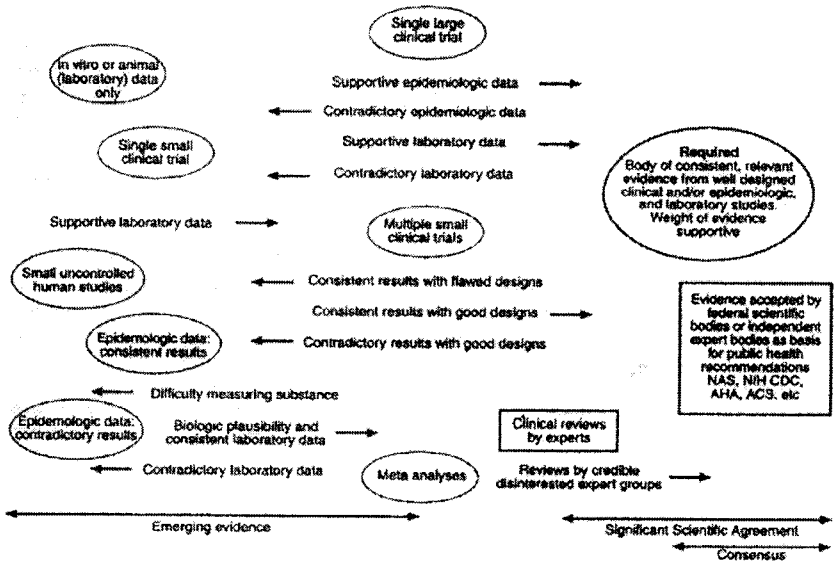
The spreading of health and nutrition claims is one of results of the population's increasing knowledge about the association between food and health: people require more information on the biological effects of food; they want help in choosing from a tremendous range of foods. No doubt, health claims are used also for marketing reasons. From the consumer's point of view, claims need to be examined to prevent customers being misinformed and to promote healthy eating to achieve a balanced diet. Regulation and authorisation are needed to prevent misleading claims.

Health and nutrition claims should inform the consumers in layman's language - simply and understandably. The emphasis should be on improving eating habits. The benefits claimed must be clear to everyone and consistency is essential. It is useful to examine the background of claims to discover what they mean. Claims linking food ingredients to health require sound scientific evidence and significant scientific agreement. An increasing amount of research now tends to support the claim that eating certain foods and their physiologically active components may reduce the risk of disease. The majority of these ingredients are obtained from plants but there are several biologically active substances of animal origin.

The US Food and Drug Administration (FDA) released the criteria for assessing the strength and consistency of scientific evidence in 1999. There is a discrepancy between "emerging evidence" (characterized by in vitro or animal experiments, uncontrolled human studies, and inconsistent epidemiological evidence) and "significant scientific agreement." To reach such an agreement, a string of conditions are required: support of consistent, relevant evidence from well-designed epidemiologic, clinical and animal studies and statement of scientific rank-and-file opinion.

The schema for assessing the strength of evidence is shown below (4).

Schema for Assessing Strength and Consistency of Scientific Evidence Leading to Significant Scientific Agreement



In this briefing note, we present the level of scientific evidence concerning the nutrition (nutrient content) claims on the basis of references.

The lower energy uptake is basically involved in the prevention of overweight/obesity, thus in the avoidance of overweight/obesity-related disorders such as cardiovascular diseases (CVD), certain types of cancer, type 2 diabetes mellitus, ill health of locomotorium etc. A lower fat intake - above all saturated fatty acids (SFA) - contributes to the prevention of overweight/obesity, CVD and certain types of cancer. Excess sugar and high gycemic index (GI) may promote overweight/obesity and increase serum triacylglycerol (TAG). Sugar is strongly associated with caries. Therefore the reduction of high sugar intake is reasonable.

Dietary fibre enhances bowel motility, controls the absorption of carbohydrates and fat and, participates in the prevention of colon cancer. A higher dietary fibre intake is advisable. The decrease of salt/sodium intake appears beneficial for the prevention (and treatment) of hypertension. The advantage of a reasonable vitamin, macro- and microelement intake in the prophylaxis of chronic non-communicable diseases is widely accepted.

Nutrition (nutrient content) claims concerning the above-mentioned issues are supported by "significant scientific agreement", moreover by "consensus", although there are some controversial opinions, not all studies or scientists can agree on all issues. Interestingly,: higher SFA intake in postmenopausal women with low fat intake (~25%en) is associated with slower progression of coronary atherosclerosis, but carbohydrates of high GI facilitate the progression. Polyunsaturated fatty acids (PUFAs) produce similar effects as carbohydrates when they replace another fat but when they replace a carbohydrate or a protein [155, 156]. As for claims regarding protein content, though a scientific consensus exists on the significance of protein, it may be advisable to reconsider the necessity and/or formulation of these claims.

One finds a very different situation in the domain of other biologically active non-nutritive substances. Results are often inconsistent. As for the conclusions regarding their healthy effects, scientific evidence is at the level of "emerging evidence" at the very most; therefore in each case, the dossier of the advantageous biological effects must be considered individually.

Nutrition claims influence eating behaviour. A prerequisite is consumer knowledge about the nutritional fact. When consumers are well-educated and well-informed, the effect of nutrition claims is more intensive. The nutrition claim expediently appears on packaging, eventually in advertisements to influence food choice more effectively ("exposition" factor). The nutrition claim must arouse the consumer's interest or it will be ignored.

The nutrition claim informs the consumers and is an effective tool to influence the food choice facilitating the implementation of a balanced diet. However, it should be emphasized that the nutrition claim is only one factor in the complex system determining the parameters of the successful adoption of a balanced diet, though there is no doubt of its significance. The nutrition claim may change the determinants of nutritional behaviour (attitude, social influence and self-efficacy) [157]. Claims could lead to a change in attitudes by anticipating consumers' considerations when buying or consuming products. The effect of nutrition claims on attitude could be increased by developing information interventions. Nutrition claim efficiency and nutritional education go hand in hand.

CONTENTS

1. TERMS USED IN THE FIELD OF NUTRITION AND HEALTH CLAIMS

To speak a common language, it should be determined the crucial terms. One of the suitable definitions is the following [1]:

Health claim: any representation that states, suggests, or implies that a relationship exists between a food, nutrient, or other substances in a food and a disease or health-related condition.

Nutrition claim: any representation that states, suggests, or implies that a food has particular nutritional properties, including but not limited to the energy value; protein, fat, and carbohydrate values; and content of vitamins and minerals.

Nutrient content claim: a nutrition claim that describes the level of a nutrient in a food, such as "High fibre and low in fat".

The background of these claims seems to be the awareness that the ingredients of foods, i.e. nutrients and biologically active non-nutritive components are in a very strong correlation with the health status, physiologic and biologic well-being and ill health of humans, respectively. It has become clear that food can provide more than just energy and nutrients. Through its capacity to contribute to and control many metabolic, physiological, psychological functions, moreover gene expression, food has effects beyond what is generally accepted as nutrition. Food can play an important role in preventing disease, and can help optimise and enhance normal function, thereby improve the quality of life.

2. THE TARGET OF NUTRITION AND HEALTH CLAIMS

The claims are useful tools for

- arousing attention of everyday people to the balanced nutrition,
- providing advices to choose appropriate foods for this purpose,
- giving information on relationship between food and health,
- enhancing the conscious nutrition, the knowledge on function of human organism, on metabolic processes,
- diminishing the nutritional false beliefs,
- performing guidelines to manufacturer.

Although the nutrition and health claims indicate one particular form of nutrition labelling, they should be more strongly controlled, because they have direct health impact, they include food-related advices, in fact not only bare statements of nutrient function or to feel-good effects, but are disease-related, though not quite medicinal [2, 3].

3. SOME PRIOR EVENTS

In the past mainly the health claims were investigated, the conclusions of which provide an excellent background for the nutrition claims, particularly for the nutrient content claims. There are many legislative measures chiefly regarding the functional foods. On Japan the issues of "foods for specific health uses" (FOSHUs) have been regulated, Sweden and the Netherlands have codes of practice for health claims. In Australia and New Zealand the authorities reviewed the policy on health claims. In the United Kingdom the Food Advisory Committee dealt with health claims and functional food ([2]). In the United States the Nutrition Labelling and Education Act of 1990 (NLEA) and the Food and Drug Administration Modernization Act of 1997 regulate the health claims, and there is health claim (i.e. declaration of health-disease relationship) approved by FDA on the basis of NLEA, and FDA Modernization Act, as well as following petitions submitted by the food industry. Altogether 15 items present correlation between food/food component and disease [(4).]

In the last period a European Commission funded concerted action, the Process for the Assessment of Scientific Support for Claims on Foods (PASSCLAIM) [(5, 6)] set the establishing criteria for the scientific support of health claims as an aim. It was recommended that substantiation of the strength of a claim should follow an evidence-based decision-making process and that claims should be re-evaluated every five to six years. At the present time most codes of practice for validating health claims for food are evolving voluntarily. The need for uniform definitions of the types of claims was stressed. The scientists worked in PASSCLAIM studies investigated a large number of issues (e.g. diet-related cardiovascular diseases, bone health and osteoporosis, body weight regulation, diet-related cancer etc.).

4. METHODOLOGY FOR SCIENTIFIC SUBSTANTIATION OF NUTRITION AND HEALTH CLAIMS

The claims proposed in the motion to European Parliament belong to the group of "Nutrition claim", or rather "Nutrient content claim". However, the scientific substantiation of different claim types is similar, unless identical.

For evaluation of nutrition, for seeking the human biological consequences of food consumed, one should be acquainted partly with the health status of the investigated population, partly with the food consumption. The sources of information include: morbidity data (diet-related diseases) from notification of diseases, general practitioner statistics, hospital in- and out-patient statistics, mortality data, post-mortem statistics (autopsy); food consumption (food disappearance, food availability, food production, food balance sheets, households statistics). The direct data come from ***nutritional epidemiological studies***. The main types of them are as follows [7, 8, 9]:

4.1. Observational studies

- Ecological (correlation) studies: to detect by means of information mentioned above the correlation between nutrition and biological sequels.
- Cross-sectional (prevalence) studies: momentary features of public nutrition, chiefly based on 24-hour recall(s) or record(s)
- Case-control studies have retrospective character and analyze the earlier nutrition of patients suffering from a verified or suspect diet-related disease.
- Cohort (following) studies are prospective ones and compare the biomarkers and the nutrition of subjects who are exposed to given nutritional factors with those who are not exposed. As reference value serve the nutritional characteristics at the base-line of investigation.
- In the case of nutritional surveillance (monitoring) the data of morbidity and mortality, as well as nutritional risk factors provide information on the surveyed population. The nutritional behaviour and the level of knowledge may be included, together with 24-hour recall(s) and food frequency sheets.

4.2. Experimental studies

- Nutritional intervention starts with establishing of basic nutritional values and continues with targeted nutritional changes, finally closes with the registration of dietary transformation and biological consequences.
- In a random, controlled clinical trial the dosage of pharmaceutical products makes the nutritional intervention complete.

We have to mention the importance of meta-analysis in nutritional epidemiology. This method provides often a comprehensive overview and very valuable, new considerations.

4.3. Assessment of dietary peculiarities:

- 24-hour recall informs on exact, qualitative and quantitative food intake during the previous 24-hour period on preceding day(s), estimated in household measures. If habitual intakes of subjects are required, multiple replicate 24-hour recall must be used (at the very least one working day and one weekend day). Nutrient intake is calculated with the aid of food composition tables.
- Estimated food record: it means to record qualitatively and quantitatively all food and beverages as consumed over periods from one to seven days (in practice most frequently three days). Other procedures like 24-hour recall.
- Weighed food record: The method is identical with the estimated food record but all food is weighed. The nutrient content may be calculated from food composition tables or analytically measured from a parallel sample.
- Food frequency questionnaire: it is designed to obtain qualitative data on usual intakes of foods or group of foods. It is based on a comprehensive list of 50-150 or more food items. The questionnaire can be semi-quantitative when the investigated subjects quantify the usual portion sizes.
- Dietary history: a combined method with 24-hour recall, semi-quantitative food frequency questionnaire and information on usual eating pattern.

We should emphasize the results of European Commission funded European Food Consumption Survey Method (EFCOSUM). Since the 24-hour recall method is applicable in large European population, has relatively low respondent and interviewer burden, is open-ended and cost-effective, this method can be considered as the best method for EFCOSUM to get population mean intakes and distributions for subjects aged 10 years and over. Usual intake should be estimated by statistical modelling techniques, using two non-consecutive 24-hour recalls and a food list to assess the proportion non-users for infrequently consumed foods [(10)]

It should be emphasized that the **post-market control** must be systematically carried out in the case of each claim regarding the physiological and behavioural consequences.

5. SCIENTIFIC SUBSTANTIATION OF NUTRITION CLAIMS (NUTRIENT CONTENT), THE SIGNIFICANCE OF FOODS WITH NUTRITION CLAIMS IN THE HEALTH PROMOTION

5.1. Low Energy, Energy Reduced, Energy-Free

One of the most common problems of European health services today is overweight or obesity, which is a key risk factor in the development of many chronic diseases, such as cardiovascular (including hypertension) and respiratory diseases, type 2 diabetes, certain malignant tumours [(11)]. Already in 1997, the WHO Expert Consultation on Obesity warned the global community of an escalating epidemic of obesity. The experts pointed out that a concerted action is needed to stem the pandemic. Millions of people in both developed and developing countries would be at risk of developing chronic non-communicable diseases and other diet-related health disorders [(12)].

Many factors are included in the aetiology of the overweight and obesity: hereditary, nutritional, environmental and behavioural factors, likewise the physical activity. Undoubtedly, the obesity is not always a result of overeating or of lack of physical activity, a number of biological factors such as stress, drugs and ageing also play role in its development. However, the dietary patterns strongly influence the energy balance in the organism; moreover this major modifiable factor is relatively easily influenced.

When the energy intake decreases, as a rule, the body mass gain stops, unless goes down because energy intake is less than energy expenditure. An increased physical activity enhances the weight loss. High-fat, energy-dense diet and sedentary lifestyle worldwide are most strongly associated with increased prevalence of obesity.

The experts of WHO have found convincing evidence that high intake of energy-dense food promotes weight gain [(13)]. In contrast to that, low energy-dense diets are associated with lower energy intake and body mass. Dietary energy density can be lowered by adding water, cooked grains, and soups to the diet, and by reducing fat content [(14)]. The European diet seems to be increasingly energy rich but micronutrient poor. To improve nutrient-to-energy ratio, it is recommended that consumer replace some foods with more less nutrient-dense options. The FDA has considered approving nutrient claims based on the ratio of a beneficial nutrient to the food's energy content, as opposed to a specified minimum amount of a nutrient per serving size [(15)].

Most commonly used interventions for prevention or treatment of obesity are low-calorie diets that modify the macronutrient composition of diet. The low-calorie diet requires on the one hand portion size control, on the other to lessen energy density, to cut the total energy content of foods below the usual energy expenditure. However, the diet must be balanced and cover the nutrient needs [(16, 17)]. In the aetiology of obesity the diet plays a role pointed-up [(18)]. In the prevention of obesity both the diet and physical activity are of vital importance [(19)].

The consumption of sugar sweetened soft drinks is associated with childhood obesity, each drink means in average an increase of Body Mass Index (BMI)[1] by 0.18 kg/m^2 (95% CI 0.09-

[1] Body mass in kg divided by the square of body height in m.

0.27, p=0.02) [20]. High-fat diets promote development of obesity in several species of laboratory animals. Many nutritionists accept that high-fat diets predispose human population to obesity. These observations in experimental animals and human beings provide basis for the widely advocated use of low-fat diets for general public, because obesity rarely occurs when the diet contains very little fat. However, regarding the obesity, one should discriminate between high percentage of fat relative to other macronutrients (in a diet that otherwise contains a desirable amount of total energy) and a diet containing more nutrient energy than needed to maintain desirable weight and in which fat contributes to the excess. The last one looks to be hazardous as far as development of obesity is concerned [21, 22, 23].

There are data that by 68% more energy is needed for the development of 1 kg fat store from carbohydrates than from fat, because the thermogenetic effect of carbohydrates is much higher. Other researcher reported that there can be considerable lipogenesis from carbohydrate in humans during isoenergetic feeding, the energy cost of conversion must be small [24, 25]. Also the fatty acid pattern influences the human adiposity. High fat intake (excessive energy intake from fat) results in obesity and the predictors for the obesity markers are the saturated and monounsaturated fatty acids, by contrast the polyunsaturated fatty acids don't have such an effect [26].

Another macronutrient, the protein takes part in the body mass regulation. The protein content of food is a determinant of short-term satiety and of how much food is eaten. The increased satiety contributes to the ease of acceptance or adherence to a reduced energy diet and to reduction in energy intake [27].

As for carbohydrates, the low glycaemic index facilitates the weight loss; the high glycaemic index does the weight gain [28].

Conclusions:

The energy intake over the energy expenditure is a critical factor in the development of obesity. Thus the lower energy content in certain foods and mainly in the entire diet contributes to the prevention of weight gain and to the treatment of obesity. Besides this the amount of food and the eating frequency are important. Therefore the risk of obesity may decrease, its sequels are avoidable. The weight loss contributes to the improvement of structural heart disorders, of the cardial function. At the same time the health status of hypertensive patients and of people suffering in type 2 diabetes gets better [29]. Several other possible factors may appear in aetiology of overweight and obesity: cessation of smoking, treatment with steroids, some psychotropic drugs, hormone replacement, and oral contraceptive preparations. The unfavourable sequels of them for the most part may be avoided by a prudent diet with low energy content.

The concise summary of obesity's consequences is shown in the box below [30].

Consequences of obesity	
Metabolic Impaired glucose tolerance Non-insulin dependent diabetes Dyslipidaemia Metabolic syndrome Fatty liver Gallstones Infertility in women	*Cardiovascular* Hypertension Coronary heart disease Varicose veins Peripheral oedema
Mechanical Osteoarthritis Spinal problems Obstructive sleep apnoea	*Social* Low self-esteem Adverse judgement by society

The selection of low-energy foods is one step only in an anti-obesity program, which should be launched with healthful dietary practices to children and continued lifelong [31].

The nutrition (nutrient content) claims concerning energy content of foods are supported by commonly accepted scientific evidences.

5.2. Low Fat, Fat-Free, Low Saturated Fat, Saturated Fat-Free

The relation between dietary fats (lipids) and cardiovascular disease (CVD), the "diet-heart" or "fat-atherosclerosis" hypothesis has been in the centre of strategies for risk reduction in individuals and population. There is a tremendous number of references containing scientific evidences on human biological effects of fats, on different physiological consequences of different fatty acids. We have to strive for putting together an explicit compilation containing the main lines, which aims at substantiating (or deny) nutrition claims presented above.

Dietary fats provide for the body energy, carry vital nutrients. Fats also play an important role in food manufacture and cooking.

Fats in foods have different origins: the main sources of fats of animal origin in Europe are meat, meat product, milk and dairy products, eggs; the fats of vegetable origin can be found in plant seeds (sunflower, rapeseed, maize etc.), fruits (olive, avocado etc.), and nuts (peanuts, almond etc.). Edible oil is obtained from these plant constituents by appropriate procedures. Fat is a heterogeneous mixture of lipids. The dietary and the human body fats are mostly in form of triglycerides (triacylglycerols, TGs, TAGs), which is built up from a molecule of glycerol (shaped like a three prongs fork) and three fatty acids molecules (connected to the prongs).

All fats are made up of a combination of saturated, monounsaturated and polyunsaturated fatty acids (SFA, MUFA, PUFA). The polyunsaturated fatty acids (PUFAs) are categorised into two families according to the position of the first double bond (n–3 and n–6 fatty acid family). The molecules of unsaturated fatty acids belongs mostly to the "cis" group (bent form), more rarely to the "trans" group (straight form). There are in the foods and the human body also phospholipids, glycolipids, waxes, fat-like substances (lipoids): cholesterol, other sterols.

From a meta-analysis of 27 studies (randomised controlled trials to reduce or modify fat or cholesterol intake in healthy adults over at least six months) and 30902 person years of observation, it was shown that a reduction of dietary fat intake had small effect on total mortality (rate ratio 0.98; 95% CI 0.86-1.12). The cardiovascular mortality diminished by 9%, the cardiovascular events by 16%. Trials with at least two years' follow-up provided stronger evidence concerning the CVD events [32].

The TAGs from fats of animal origin contain a high proportion of saturated fatty acids; the dominant fatty acids in fat of vegetable origin are unsaturated. To the n–6 family belonging linoleic acid (LA) and to the n–3 family belonging alpha-linolenic acid (ALA) cannot be synthesised in the body. They are necessary for several biological processes; this is why they are essential fatty acids [7, 33].

The marked serum cholesterol-increasing effect of SFAs has been known for a long time. The SFA intake has a far greater effect on blood cholesterol level than the amount of dietary cholesterol. However, there are several evidences that only lauric acid, myristic acid and palmitic acid (medium-chain fatty acids) significantly influence total and LDL-C (low-density lipoprotein cholesterol), namely to a decreasing degree in order of list. These fatty acids decrease the cellular LDL uptake. The short- and long-chain fatty acids (including stearic acid), also the fatty acids with longer chain length considerably don't increase the cholesterol level. Palmitic and stearic acid are neutral in absence of cholesterol only but in the European diet the main sources of these fatty acids are milk-, beef- and chicken-fat, which contain also cholesterol and so the neutrality cannot become effective. A high concentration of LDL-C is a risk factor for coronary heart disease (CHD). In contrary, the HDL-C (high-density lipoprotein cholesterol) removes from tissues the deposited cholesterol and its high concentration is beneficial; the higher HDL-C level lowers the risk of CHD.

The results that certain SFAs are neutral and don't influence the blood cholesterol or the development of CHD looks to be relevant to a small extent for practical dietary recommendations because the foods contain a mixture of fatty acids and also SFAs with different chain length. In addition, the majority of our fatty acid intake comes from SFAs increasing blood cholesterol. A differentiated recommendation is impossible but we have epidemiological evidence supporting the theory that a positive association exists between SFA uptake and CVD [34].

Fatty acids play role not only in prevention of CVDs via their effect on serum lipids but because they directly influence several other risk factors and have effect on atherogenesis. It is still mostly recommended not to exceed a total daily fat intake of 30%en [35, 36]. The beneficial or detrimental effects of dietary fats on people's health largely depend on their fatty acid composition. There is a common consensus to limit the SFAs' intake, to reduce the ratio of SFAs to below 10% of total daily energy supply [37].

Trans-fatty acids (tFAs) are metabolised in similar way to SFAs. They are unsaturated fatty acids with particular chemical configuration and show the same positive association with CVD risk as SFAs. The tFAs have negative impact on the biomarkers indicating the increase of CHD risk: higher LDL-C level, lower HDL-C level, worse LDL-C/HDL-C ratio, higher TAG and Lp(a)[2] levels. The natural sources of tFAs are food products from ruminants. Earlier the margarine belonged to major sources of tFAs but it is been forgotten by now because the manufacturing processes have been modified. However the hardened fat with tFA content plays role in the production of pastries, chips as well as fried and convenience food [35, 38, 39, 40]. From human biological point of view, the tFAs should be taken into account together with SFAs.

The results of trials are sometimes inconclusive. In a Finnish cohort of 21930 men who were initially free of diagnosed CVD after more than 6 years of follow up there was no association between intakes of SFAs, cis-MUFAs, LA or ALA, dietary cholesterol and the risk of fatal CHD. On the other hand, the investigators observed a significant positive correlation between the tFA intake and the risk of CHD death [41].

Unsaturated fatty acids possess favourable effects on blood lipids and in the prevention of CVD. We wish to emphasize the importance of n–3 PUFAs. Dietary ALA is associated with reduced risk of fatal CHD but may enhance the prostate cancer risk. In the case of higher ALA intake the combined relative risk fell to 0.79 and the risk of prostate cancer increased to 1.70 according to meta-analysis of 5 prospective cohort studies, 3 clinical trials (CVD), also 9 cohort and case-control studies (prostate cancer) [42]. Epidemiological studies and dietary trials in humans suggest that ALA is a major cardio-protective nutrient [43]. Long chain n–3 PUFAs found in fatty fish and in fish oils might act to stabilize advanced atherosclerotic plaques maybe through their anti-inflammatory effects [44]. Recommended ALA intake 2.2 g/d (US Expert Committee) or 2.4 g/d (British Nutrition Foundation), recommended very long-chain n–3 fatty acid intake (eicosapentaenoic and docosahexaenoic acid, EPA and DHA) 0.65g/d or 1.2 g/d [45]. Very long-chain n–3 fatty acids (and maybe ALA) suggest evidence in exerting advantageous influence for both CVD prevention and post-infarction treatment [46, 47, 48]. The efficacy of LA and ALA is synergetic regarding the prophylaxis of CHD [49].

The PUFAs are insignificantly, inversely associated with CHD in both genders. By 5% higher dietary uptake of SFAs increases the prevalence of CHD by 38% in women, but SFAs indicate positive relationship in young members of both genders, still not in older persons [50]. High n–6/n–3 ratio lessens the bone density in both men and women [51]. 1–2 g/d long chain PUFA, together with low uptake of SFA, is favourable for the prevention of type 2 diabetes mellitus [52].

Some researchers didn't demonstrate the CHD risk decreasing effect of LA [53], still abundant seafood consumption containing much EPA and DHA goes hand in hand with low ischaemic heart disease mortality [54, 55]. The combination of fish oil and evening primrose oil shows a higher efficacy in prophylaxis of atherosclerosis. The n–3 fatty acids have an effect also on TAG [56]. Surprisingly in the case of over 800 middle-aged men the fat, SFA, moreover MUFA diminished the risk of ischaemic stroke during 20 years follow-up. However, one may not overestimate the results and give to the population such a recommendation [57, 58]. Fats influence the haemostasis, fatty meals have a procoagulative haemostatic profile but the effect of each fatty acids is inconclusive within a three day period (maybe this duration is too short) [59].

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[2] Lipoprotein(a): chemically modified low-density lipoprotein with high cholesterol content. It increases the risk of CVD.

Conclusions

The high fat diets that characterize most industrialized countries for the past several decades promote certain chronic non-communicable diseases including the group of cardiovascular diseases, some types of cancer, obesity and a number of other disorders. There is overwhelming evidence that the most preferable approach of the issue how to achieve overall reduction of dietary fat intake and to improve the fatty acid pattern on a population basis. The potential for lower-fat diets to reduce the risk of chronic diseases requires consistent choices and consistent use of lower-fat food items [60].

CVD has a multifactorial aetiology and many potential risk markers are known. Cardiovascular epidemiology in the area of nutrition is focused primarily on the type of fat. The hypothesis connecting the risk of atherosclerosis in general, and coronary artery disease in particular with the quantity and quality of dietary fats (SFA, MUFA, PUFA) and with the dietary cholesterol was established many years ago based on different types of evidence [61, 62]:

1. The geographic variations and mortality from CHD associate with estimated fat, and mainly SFA intake on population's level.

2. Changes in fat or SFA intake are associated with definable changes in blood lipids, i.e. in biomarkers of CVD risk factors.

3. Animal experiments demonstrated a real relationship between dietary fats and CVD biomarkers.

4. Human observations produced evidence that similar conditions cause similar changes in humans, too.

The next box contains the general lipoprotein response to major dietary fatty acids in human, relative to oleic acid (cis 18:1) [38].

Fatty acid	**Typical**	**Serum lipid fraction**			
	%en	**VLDL[3]**	**LDL**	**HDL**	**Lp(a)**
SFAs					
12:0+14:0	1-4	Incr.	Incr.	Incr.	Decr.
16:0	6-12	Incr.	Neutr.-incr.	Incr.	Decr.
18:0	2-4	Neutr.	Neutr.	Neutr.	Neutr.
tFAs (t18:1n–9)	2-7	Incr.	Incr.	Decr.	Incr.
MUFAs (c18:1n–9)	10-18	Neutr.	Neutr.	Neutr.	Neutr.
PUFAs (18:2, 18:3)	3-8	Decr.	Incr.	Neutr.-decr.	Neutr.

[3] Very-low-density lipoprotein

Why is advised to reduce the fat intake and to optimize the fatty acid pattern? We summarize the evidence-based causes [38, 63, 64, 65, 66, 67, 68, 69]:

1. High fat intake leads to obesity – as it is revealed in the previous chapter.
2. High fat intake is related to CVD, mainly to coronary artery disease (CAD), therefore it is advised to reduce the total fat intake.
3. Also high SFA intake promotes CVD, therefore it is recommended to reduce SFA intake.
4. MUFAs contribute to the prevention of CVD.
5. PUFAs possess favourable effect concerning CVD.
6. Therefore it is advised to increase the unsaturated fatty acid intake, chiefly from natural sources (e.g. fish, seafood, oleic acid rich vegetable oil). It seems to be particularly important the increase of n–3 PUFA intake because its low intake is – except some regions – rather a common phenomenon and the n–6 to n–3 ratio stands far below the desirable value.
7. It is advised to reduce the intake of unfavourable tFAs.

The nutrition (nutrient content) claims concerning fat content of foods are supported by commonly accepted scientific evidences.

5.3. Low Sugars, Sugars-Free, With no Added Sugars

Carbohydrates form one group of the three major macronutrients supplying the body with energy. The primary structure elements of carbohydrates are sugars. The carbohydrates include simple sugars or monosaccharides (one molecule of sugars), disaccharides (from two molecules), oligosaccharides (from more molecules), starch and non-starch polysaccharides (from >10 up to several thousand molecules), and polyols (sugar alcohols). Mono- or disaccharides ordinarily are named sugars. Glucose, fructose, galactose, mannose belongs to monosaccharides, sucrose (beet or cane sugar, table sugar) and lactose to disaccharides. In foods sugars may be present in natural form or may be added to supplement the flavour; the sugars give sweetness to food. The carbohydrates contribute to the taste, texture and appearance of foods [7, 70].

A diet containing an optimum amount of carbohydrates (≥55%en) may help prevent body fat accumulation. Sugars and starches provide readily accessible fuel for physical activity. Monosaccharides are adsorbed directly from the small intestine, while the disaccharides relative quickly, the starches (structured from glucose molecules) more slowly are broken down by digestive enzymes into simple sugars.

When a food containing carbohydrates is eaten, subsequently a rise and later decrease in blood glucose is shown known as glycaemic response. Several factors influence its rate and duration:

- type of sugar that forms carbohydrate,
- chemical structure of carbohydrate,
- cooking and processing methods used,
- other nutrients in the food,

- the metabolic features of individual,
- the time of day when the food was consumed.

The impact on the glycaemic response is classified according to the glucose standard. This measurement is called the glycaemic index (GI). The sugars have the highest GI. As far as the association between GI and chronic non-communicable diseases, the conclusions of several trials have demonstrated a higher risk of a diet with higher GI. The studies don't provide final evidence in the case of obesity, CHD and some other disorders [71]. The diet of poor obese subjects in the US contains more fat and added sugar; they purchase inexpensive foods of high energy density [72]. Starchy foods containing high levels of slowly digestible carbohydrates produce more limited hyperglycaemic peak than foods with rapidly digested carbohydrates, i.e. with sugar having high GI. The foods with low GI seem to have a protective effect against CVD, and they may have role in weight control, but GI is not in itself sufficient to define the nutritional quality of food, it describe only aspect [72]. In animal experiments, sucrose feeding decreases energy utilization and increases gene expression of uncoupling protein, consequently sucrose may contribute to the regulation of body weight [73]. Furthermore sugars stimulate short-term satiety mechanisms and reduce food intake [74]. Sugars, particularly sucrose and fructose, increase serum TAG concentration up to 60%. The available epidemiologic evidence does not indicate direct association between sugars and CVD but the high TAG concentration signifies a greater risk of CHD above all in women [75]. Too much carbohydrate undoubtedly increases serum TAG but some adaptation exists, so this effect may be moderated [76]. Free sugars promote positive energy balance. Soft drinks rich in free sugar increase energy uptake, which is lesser than consequent compensatory reduction of food intake. On the other hand, the diets with high GI and sucrose result in higher weight gain than a low GI diet, it could be observed a weight increase on the sucrose diet being nearly double that on the high fat or high GI diet and significantly higher than on the low GI diet [77, 78]. Excessive intake of added sugars leads to undesirable health risk of obesity related to type 2 diabetes mellitus and its complications, namely CVD [79]. However, if caloric intake is appropriate, results of animal experiments put forward that feeding a high-sugar diet does not induce obesity in lean rats or enhance weight gain in obese rats, nevertheless TAG increases [80]. Observational studies suggest that diets with high glycaemic load (GI x carbohydrate content) are associated with increased risk of type 2 diabetes and CVD [81]. Other authors highlight the need of further evidence concerning detrimental effects of sugars, notwithstanding they recognize the nutrient-diluting consequence [82, 83]. The fructose elevates the postprandial TAG concentration particularly in men, so its addition to foods is undesirable ([84]).

In middle-aged adults a significant inverse correlation exists between GI and HDL-C level, so the GI seems to be a good predictor for HDL-C ([87], [88]). GI and glycaemic load positively correlate with the risk factors of CVD in rice as staple food eating Japanese women (89). Increase of consumption of high-fructose corn syrup runs in the US parallel with type 2 diabetes, even tough causal explication does not exist [90, 91]. The moderate intake of sugars (≤10%en) is considered as harmless [92, 93].

Sugars and oral health are in strong correlation. Sugars and other fermentable carbohydrates provide substrate for oral bacteria, which in turn lower plaque and salivary pH. Other dietary factors may reduce the risk of caries. A balanced diet with the required amount of fluoride and a good oral hygiene may prevent the dental caries [85, 86].

Conclusions

Sugar or sucrose is a readily available source of energy that almost all cells in body utilize, and especially important for the cells of brain. Many foods contain natural sugars and in several food items sucrose, more rarely glucose, fructose, or other sugars appear as added sugar in technological processes. It is advised to reduce the intake of sugars, because its excessive amount causes several disadvantageous consequences:

1. High intake of added sugars may predispose to obesity.
2. Obesity looks to be risk factor for type 2 diabetes mellitus and CVD, including CHD.
3. High sugar diet elevates the fasting serum TAG level, which is also a CVD risk factor.
4. The cariogenic effect of sugars is indisputable. (Other carbohydrate-rich foods have similar sequels but the individual items have different cariogenic potential.) Sucrose, glucose and fructose belong to the most cariogenic nutrients. Fluoride in drink water, in toothpastes, careful oral hygiene may powerfully reduce the caries frequency.

The disadvantages of sugars rich foods enumerated above justify the reduction of sugars intake and to keep uptake within the range of modest intake.

The nutrition (nutrient content) claims concerning sugars content of foods are supported by commonly accepted scientific evidences.

5.4. Source of Fibre, High Fibre

The dietary fibre consists of edible parts of plants that cannot be digested or absorbed in the small intestine, and unchanged passes into the large bowel. The compounds belonging to the family of dietary fibre comprise carbohydrates, mostly polysaccharides. Non-starch polysaccharides are the main components of dietary fibre. They include: cellulose, hemicellulose, pectins and gums. Oligosaccharides, lignin, associated plant substances (e.g. waxes), and the so called resistant starch constitute further members of family. The separate components of dietary fibre have different structure, properties and biological effects. The gastrointestinal organs are unable to digest dietary fibre that helps to ensure gut function by increasing physical bulk in the bowel and stimulating the intestinal transit. Some fibres such as pectins, gums and oligosaccharides but also resistant starch and non-starch polysaccharides are partially fermented by the gut microflora. Some scientists exclude resistant starch and lignin and propose to use the naming "non-starch polysaccharide" but naturally it isn't equal with total dietary fibre.

Dietary fibre is found solely in plants: in fruits, vegetables and whole grains.

Dietary fibres are classified according to their physical characteristics, i.e. solubility, which is related to biological effect. The two types are soluble and insoluble fibres, both of them are found beside each other. Oats, barley, fruits, vegetables and pulses are good sources of soluble fibres, wholegrain cereals, wholemeal bakery products those of insoluble fibres ([7, 93, 94]).

As a result of microbial fermentation short-chain fatty acids (SCFAs) are formed in the colon. SCFAs are taken up by the colonic epithelial cells. This process is thought to be important for the maintenance of epithelium health and for the prevention of colon cancer.

In addition, particularly insoluble fibres increase the stool mass, boost the bowel peristaltic, and so decrease gut transit time; the fibres may bind and quickly remote the carcinogenic substances, too. This mechanism reduces the prevalence of constipation, the risk of diverticular disease, haemorrhoids and proliferation of tumour cells [95].

Soluble fibres help to improve the glycaemic control as well as dyslipidaemia via delaying digestion and adsorption of carbohydrates and fats. In clinical trials it was shown that isolated viscous fibres (pectin, oat or rice bran) lower serum total cholesterol and LDL-C that promote the protection against CHD. Majority of studies indicates that an increase of both soluble and insoluble fibres prolong post meal satiety, decrease subsequent hunger, mitigate energy intake, promote weight loss. Since the low energy density facilitates the loss of weight, and the fibres "dilute" the energy content in foods, the fibres form a very favourable tool for the slimming diet [96, 97, 98].

Very high intake of dietary fibre may have some adverse effects. Loose stools, increased bowel peristaltic, flatulence have been observed. High fibre diet may decrease the bioavailability of minerals such as iron, calcium and zinc, that of other bioactive substances such as β-carotene, lycopene, lutein, [93, 99]

Conclusions

Several health advantages go together with dietary fibre intake of desirable level. It is advised to include foods with higher dietary fibre content into the diet because the dietary fibre level of common Western diet is low and the dietary fibres

1. contribute to prevention of colon cancer,
2. boost the colon peristaltic, take part in prophylaxis of diverticular disease, haemorrhoids,
3. control the postprandial glycaemia and dyslipidaemia,
4. support weight loss or desirable weight maintenance.

Disadvantageous effects are shown in the case of very high intake only.

The nutrition (nutrient content) claims concerning dietary fibre content of foods are supported by commonly accepted scientific evidences.

5.5. Low Sodium/Salt, Very Low Salt, Sodium-Free or Salt-Free

The correlation between salt intake and prevalence of hypertension is well known from the second half of last century. The measurement of urinary sodium excretion and blood pressure proved this association in INTERSALT study in the eighties: people with very low sodium excretion had low median blood pressure, a low prevalence of hypertension and no increase of blood pressure with ageing. In other cases the sodium excretion was related to blood pressure that increased with age. At the final evaluation the sodium excretion showed a powerful association, particularly with stroke mortality; whereas the association with blood pressure was weaker. It was revealed that a positive difference in sodium intake of 100 mmol/d over a 30-year period results in an increase of about 3-6 mmHg in systolic blood pressure and 0-3 mmHg in diastolic blood pressure.

The results of other surveys don't support the rather strong relationship, e.g. according to the data from the National Health and Nutrition examination Survey (NHANES I, 1971-1975) all-cause mortality rates were *inversely* and significantly related to daily sodium intake. A similar inverse relationship was observed in the case of CVD mortality rates from lowest to highest quartile of sodium intake [100, 101, 102, 103].

An overview of the salt debate highlights this complicated issue [104]:

- 1972: Clinical, ecological, and rat studies supporting salt-blood pressure link.
- 1973. 27 ecological studies suggest a direct linear relationship between salt and blood pressure.
- 1979. Study on several hundred schoolchildren shows "not wholly negative" relationship between salt and blood pressure.
- 1984. Analysis of NHANES database advocates that salt is harmless, and calcium and potassium are protective against hypertension.
- 1988. A study on Scottish men finds no relationship.
- 1988. The INTERSALT study shows weak (or no) relationship between salt and blood pressure.
- 1996. INTERSALT Revisited: after statistical re-analysis, the original data now signal consistent positive association.
- 1991. Meta-analysis of 27 clinical trials finds that salt reduction lowers blood pressure.
- 1991. A review of 24 ecological studies, 24 intrapopulation studies, and 78 clinical trials finds, that salt–blood pressure link is substantially larger than generally appreciated and increases with age.
- 1996. It is concluded from meta-analysis of 56 clinical trials that benefit from salt reduction is small.
- 1997. Meta-analysis of 32 clinical trials shows that benefit of salt reduction is larger.
- 1997. Clinical trial indicates that long-term reduction of salt intake results in little or no reduction of blood pressure.
- 1997. Clinical trial shows that dietary factors other than sodium have much greater effect on blood pressure.
- 1998. Meta-analysis of 114 clinical trials does not support a general recommendation to reduce salt intake.

Undoubtedly, the hypertension is also a multifactorial disorder. Apparently the sodium/salt intake is one of causes, which might for the most part explain these contradictory, mixed features. An important factor is the salt sensitivity as a genetic background. The salt sensitivity can be inherited. There are people who are sensitive for salt and react with higher blood pressure to a higher salt intake. The others behave neutrally, without higher blood pressure. The ratio of sensitive subjects is about 30-50% in people suffering from hypertension and 15-25% in people with normal blood pressure. The heterogeneous responses are partly required (age, intake of other electrolytes, certain medicaments). Genetic predisposition also play role. Salt sensitivity is increased in person with type 2 diabetes mellitus. Short-term and long-term dietary interventions with salt expansion and restriction are suitable to identify salt-sensitive and salt-resistant individuals. However, they are cumbersome and cannot be used in a large scale.

Molecular genetic techniques for identifying sensitive and resistant individuals are not yet available but if the putative gene polymorphisms will be identified such techniques may replace the current trial-and-error methods [9, 105, 106]. Salt influences the function sympathetic nervous system and endothelium, insulin resistance, hypertension [107]. Much salt increase independently the volume of thrombocytes and blood pressure [108].

Conclusions

The debate is going on; one may read and hear pros and cons [109, 110, 11, 112]. Dietary sodium restriction may be considered in old hypertonic persons but it is not evident for healthy population with normal blood pressure [113, 114, 115]. Other scientists universally think the reduction necessary [116, 117]. Lower salt intake is advised for the sake of prevention, because higher sodium excretion leads to higher blood pressure [118, 119, 120]. The moderation of salt/sodium intake to the majority of population is categorically beneficial (reduction of risk of stroke, other CVDs) [121].

The nutrition (nutrient content) claims concerning sodium/salt content of foods are supported by widely accepted scientific evidences.

5.6. Source of Protein, High Protein

The word "protein" comes from classical Greek; it means originally "of primary importance". The life is tied to proteins, indeed. The proteins have a wide range of functions in the human body: structural functions from cells to the entire body, protective functions in immune reactions, transport and communication functions from intracellular communications to hormones and transport plasma proteins, and enzymic functions from metabolic pathways to digestion. The daily protein intake is adequate at the level of 0.6-0.8 g/BMkg[4] when a mixed diet is eaten that is common in developed countries. In Europe, apart from some peculiar conditions, the dietary protein abundantly covers the requirements, supplementation appears unnecessary.

The protein requirement elevates in the case of

- an increase in demand (e. g. growth, pregnancy, breastfeeding, infection, reconvalescence, corticosteroid treatment, postoperative status, stress situation),
- an increase of losses (e.g. bleeding, burns, diarrhoea, nephrosis).

The foods with declared higher protein content may give information for the general public but they are important particularly for persons having higher protein requirements [7, 9, 122, 123].

If we reckon with the physiological effects of different protein types, it looks to be interesting the origin of protein, whether it is a complete or incomplete protein. The planned claims don't reveal this issue although this is a crucial criterion: the consumers with elevated protein requirement need mainly complete proteins; the consumers who seek foods for health promotion, look for proteins of plant origin, i.e. chiefly incomplete but in another respect more desirable ones.

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[4] Body mass kilogram

These nutrient content claims bring to mind the much sophisticated sport food preparations, and they might strengthen the myth of the protein as the only factor in the growth of muscle mass.

Conclusions

The vital significance of proteins is indisputable, detailed scientific support is needless. However, nutrition claims referring to higher protein content may be argued. To whom are they addressed? To everyday people? But their protein intake may be considered as adequate in the European Union, moreover no rarely superfluous. To sportsmen? But it is so misleading. To not healthy subjects who have higher protein requirement from different causes? Yes, for them the claims are useful, when they need such foods according to advice of practitioner or dietician. A question arises: is a sensible idea to propose claims with a limited competence?

The nutrition (nutrient content) claims concerning higher protein content of foods are useful for a cohort of people, not for everybody, albeit the significance of proteins is supported by commonly accepted scientific evidences. However, their necessity and/or their wording may be reconsidered (e.g. "Source of plant protein", Source of complete protein" etc.).

5.7. Natural Source of Vitamins and/or Minerals, Enriched or Fortified in Vitamins and/or Minerals, High Vitamins and/or Minerals, Contains (name of the nutrient or other substance), Increased (name of the macronutrient), Reduced (name of nutrient), Light/Lite

There is here a beautiful bouquet of claims with similar meanings. In several European populations considerable subgroups have intakes below the recommended levels for some vitamins, trace and macroelements. The choice of naturally rich foods or the voluntary addition of micronutrients to the appropriate foods may help to reduce the risk associated with suboptimal micronutrient intakes [124].

There is no doubt about the biological significance of each vitamin, macro- and microelements. Some examples:

- The clinical symptoms of vitamin deficiencies are unusual in the European Union, suboptimal vitamin status may occur. This status is associated with a number of chronic non communicable diseases, such as CVD, cancer, osteoporosis. Cohort studies, randomized trials and meta-analyses show relation between vitamin intakes and that kind of diseases. Folate and vitamins B_6 and B_{12} are required for homocysteine metabolism and are so associated with CHD risk. Vitamin E and the carotenoid lycopene may decrease the risk of prostate cancer. Vitamin D is associated with decreased occurrence of fractures when taken with calcium. Higher folate intake appears to reduce the risk of colon and breast cancer, particularly among moderate consumers of alcohol and in pregnant women to prevent the neural tube defects of infants. Vitamin E may be useful in primary prevention of CVD when taken thorough long periods. Vitamin C and E together might have additional benefits for preventing CHD. Vitamin K may take part in prevention of fractures in elderly people, though the evidence is not strong [125, 126].
- On the other hand, findings provide strong support that, at least among smokers, beta-carotene (provitamin) supplementation increases the risk of lung cancer, but other carotenoids from foods may decrease the risk. Beta-carotene does not reduce CHD risk. Probably CHD is influenced by another dietary and/or lifestyle factors that lead to CHD.

Some researcher reported that higher plasma concentration of lutein/zeaxanthine, retinol slightly increased the risk of CVD, and there was no association between vitamin E, beta-cryptoxanthine, alpha- and beta-karotin, lycopene and CVD. Giving antioxidant vitamins as supplements is not going to correct the basic problem of dietary lipid peroxidation that has a possible role in atherogenesis. Oxidation might promote, antioxidants might inhibit atherosclerosis [127]. There are no studies suggesting that vitamin C supplementation is associated with decreased cancer risk. Its role in CHD prevention is unconvincing. High doses of vitamin A (retinol) increases the risk of hip fractures in postmenopausal women, and among women of child-bearing age during early pregnancy may cause foetal damage [128, 129, 130, 131, 132, 133]. The data accumulated yet don't support the preventive effect of beta-carotene but a diet rich in carotenoids affects favourably the risk of CHD in accordance with epidemiological studies [134].

- Activation of immune system will exert a stress upon the antioxidant defences of the body. The oxidative damage is a by-product of attempts by the immune system to combat invading pathogens by production of oxidant molecules. The degree of depletion of components of antioxidant defences can be modulated by intake of nutrients, particularly antioxidative vitamins. Deficiencies in vitamins E, B_2 and B_6 produce functional abnormalities in the cell-mediated immune response. Vitamins C and E exert anti-inflammatory effects, which is most apparent in the elderly [135, 136]. Supplements containing vitamins and trace elements strengthen the immune responses in older people [137].

The generation of reactive oxygen species (ROS) is associated with life in aerobic conditions. ROS are implicated in the pathogenesis of several human diseases since they are capable of damaging biological macromolecules such as desoxyribonucleic acid (DNA), carbohydrates, lipids and proteins. A number of non-nutritive components in foods exert antioxidative effect. Flavonoids include over 4000 naturally occurring compounds, which provide colour, texture and taste for plant foods. As free radical scavengers, flavonoids inhibit lipid peroxidation, promote vascular relaxation and help to prevent atherosclerosis, to inhibit tumour development. A sufficient supply with antioxidants, including also non-flavonoid phenolics, from diet might contribute to prevent or delay the occurrence of pathological changes associated with oxidative stress [138, 139, 140, 141]. Flavonols affect platelets like aspirin, also inhibit activation [142]. Oxidative stress is a major mechanism leading to impaired endothelial function and is present in hypertension and dyslipidaemia. Theoretically the antioxidants can bring benefits for the prevention of CVD and cancer but in the practice they are unstable [143]. Several brain functions are associated with free radicals, advanced glycation end products, on the other hand with antioxidants, antioxidative capacity. The brain tissue is very sensible to oxidative damage that might play role in Alzheimer's disease, vascular dementia, moreover in Down's syndrome [144, 145].

A number of macro- and micronutrient (calcium, proteins, magnesium, zinc, copper, fluoride, vitamins K, D, and sodium/salt) influences the osteoporosis, and are significant in its prevention in the elderly, above all in women [146].

Hypertension is associated with the deficiency of some micronutrients. Some hypertensive subjects who are not amenable to treatment by antihypertensive drugs do respond to minerals and vitamins: calcium, antioxidant vitamins, certain metabolites of vitamin D [24,25-$(OH)_2D_3$], vitamin B_6, moreover vitamins C, and E inhibit the oxidation of NO, the most important vasodilator of the vascular endothelium [147].

Copper and iron induce the oxidation of LDL but copper deficiency elevates the peroxidative susceptibility of LDL. Zinc deficiency may lead to oxidative damage [148] Dietary phytoestrogens (isoflavones and lignans) improve the TAG, and the mean cardiovascular risk factor metabolic score [149]. The isoflavones are implicated in prevention of cancer, osteoporosis and influence the immune system [150, 151].

It has been known for nearly 50 years that plant sterols and stanols (phytosterols and phytostanols) lower blood cholesterol levels. People can reduce their cholesterol and cardiovascular risk by eating balanced diet (low in SFA and cholesterol, with optimal ratio of n–3 to n-6 PUFA, high in fruit and vegetables), and by continuous physical activity. It is proved in many clinical studies that dietary plant sterols and stanols lower total and LDL cholesterol by partly inhibiting cholesterol absorption and so strengthen the effects achieved by other strategies [152, 153, 154].

In the case of enriched or fortified foods a particular caution is essential. One must take into account the tolerable upper intake levels (UL), an estimate of current intakes of micronutrients from non-fortified foods in Europe at the 95th percentile (CI_{95}), similarly an estimate of current energy intakes (E_{95}), the maximum amount of each nutrient added to the whole diet with a minimum risk of adverse health effects (MA_n), the amount of each nutrient may be added safely to each 100 kcal portion (FA_n), the fraction of foods in the market, which is available for fortification, for each individual nutrient (PFF_n). As a consequence, in the case of enriched or fortified foods the prescription of generally valid rule is impossible; the judgement should be carried out individually [124].

Conclusions

The benefits of vitamins in reasonable amount are scientifically well-established without respect to that whether they come from natural sources or from enriched/fortified foods. The conditions in the case of macro and microelements are very similar. The scientific background at all times depends on specific micronutrient, on its chemical, human biological peculiarities. Mention must be made of the sometimes inconclusive results concerning the effects of micronutrients beyond the classic efficacy, i.e. in the domain of chronic non-communicable diseases' prevention.

The field of non-nutritive, biologically effective food components appears to be much more controversial. There are many, many studies of various types that investigate isolated and non-isolated, combined or non-combined substances; the number of papers is almost endless. One part of results provides positive consequences, the other part does neutral ones, and maybe a third part reports on disadvantageous, moreover hazardous outcomes. This also goes to show that the food/diet environment vigorously can influence the final biological events.

These compounds must be judged individually, concrete scientific study of efficiency is absolutely needed and the conditions of application (amount, frequency, milieu etc.) should be scrutinized.

The claim "Increased (name of macronutrient)" appears redundant. The increased protein content has two separate claims; the increase of fats and sugars isn't desirable. The only complex carbohydrates may be favourable. What purpose does this claim serve?

The claim "Light/Lite" repeats the claims "LOW ENERGY", "ENERGY REDUCED", "ENERGY-FREE", "LOW FAT", "FAT-FREE", "LOW SUGARS", "SUGARS-FREE" and "WITH NO ADDED SUGARS" but it is a well-known and popular term, therefore it is acceptable.

The nutrition (nutrient content) claims concerning foods as natural source of vitamins and/or minerals, foods enriched or fortified in vitamins and/or minerals, foods with high vitamins and/or minerals are supported by widely accepted scientific evidences. During the process of application the UL levels, the intakes from other sources, the safe amounts and other factors individually should be taken into account.

The nutrition (nutrient content) claims concerning foods containing specific nutrient or other substances, foods with increased macronutrient content, and foods with reduced nutrient content are supported by scientific evidences in particular cases only. For licensing of each substance detailed scientific documentary evidence (dossier) is necessary. During the process of application – among other factors – the safe and efficient amounts should be taken into account. The use of hazardous or inefficient substances must be avoided.

5.8. Proposal for a new nutrition (nutrient content) claim concerning n–3 PUFA content

In the interest of improvement of the proportion of dietary fatty acids, taken into consideration the human biological effects of n–3 PUFAs in prevention of chronic non-communicable diseases, and their low intake in several European region, on the basis of scientific evidence stated in chapter on fats, the need of the following claim arises:

SOURCE OF n–3 POLYUNSATURATED FATTY ACIDS

A claim that a food is high in n–3 PUFAs, and any claim likely to have the same meaning for the consumer, may only be made where the product contains at least 0.8 g n–3 PUFA per 100 g, from which at least 0.3 g EPA and DHA per 100 g.

In the case of foods naturally high in n–3 PUFAs, including EPA and DHA, the term "naturally" may be used as a prefix to this claim.

5.9. Proposal for a new basic principle

The advantageous food component declared in nutrition claim must guarantee that in the given food doesn't contain other components in excess or any harmful, potential harmful substance. For instance, it is not allowed that a food with claim "LOW SUGARS" has high fat content. The nutrition claim should mean safety that the food favourably may be incorporated into a balanced diet and take part in promoting health.

5.10. Some words on health and nutrition claims – on the whole

The human beings obtain some nutritional practice from the time of infancy, day by day, and they believe that they are perfectly skilled in the field of nutrition. However, it is not true. One should learn not only to eat but to prudently eat. And it is a complicated, cognitive process influenced by several economical, social, and physiological factors.

Everybody must be familiar with the basic rules of nutrition maintaining and promoting health, preventing diet-related diseases. The first stage of the attainment passes off in the family, in narrower surroundings. The outcome doesn't – saying in gentle manner – aim always at balanced diet. The second stage follows in the school years. The pupils get more specific information on nutritional biology, on relationships between food and health, on right food choice etc. The numbers of lessons for nutrition, the range of nutritional knowledge taught in schools are on different level in various countries; at the beginning of fight for life the new generation by no means possesses a comprehensive, practically useful nutritional well-informedness. Now there is here an adult with often deficient, even more faulty familiarity in nutrition and he/she is exposed to the bombardment of food advertisement, many fruitless, scientifically very little based nutritional recommendations with a grain of truth. This man errs, cannot see clearly, cannot decide and cannot use the benefits of a balanced diet. The nutritional professional must help to this adult. One way is the correct popular education. The other one is the correct food labelling and the health and nutrition claims. The labelling gives valuable information to people knowledge of whom suffices for evaluating the energy and nutrient content, likewise the other components in food. The claims are meant for all the people, catch their hand; guide them to the right choice. The evidence-based claims may prevent – together with a wide-ranging education – the consumers from misleading influences. It's essential to call the population's attention to read the information on the wrapper of foods including health and nutrition claims. If the claims are very easily understandable as the proposed ones, the consumer can weigh that he/she buy the product or not. The clients compare their own experiences and earlier information and they are able to decide rightly using the direct influence of the given claim. This process means at the same time a very slow, step by step change of consumer's eating behaviour in the desired direction, if it is necessary. The claims can make often salient health advantages and help to know the ins and outs of the huge food selection. Consumers who already have favourable attitudes about healthy eating practice, so become a strong stimulus to strengthen and facilitate their valuable efforts yet. Theoretically the claims can cause adverse effect: when one claim is overestimated and results throw the nutrition out balance (e.g. intake too much fibre) but it is perfectly unlikely if the use of claims is accompanied by appropriate nutritional education excludes the misunderstanding.

From industrial point of view the health and nutrition claims stimulate the producers to develop foods of composition, which better corresponds to the requirements of a balanced diet. Thus the choice of foods that more easily may be included in a healthier nutrition will be broadened.

REFERENCES

1. Clydesdale, F. M.: A proposal for the establishment of scientific criteria for health claims for functional foods, Nutr. Rev., 1997, 55, 413-422.

2. – Control of health claims in foods, Lancet, 1998, 351, 609.

3. – Dietary Reference Intakes: Guiding principles for nutrition labelling and fortification (Committee on Use of Dietary Reference Intakes in Nutrition Labeling of the Institute of Medicine, National Academies), Nutr. Rev., 2004, 62, 73-79.

4. Hasler, C. M.: Functional foods: Benefits, concerns and challenges – A position paper from the American Council on Science and Health, J. Nutr., 2002, 132, 3772-3781.

5. Asp, N.-G., Contor, L.: Process for Assessment of Scientific Support for Claims on Foods (PASSCLAIM): overall introduction, Eur. J. Nutr., 2003, 42, I/3-I/5.

6. Contor, L., Asp, N.-G.: Process for Assessment of Scientific Support for Claims on Foods (PASSCLAIM), Eur. J. Nutr., 2004, 43, II/3-II/6.

7. Eastwood, M.: Principles of human nutrition (Second edition), Blackwell Science, Oxford, 2003.

8. Gibson, R.: Dietary assessment, in Essentials of human nutrition (Second edition)(Eds.: Mann, J., Truswell, A.S.), Oxford University Press, Oxford, 2002, pp. 449-466.

9. Biró, G., Biró Gy.: Élelmiszer-biztonság. Táplálkozás-egészségügy (Food safety. Public health nutrition), Agroinform Kiadó, Budapest, 2000, pp. 217-221.

10. Biró, Gy., Hulshof, K. F. A. M., Ovesen, L., Amorim Cruz, J. A. for the EFCOSUM Group: Selection of methodology to assess food intake, Eur. J. Clin. Nutr., 2002, 56, S25-S32.

11. – Backgrounder on obesity and overweight, www.eufic.org/en/quickfacts/obesity.htm

12. Nishida, C., Mucavele, P.: Monitoring the rapidly emerging public health problem of overweight and obesity: The WHO global database on Body Mass Index, SNC News, 2005, Number 29, 5-12.

13. – Diet, nutrition and the prevention of chronic diseases, World Health Organization, Technical Report Series No. 916, WHO, Geneva, 2003.

14. Rolls, B. J., Drewnowski, A., Ledikwe, J. H.: Changing the energy density of diet as a strategy for weight management, J. Am. Diet. Assoc., 2005,105, S98-S103.

15. Drewnowski, A.: Concept of nutritious food: toward a nutrient density score, Am. J. Clin. Nutr., 2005, 82, 751-732.

16. – Position of the American Dietetic Association: Weight management, J. Am. Diet. Assoc., 2002, 102, 1145-1155.

17. Rosenbaum, M., Leibel, R. L., Hirsch, J.. Obesity, N. Engl. J. Med., 1997, 337, 396-406.

18. Hoffman, D. J.: Obesity in developing countries: causes and implications, FNA/ANA, 2001, No. 28, 35-42.

19. Williamson, D. F.: The prevention of obesity, N. Engl. J. Med., 1999, 341, 1140-1141.

20. Ludwig, D. S., Peterson, K. E., Gortmaker, S. L.: Relation between consumption of sugar sweetened drinks and childhood obesity: a prospective, observational analysis, Lancet, 2001, 357, 505-508.

21. Seidell, J. C.: Dietary fat and obesity: an epidemiologic perspective, Am. J. Clin. Nutr., 1998, 67, 546S-550S.

22. Willett, W. C.: Is dietary fat a major determinant of body fat? Am. J. Clin. Nutr., 1998, 67, 556S-562S.

23. Grundy, S. M.. Overview, Am. J. Clin. Nutr., 1998, 67, 497S-499S.

24. Astrup, A., Raben, A.: Sugar as a slimming agent? Br. J. Nutr., 2000, 84, 585-587.

25. Hirsch, J., Hudgins, L. C., Leibel, R. L., et al.: Diet composition and energy balance in humans, Am. J. Clin. Nutr., 1998, 67, 551S-555S.

26. Doucet, E., Alméras, N., White, M. D. et al.: Dietary fat composition and human adiposity, Eur. J. Clin. Nutr., 1998, 52, 2-6.

27. Anderson, G. H., Moore, S. E.: Dietary proteins in the regulation of food intake and body weight in humans, J. Nutr., 2004, 134, 974S-979S.

28. Brand-Miller, J. C., Holt, S. H. A., Pawlak, D. B. et al.: Glycaemic index and obesity, Am. J. Clin. Nutr., 2002,76, 281S-285S.

29. Karason, K., Wallentin, I., Larsson, B. et al.: Effects of obesity and weight loss on left ventricular mass and relative wall thickness: survey and intervention study, BMJ, 1997, 315, 912-916.

30. Caterson, D.: Overweight and obesity, in Essentials of human nutrition (Second edition)(Eds.: Mann, J., Truswell, A.S.), Oxford University Press, Oxford, 2002, pp. 273-287.

31. Elrick, H., Samaras, Th. T., Demas, A.: Missing link in the obesity epidemic, Nutr. Res., 2002, 22, 1101-1123.

32. Hooper, L., Summerbell, C. D., Higgins, J. P. T. et al.: Dietary fat intake and prevention of cardiovascular disease: systematic review, BMJ, 2001, 322, 757-763.

33. – Backgrounder on fats, www.eufic.org/en/quickfacts/obesity.htm

34. Dwyer, J.: Overview: dietary approaching for reducing cardiovascular risk, J. Nutr., 1955, 125, 656-665.

35. Wahrburg, U.: What are the health effects of fat? Eur. J. Nutr., 2004, 43, I/6-I/11.

36. German, J. B., Dillard, C. J.: Saturated fats: what dietary intake? Am. J. Clin. Nutr., 2004, 80, 550-559.

37. German Nutrition Society, Austrian Nutrition Society, Swiss Society for Nutrition Research, Swiss Nutrition Association: Reference values for nutrient intake, Umschau Braus GmbH Publisher, German Nutrition Society, Frankfurt/Main, 2002.

38. Hayes, K. C.: Dietary fat and coronary heart disease, in Preventive nutrition (Eds.: Bendich, A., Deckelbaum, R. J.), Humana Press, Totowa, N. J., 1997, pp. 153-169.

39. Oomen, C. M., Ocké, M. C., Feskens, E. J. M. et al.: Association between trans fatty acid intake and 10-year risk of coronary heart disease in the Zutphen Elderly Study: a population-based study, Lancet, 2001, 357, 746-751.

40. Weel, C. van: Complexity of issue of dietary trans fatty acids, Lancet, 2001, 357, 732-733.

41. Pietinen, P., Ascherio, A., Korhonen, P. et al.: Intake of fatty acids and risk of coronary heart disease in a cohort of Finnish men, Am. J. Epidemiol., 1997, 145, 976-887.

42. Brouwer, I. A., Katan, M. B., Zock, P. L.: Dietary α-linolenic acid is associated with reduced risk of fatal coronary heart disease, but increased prostate cancer risk: A meta-analysis, J. Nutr., 2004, 134, 919-922.

43. Lorgeril, M. de, Salen, P.: Alpha-linolenic acid and coronary heart disease, Nutr. Metab. Cardiovasc. Dis., 2004, 14, 162-169.

44. Calder, P. C.: n–3 Fatty acids and cardiovascular disease: evidence explained and mechanism explored, Clin. Sci., 2004, 107, 1-11.

45. Harper, Ch. R., Jacobson, T. A.: The fats for life. The role of ω-3 fatty acids in the prevention of coronary heart disease, Arch. Intern. Med., 2001, 161 2185-2192.

46. Calder, P. C.: Long-chain n–3 fatty acids and cardiovascular disease: further evidence and insights, Nutr. Res., 2004, 24, 761-772.

47. Harris, W. S.: n–3 long-chain polyunsaturated fatty acids reduce risk of coronary heart disease death: extending the evidence to the elderly, Am. J. Clin. Nutr., 2003, 77, 279-280.

48. Lemaitre, R.B., King, J. B., Mozzafarian, D., et al.: n–3 polyunsaturated fatty acids, fatal ischaemic heart disease, and nonfatal myocardial infarction in older adults: the cardiovascular Health Study, Am. J. Clin. Nutr., 2003, 77, 319-325.

49. Djoussé, L., Pankow, J. S., Eckfeldt, J. H. et al.: Relation between dietary linolenic acid and coronary artery disease in the National Heart, Lung and Blood Institute Family Heart Study, Am. J. Clin. Nutr., 2001, 74, 612-619.

50. Jakobsen, M.V., Overvad, K., Dyerberg, J. et al.: Dietary fat and risk of coronary heart disease: possible effect modification by gender and age, Am. J. Epidemiol., 2004, 160, 141-149.

51. Weiss, L. A., Barrett-Connor, E., von Mühlen, D.: Ratio of n–6 to n–3 fatty acids and bone mineral density in older adults: the Rauch Bernardo Study, Am. J. Clin. Nutr., 2005, 81, 934-938.

52. Nettleton, J. A., Katz, R.: n–3 long chain polyunsaturated fatty acids in type 2 diabetes: A review, J. Am. Diet. Assoc., 2005, 105, 428-440.

53. Oomen, C. M., Ocké, M. C., Feskens, E. J. M. et al.: α-Linolenic acid intake is not beneficially associated with 10-y risk of coronary artery disease incidence: the Zutphen Elderly Study, Am. J. Clin. Nutr., 2001, 74, 457-463.

54. Connor, W. E.: n–3 fatty acids from fish and fish oil: panacea or nostrum? Am. J. Clin. Nutr., 2001, 74, 415-416.

55. Dewailly, E., Blanchet, C., Lemieux, S. et al.: n–3 Fatty acids and cardiovascular disease risk factors among the Inuits of Nunavik, Am. J. Clin. Nutr., 2001, 74, 464-473.

56. Haglund, O., Wallin, R., Wretling, S. et al.: Effect of fish oil alone and combined with long-chain (n–6) fatty acids on some coronary risk factors in male subjects, J. Nutr. Biochem., 1998, 9, 629-635.

57. Gillman, M. W., Cupples, A., Millen, B. E. et al.: Inverse association of dietary fat with development of ischemic stroke in men, JAMA, 1997, 278, 2145-2150.

58. Sherwin, R., Price, T. R.: Fat chance. Diet and ischemic stroke, JAMA, 1997, 278, 2185-2186.

59. Schreiner, P. J., Salomaa, V. V., Rasi, K. P. et al.: Dietary fat intake and hemostasis among participants in the 1992 FINRISK Hemostasis Survey, Nutr. Metab. Cardiovasc. Dis., 1998, 8 21-30.

60. Harrison, Gail. G.: Reducing dietary fat: Putting theory into practice, J. Am. Diet. Assoc., 1997, 97, S93-S96.

61. Kuller, L. H.: Dietary fat and chronic diseases: Epidemiologic overview, J. Am. Diet. Assoc. 1997, 97, S9-S15.

62. Kwiterowich, P. O.: The effect of dietary fat, antioxidants, and pro-oxidant on blood lipid, lipoproteins, and atherosclerosis, J. Am. Diet. Assoc. 1997, 97, S31-S41.

63. Hu, F. B., Willett, W. C.: Optimal diets for prevention of coronary heart disease, JAMA, 2002, 288, 2569-2578.

64. Hu, F. B., Bronner, L., Willett, W. C. et al.: Fish and omega-3 fatty acid intake and risk of coronary heart disease in women, JAMA, 2002, 287, 1815-1821.

65. Kritchewsky, D.: Overview: Dietary fat and atherosclerosis, Asia Pacific J. Clin. Nutr., 2000, 9, 141-145.

66. Sugano, M., Koga, T., Yamada, K.: Lipids and immunology, Asia Pacific J. Clin. Nutr., 2000, 9, 146-152.

67. Zyriax, B.-Ch., Windler, E.: Dietary fat in the prevention of cardiovascular disease – a review, Eur. J. Lipid Sci. Technol., 2000, 102, 355-365.

68. Spady, D. K.: Dietary fatty acids and atherosclerosis regression, Br. J. Nutr., 1999, 82, 337-338.

69. Mensink, R. P., Aro, A., Hond, E. D. et al.: Diet-related cardiovascular disease, Eur. J. Nutr., 2003, 42, I/6-I/27.

70. – Backgrounder on carbohydrates, www.eufic.org/en/quickfacts/obesity.htm

71. Deutsche Gesellschaft für Ernährung: Glykämischer Index und glykämische Last – ein für die Ernährungspraxis des Gesunden relevantes Konzept? Ernährungs-Umschau, 2003, 51, Teil 1: 84-91, Teil 2: 128-132.

72. Drewnowski, A., Specter, S.E.: Poverty and obesity: the role of energy density and energy costs, Am. J. Clin. Nutr., 2004, 79, 6-16.

72. – Slowly digestible carbohydrates, Danone Nutritopic, 2003, No. 28, 2-17.

73. Levine, A. S., Kotz, K. M., Gosnell, B. A.: Sugars: hedonic aspects, neuroregulation, and energy balance, Am. J. Clin. Nutr., 2003, 78, 834S-842S.

74. Anderson, G. H., Woodend, D.: Consumption of sugars and the regulation of short-term satiety and food intake, Am. J. Clin. Nutr., 2003, 78, 843S-849S.

75. Fried, S. K., Rao, S.: Sugars, hypertriglyceridemia, and cardiovascular disease, Am. J. Clin. Nutr., 2003, 78, 873S-880S.

76. J. Mann., Chisholm, A.: Nutritional determinants of serum triglycerides, Nutr. Metab. Cardiovasc. Dis., 1999, 9, 1-8.

77. Mann, J.: Sugar revisited – again, Bull. WHO, 2003, 81, 552.

78. Roberts, S. B.: High-glycemic index foods, hunger, and obesity: is there a connection?, Nutr. Res., 2000, 58, 163-169.

79. Coulston, A. M., Johnson, R. K.: Sugar and sugars: Myth and realities, J. Am. Diet. Assoc., 2002, 102, 351-353.

80. Kanazawa, M., Xue, Ch. Y., Kageyama, H. et al.: Effects of a high-sucrose diet on body weight, plasma triglycerides, and stress tolerance, Nutr. Res., 2003, 61, S27-S33.

81. Brand-Miller, J. C.: Glycemic load and chronic disease, Nutr. Res., 2003, 61, S49-S55.

82. Raxton, C. H. S.: Dietary guidelines for sugar: the need for evidence, Br. J. Nutr., 2003, 90, 245-247.

83. Alexy, U., Sichert-Hellert, W., Kersting, M.: Association between intake of added sugars and intakes of nutrients and food groups in the diets of German children and adolescents, Br. J. Nutr., 2003, 90, 441-447.

84. Bantle, J. P., Raatz, S. K., Thomas, W. et al.: Effects of dietary fructose on plasma lipid in healthy subjects, Am. J. Clin. Nutr., 1128-1134.

85. Ruxton, C. H. S., Garceau, F. J. S., Cottrell, R. C. : Guidelines for sugar consumption in Europe: Is a quantitative approach justified? Eur. J. Clin. Nutr., 1999, 53, 503-513.

86. Touger-Decker, R., van Loveren, C.: Sugars and dental caries, Am. J. Clin. Nutr., 2003, 78, 881S-892S.

87. Katan, M. B.: Are there good and bad carbohydrates for HDL cholesterol? Lancet, 1999, 353, 1029-1030.

88. Frost, G., Leeds, A. A., Doré, C. J. et al.: Glycemic index as determinant of serum HDL cholesterol concentration, Lancet, 1999, 353, 1045-1048.

89. Amano, Y., Kawakubo, K., Lee, J. S. et al.: Correlation between dietary glycemic index and cardiovascular disease risk factors among Japanese women, Eur. J. Clin. Nutr., 2004, 58, 1472-1478.

90. Jenkins, D. J. A., Kendall, C. W. C., Marchie et al.: Too much sugar, too much carbohydrate, or just too much? Am. J. Clin. Nutr., 2004, 79, 711-712.

91. Kross, l. S., Li, L., Ford, E. S. et al.: Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States: an ecologic assessment, Am. J. Clin. Nutr., 2004, 79, 774-779.

92. Langguth, S., Ziesenitz, S. C.: Zucker in der Ernährung – Neue Studien ermöglichen eine neue Bewertung, Ernährung/Nutrition, 1996, 20, Teil 1: 220-224, Teil 2: 294-297.

93. Brand-Miller, J.: Carbohydrates, in Essentials of human nutrition (Second edition)(Eds.: Mann, J., Truswell, A.S.), Oxford University Press, Oxford, 2002, pp. 11-29.

94. – Dietary fibre – what's role in a healthy diet? FoodToday, 2005, No. 49. www.eufic.org/gb/food/pag/food49/food492.htm

95. – Position of the American Dietetic association: Health implications of dietary fiber, J. Am. Diet. Assoc., 2002, 102, 993-1000.

96. Howarth, N. C., Saltzman, E., Roberts, S. B.: Dietary fiber and weight regulation, Nutr. Rev., 2001, 59, 129-139.

97. Rolls, B. J., Drewnowski, A., Ledikwe, J. H.: Changing the energy density of the diet as a strategy for weight management, J. Am. Diet. Assoc., 2005, 105, S98-S103.

98. Howarth, N. C., Saltzman, E., Roberts, S. B.: Dietary fiber and weight regulation, Nutr. Rev., 2001, 59, 129-139.

99. Riedl, J., Linseisen, J., Hoffmann, J. et al.: Some dietary fibers reduce the absorption of carotenoids in women, J. Nutr., 1999, 129, 2170-2176.

100. Mann, J.: Cardiovascular diseases, in Essentials of human nutrition (Second edition)(Eds.: Mann, J., Truswell, A.S.), Oxford University Press, Oxford, 2002, pp. 273-334.

101. Stamler, J.: The INTERSALT study: background, methods, findings and implications, Am. J. Clin. Nutr., 1997, 65, 626S-642S.

102. Antonios, T. F. T., MacGregor. G. A.: Salt – more adverse effect, Lancet, 1996, 348, 250-251.

103. Esslinger, K. A., Jones, P. J. H.: Dietary sodium intake and mortality, Nutr. Rev. 1998, 56, 311-313.

104. Taubes, G.: The (political) science of salt, Science, 1998, 281, 898-907.

105. Cowley A. W. Jr.: Genetic and nongenetic determinants of salt sensitivity and blood pressure, Am. J. Clin. Nutr., 1997, 65, 587S-593S.

106. Luft, F. C., Weinberger, M. H.: Heterogeneous responses to changes in dietary salt intake: the salt-sensitivity paradigm, Am. J. Clin. Nutr., 1997, 65, 612S-617S.

107. Siani, A., Guglielmicci, F., Farinaro, E. et al.: Increasing evidence for the role of salt and salt-sensitivity in hypertension, Nutr. Metab. Cardiovasc. Dis., 2000, 10, 93-100.

108. Bath, P. M. W., Capuccio, F. P., Singer, D. R. J., et al.: A high salt intake is associated with an increase in platelet size: A further risk factor for vascular disease? Nutr. Metab. Cardiovasc. Dis., 1996, 5, 71-74.

109. McCarron D. A.: The dietary guideline for sodium: should we shake it up? Yes! Am. J. Clin. Nutr., 2000, 71, 1013-1019.

110. Kaplan, N. M.: The dietary guideline for sodium: should we shake it up? No! Am. J. Clin. Nutr., 2000, 71, 1020-1026.

111. McCarron: Diet and blood pressure – The paradigm shift, Science, 1998, 281, 933-934.

112. Alderman, M. H.: Salt, blood pressure and health: a cautionary tale, Int. J. Epidemiol. 2002, 31, 311-315.

113. Esslinger, K. A.: Dietary salt intake and mortality, Nutr. Rev. 1998, 56, 311-313.

114. Midgley, J. P., Mattgew, A. G., Greenwood, C. M. T. et al.: Effect of reduced dietary sodium on blood pressure. A meta-analysis of randomised controlled trials, JAMA, 1996, 1590-1597.

115. Graudal, N. A., Galløe, A. M., Garred, P.: Effects of sodium restriction on blood pressure, renin, aldosterone, catecholamines and triglyceride, JAMA, 1998, 279, 1383-1391.

116. – Dietary sodium and blood pressure, JAMA, 1996, 276, 1467-1469.

117. Whelton, P. K., Appe, L. J., Espeland, M. A. et al. for the TONE Collaboration Research Group: Sodium reduction and weight loss in the treatment of hypertension in older persons. A randomized controlled Trial of Nonpharmacologic Interventions in the Elderly (TONE), JAMA, 1998, 279, 836-846.

118. Thelle, D. S.: Salt and blood pressure revisited, BMJ, 1996, 312, 1240-1241.

119. Elliott, P., Stamler, J., Nichols, R. et al. For the Intersalt Cooperative Research Group: Intersalt revisited: further analyses of 24-hour sodium excretion and blood pressure within and across population, BMJ, 1996, 312, 1249-1253.

120. Hanneman, R. L.: Intersalt: hypertension rise with age revisited, BMJ, 1996, 312, 1283-1289.

121. Perry, I. J.: Dietary salt intake and cerebrovascular damage, Nutr. Metab. Cardiovasc. Dis., 200, 10, 229-235.

122. Elmadfa, I., Leitzmann, C.: Ernährung des Menschen (Third edition), Verlag Eugen Ulmer, Stuttgart, 1998.

123. Rigó, J.: Az egységes diétás rendszer (The integrated dietary system), in Táplálkozás, diéta (Nutrition, diet), (Ed.: Barna M.), Medicina, Budapest, 1996.

124. Flynn, A., Moreiras, O., Stehle, P. et al.: Vitamins and minerals: A model for safe addition to foods, Eur. J. Nutr., 2003, 118, 118-130.

125. Fairfield, L. M., Fletcher, R. H.: Vitamins for chronic disease prevention in adults, JAMA, 2002, 287, 3116-3126.

126. Fletcher, R. H., Fairfield, L. M.: Vitamins for chronic disease prevention in adults, JAMA, 2002, 287, 3127-3129.

127. Schwenke, D. C.: Antioxidants and atherogenesis, J. Nutr. Biochem., 1998, 9, 424-445.

128. Sesso, H. D., Buring, J. E., Norkus, R. P. et al.: Plasma lycopene, other carotenoid, and retinol and the risk of cardiovascular disease in men, Am. J. Clin. Nutr., 2005, 81, 990-997.

129. Hercberg, S., Galan, P., Preziosi, P. et al.: Antioxidant vitamins and cardiovascular disease: Dr Jekyll or Mr Hyde?, Am. J. Publ. Health, 1999, 89, 289-291.

130. Riemersma, R. A.: Antioxidant vitamins and coronary heart disease. Back to the square one? Eur. J. Lipid. Sci. & Technol., 2002, 104, 77-78.

131. Vivekananthan, D. P., Penn, H. S., Sapp, S. K. et al.: Use of antioxidant vitamins for the prevention of cardiovascular disease: meta-analysis of randomized trials, Lancet, 2003, 361, 2017-2023.

132. Feskanich, J., Singh, V, Willett, W. C. et al.: Vitamin A intake and hip fractures among postmenopausal women, JAMA, 2002, 287, 47-54.

133. Denke, M. A.: Dietary retinol – a double edged sword, JAMA, 2002, 287, 102-103.

134. Kritchevsky, S. B.: β-carotene, carotenoids and the prevention of coronary heart disease, J. Nutr., 1999, 129, 5-8.

135 Grimble, R. F.: Effect of antioxidative vitamins on immune function with clinical applications, Internat. J. Vit. Nutr. Res., 1997, 67, 312-320.

136. Diaz, M. N., Frei, B., Vita, J. A. et al.: Antioxidants and atherosclerotic heart disease, N. Engl. J. Med., 1997, 337, 408-416.

137. Chandra, R. K.: Influence of multinutrient supplement on immune responses and infection-related illness in 50-65 years old individuals, Nutr. Res., 2002, 22, 5-11.

138. Giugliano, D.: Dietary antioxidants for cardiovascular prevention, Nutr. Metab. Cardiovasc. Dis., 2000, 10, 38-44.

139. Kris-Etherton, P. M., Keen, C. L.: Evidence that the antioxidant flavonoids in tea and cocoa are beneficial for cardiovascular health, Current Opinion in Lipidology, 2003, 13, 41-49.

140. Sesso, H. D., Gaziano, J. M., Liu, S. et al.: Flavonoid intake and the risk of cardovascular disease in women, Am. J. Clin. Nutr., 2003, 77, 1400-1408.

141. Katan, M. B., Hollman, P. C. H.: Dietary flavonoids and cardiovascular disease, Nutr. Metab. Cardiovasc. Dis., 1998, 8, 1-4.

142. Pearson, D. A., Paglieroni, T. G., Rerin, D. et al.: Thrombotic Res., 2002, 106,191-197.

143. – Antioxidants in cardiovascular disease: randomized trials, Nutr. Rev., 1996, 64, 175-177.

144. Lethem, R., Orrell, M.: Antioxidants and dementia, Lancet, 1997, 349, 1189-1190.

145. Stähelin, H. B.: The impact of antioxidants on chronic disease in ageing and in old age, Int. J. Vitam. Nutr. Res., 1999, 69, 146-149.

146. Müller, S.-V.: Osteoporose und Ernährung, Ernährungsforschung, 1999, 44, 1-24.

147. Dokshinamurti, K., Dokshinamurti, S.: Blood pressure regulation and micronutrients. Nutr. Res. Rev., 2001, 14, 3-13.

148. Fields, M.: Role of trace elements in coronary heart diseases, Br. J. Nutr., 1999, 81, 85-5-86.

149. Kleijn, M. J. J. de, Schouw, Y. T. van der, Wilson, P. W. F. et al.: Dietary intake of phytoestrogens is associated with a favorable metabolic cardiovascular risk profile in postmenopausal U.S. women: The Framingham Study, J. Nutr., 2002, 132, 276-282.

150. Ren, M. Q., Kuhn, G., Wegner, J. et al.: Isoflavones, substances with multi-biological and clinical properties, Eur. J. Nutr., 2001, 40, 135-146.

151. Arjmandi, B. H., Smith, B. J.: Soy isoflavones' protective role in postmenopausal women: mechanism of action, J. Nutr. Biochem., 2002, 13, 130-137.

152. – Plant sterols and stanols – proven to lower cholesterol, FoodToday, 2005, No. 50. www.eufic.org/gb/food/pag/food50/food502.htm

153. Jong, A. de, Palt, J., Mensink, R. P.: Metabolic effects of plant sterols and stanols (Review), J. Nutr. Biochem., 2003, 14, 362-369.

154. Plat, J., Mensink, R. P.: Effects of plant sterols and stanols on lipid metabolism and cardiovascular risk, Nutr. Metab. Cardiovasc. Dis., 2001, 11, 31-40.

155. Knopp, R. H., Retzlaff, B. M.: Saturated fat prevent coronary artery disease? An American paradox, Am. J. Clin. Nutr., 2004, 80, 1102-1103.

156. Mozzafarian, D., Rimm, E. B., Herrington, D. M.: Dietary fats, carbohydrate, and progression of coronary atherosclerosis in postmenopausal women, Am. J. Clin. Nutr., 2004, 80, 1175-1184.

157. Assema, P. van, Glanz, K., Brug, J. et al.: Effects of health claims on eating habits of the Dutch population, Eur. J. Publ. Hlth., 1996, 6, 281-187.

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