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# **Nutritional Management in Type 1 Diabetes Mellitus**

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#### 1. Introduction

An optimal nutrition along with insulin therapy in pations with type 1 diabetes, is the prerequisite for normal growth and development, adequate pubertal development, and regular performance in school and extracurricular activities, including sports in diabetic patients. Moreover, a balanced and healthy diet prevents hyper- or hypoglycemia and delays development of microvascular diabetic complications, including diabetic nephropathy. (Kliegman M Robert et al., 2007)

Dietary recommendations for children with diabetes are based on healthy eating recommendations suitable for all children and adults and therefore the whole family. Nutritional advice must be adapted to cultural, ethnic and family traditions and the psychosocial needs of the individual child. Likewise the choice of insulin regimen should take into account the dietary habits and lifestyle of the child.(NICE, 2004., American Diabetes Association, 2003)

A meal plan based on the individual's usual food intake should be determined and insulin therapy integrated into the usual eating and exercise patterns. Individuals on insulin therapy need to eat at consistent times synchronized with the time-actions of insulin, monitor blood glucose levels, and adjust insulindoses for the amount o food usually eaten or required (Frany J. Marion et al., 1994).

# 2. Guidelines on energy balance, energy intake and food components

No nutrition recommendations can be made for the prevention of type 1 diabetes at this time. (Franz MJ, et al., 2002), but increasing overweight and obesity in youth appears to be related to the increased prevalence of type 2 diabetes, particularly in minority adolescents. Although there are insufficient data at present to warrant any specific recommendations for



the prevention of type 2 diabetes in youth, interventions similar to those shown to be effective for prevention of type 2 diabetes in adults (lifestyle changes including reduced energy intake and regular physical activity) are likely to be beneficial. Clinical trials of such interventions are ongoing in children.

Individuals who have pre-diabetes or diabetes should receive individualized medical nutrition therapy (MNT); such therapy is best provided by a registered dietitian familiar with the components of diabetes MNT. Meta-analysis of studies in non-diabetic, free-living subjects and expert committees report that MNT reduces LDL cholesterol by 15-25 mg/dl. (Yu-Poth S. et al., 1999. Grundy SM., et al. 1997)

The prevalence of overweight children and adolescents with type I Diabetes mellitus has tripled over the past 20 years, which appears to correspond to the increasing prevalence of obesity in the general population. The authors (Kliegman M Robert et al., 2007), have observed patients with type I diabetes, normal-weight preschool children have better glycemic control than age-matched overweight children. This may mean that excess body weight status may impede achievement of therapeutic goals in this group of patients.

The basis for energy requirements calculations is the determination of ideal body-weight. It is assessed corresponding to the respective tables comprising child's age, gender and body size data.

Guidelines for daily caloric requirements in children

- 1000 Kcal + 100 Kcal/year age (for 0-12 year old)
- 1500-2000 kcal + 100 Kcal/year of age > 12 ( for females 12-15)
- 2000-2500 Kcal+ 200 Kcal/year of age > 12 ( for males 12-15)

Age	kcal/kg/d	Protein (in grams) kg/d		
6 months	115			
12 months	105	2.0		
3 years	100	1.8		
6 years	85	1.5		
10 years	86	1.2		
Male				
11-14 years	60	1.0		
15-18 years	42	0.85		
19-22 years	41	0.80		
Females				
11-14 years	48	1.0		
15-18 years	38	0.84		
19-22 years	38	0.80		
Adapted from National A	cademy of Sciences Food and	Nutrition Board		

Table 1.

A recommendations and plans on intake of certain kinds of foods depends on daily energy expenditure which is determined by the individual patient's age, gender and level of physical activity. (Lean, M.E. J et al. 1980) In reality, the practical assessment of energy intake relies on follow-up of the patients' growth and body mass gain. If the tendency towards obesity has been identified (which usually occurs in the puberty and after the growth spurt cessation, most often in female patients) the energy intake should be reduced to 80-90% of standard calculated intake. On the other hand, in children with insufficient body weight a body mass deficit can be corrected using foods with high energy content. (Stepanović R., et al. 1991)/ An adequate diet enables a pediatric patient to utilize ingested food effectively even though the spontaneous endogenous insulin secretion ceased and life depends on anabolic effects of exogenous insulin administered usually in 2-3 daily doses. Since the insulin doses are delivered in the regular diurnal manner, at the same time every day, the food intake must be adjusted according to the dosage schedule, amount and type of administered insulin. A meal should be placed 30-60 minutes after regular insulin dose (in fact, regular insulin has an onset of action 15-60 min after injection, a peak effect 2-4 h after injection, and a duration of action of ranging from 5 to 8 h) comparing to 15 min with the newer synthetic insulin analogues. Synthetic insulin analogues, both lispro and aspart have an onset of action within 15 min, a peak in activity at 60-90 min, and a duration of action of 3-5 h. Therefore, a total daily food intake should be divided into six meals. Breakfast/ lunch / supper ratio should comprise 20% / 30% / 20% of a total daily intake while two snacks and a bedtime meal should consist of 10% of the daily intake each. Also, it is important to ingest about the same amount of carbohydrates at the regullar time every day and to eat meals regullarily in order to avoid the occurrence of hypoglycemic episodes. (Stepanović R., et al. 1991)

# 3. Carbohydrate

Dietary carbohydrate has both chemical structural features and form which have gained in importance in recent years. The process of digestion of carbohydrate has been known for many years and instinctively it is held that a monosaccharide must be absorbed more readily than an oligosaccharide, which requires hydrolysis before absorption.

The recommended dietary allowance (RDA) for digestible carbohydrate is 130 g/day and is based on providing adequate glucose as the required fuel for the central nervous system without reliance on glucose production from ingested proteins and fats. Although brain fuel needs can be met on lower-carbohydrate diets, long-term metabolic effects of very-low-carbohydrate diets are unclear, and such diets eliminate many foods that are important sources of energy, fiber, vitamins, and minerals and are important in dietary palatability (Institute of Medicine:2002). There are no trials specifically in patients with diabetes restricting total carbohydrate to <130 g/day. However, 1-year follow-up data from a weight-loss trialamong the subset with diabetes indicated that the reduction in fasting glucose was 21 mg/dl (1.17 mmol/l) and 28 mg/dl (1.55 mmol/l) for the low-carbohydrate and low-fat diets, respectively, with no significant difference in A1C levels (Stern L., et al. 2004).

Department for Agriculture (USDA) reveal that – 43%-47% of calories are contributed by dietary carbohydrate, whereas 36-37% of calories are contributed by dietary fat, with 13% from saturated fatty acids, 14% from monosaturated, and 7% from polyunsaturated. A reduction in high dietary takes of saturated fats, trans-fatty acids and cholesterol (all of which contains cholesterol-raising fatty acids) is an important goal to reduce the risk of cardiovascular disease. Although diabetes mellitus is usually categorized as a disease of carbohydrate metabolism, abnormalities of lipoprotein metabolism and adipose tissue distribution are also common. Cardiovascular disease accounts for the majority of deaths in people with diabetes. Analysis of the Multiple Risk Factor Intervention Trial data for men with diabetes matched with non-diabetic men reported relative risk of death for men with diabetes was increased at a range from 2.83 to 4.46 depending on their level of serum cholesterol. (Stamler J. et al. 1993)

Approximately 70% of the carbohydrate content should be derived from complex such as starch; intake of sucrose and highly refined sugars should be limited.

An intake of simple carbohydrates with high fiber foods - such as complex grains (bran), vegetables (beans and peas containing galactomannan) and fruit (pectin) is recommended; this combination of food slows intestinal food absorption, reduce postprandial hyperglycemia and lowers serum cholesterol levels (Stepanović R., et al. 1991).

### 4. Glycemic index and glycemic load

By definition, the glycemic index (GI), compares equal quantities of available carbohydrate in foods and provides a measure of carbohydrate quality. Available carbohydrate can be calculated by summing the quantity of available sugars, starch, oligosaccharides, and maltodextrins. By definition, (Salmeron J. et al 1997), the glycemic load (GL) is the product of a food's GI and its total available carbohydrate content: glycemic load = [GI × carbohydrate (g)]/100.

Therefore, the GL provides a summary measure of the relative glycemic impact of a "typical" serving of the food. Foods with a GL ≤10 have been classified as low glycemic load and those with a value ≥20 as high glycemic load (Brand-Miller JC, Holt SHA, Petocz P: Reply to R. Mendosa. Am J ClinNutr 77: 994–995, 2003). In healthy individuals, stepwise increases in GL have been shown to predict stepwise elevations in postprandial blood glucose and/or insulin levels (Brand-Miller JC et al, 2003). It can be seen from the equation that either a low-GI/high-carbohydrate food or a high-GI/low-carbohydrate food can have the same GL. However, while the effects on postprandial glycemia may be similar, there is evidence that the two approaches will have very different metabolic effects, including differences in  $\beta$ -cell function, triglyceride concentrations, free fatty acid levels (Wolever TMS, et al. 2002), and effects on satiety (Ball SD, et al. 2003).

Hence, the distinction has important implications for the prevention and management of diabetes and cardiovascular disease. Our concern is that the use of the GL or "glycemic response" in isolation may lead to the habitual consumption of lower-carbohydrate diets.

The use of the glycemic index has been shown to provide additional benefit to glycemic control over that observed when total carbohydrate is considered alone (Brand-Miller. et al. 2003). This index compares glycaemic excursions after ingestion of a carbohydrate and compares it with the glycemic excursions after an equivalent amount of the monosaccharide glucose. Thus numerical values can be ascribed to potatoes, rice, bread, etc., which give a comparative indication of glycemic consequence.

Factors that affect the glycemic response of foods are feeding rate, the rate of food ingestion, food ingredients (fat, protein, fiber, starch) and methods of cooking and food processing. Influence on glycemic response and physiological mechanisms of degradation of consumed food (pre-gastric and gastric hydrolysis, gastric emptying rate, intestinal hydrolysis and reaction to pancreatic and intestinal hormones). Bread, crackers, grain, potatoes, millet, corn, and chips have a high GI (> 90). Bran, oatmeal, rice, buckwheat have medium glycemic index (e.g. 70-90). Black bread, pasta, barley and cooked rice have the lowest glycemic index(<70) (Dimitrijević- Srećković V. 2002).

A controlled study in children using the GI of foods found flexible dietary instruction based on the food pyramid and low-GI choices achieved significantly better glycemic control after 12 months than more traditional dietary advice. (Gilbertson H. et al. 2001). In their study, Miller JB et al confirmed the significant influence of the lower GI nutrition on postprandial glucose levels. However, the impact on long-term glycemic control and co-morbidity was less efficient than pharmacological treatment.

Choosing low-GI foods in place of conventional or high-GI foods has a small but clinically useful effect on medium-term glycemic control in patients with diabetes. The incremental benefit is similar to that offered by pharmacological agents that also target postprandial hyperglycemia. (Jennie Brand-Miller et al. 2003)

In addition, several prospective observational studies have found that the overall GI and glycemic load (GI × g carbohydrate) of the diet, but not total carbohydrate content, are independently related to the risk of developing type 2 diabetes (Salmeron J et al, 1997), cardiovascular disease (Liu S et al, 2000), and some cancers (Augustin L, 2001, Franceschi S et al, 2001)

Low GI carbohydrate foods (GI < 55) may lower post-prandial hyperglycemia when they are chosen to replace higher GI foods (GI > 70) (Brand-Miller J. et al. 2003) Examples of low GI food sources include wholegrain breads, pasta, temperate fruits and dairy products. (Foster-Powell K. et al. 2002) Glycemic load (GL) is another method of predicting the postprandial blood glucose response, which takes into account both the GI of the food and the portion size. (Colombani PC, 2004). There has been no assessment of its efficacy in children.

Artificial sweeteners are widely used among diabetic patients. Two kinds of sweeteners may be distinguished:nutritive sweeteners which contain calories (fructose, sorbitol, mannitol) and non-nutritive which are calories-free (saccharin, cyclamate, aspartame). Fructose has the advantage over sucrose; for its better taste, slow absorption from the digestive system; no insulin is required for itsutilization and it causes hyperglycemia less often The fructose intake should be limited to 25g/d. Saccharin is about 500 times the sweeter than sucrose; its use may be connected to the increased risk of bladder carcinoma (Dimitrijevic-Srećković V., 2002).

#### 5. Fat

The proportion of fat content in total energy intake should be approximately 35% in young children, and 25-30% in older children.

The vegetable fats have clearly advantage over animal ones. Intake decreases during child-hood from approximately 2 g/kg/day in early infancy to 1 g/kg/day for a ten year old and to 0.8–0.9 g/kg/day in late adolescence. (Kauffman FR, 2005). Substituting butter with margarine, vegetable oil for animal oil, and lean cuts of meat, poultry, and fish for fatty meats, such as bacon, is advisable. These simple measures reduce serum low-density lipoprotein cholesterol, a predisposing factor to atherosclerotic disease.

Uncontrolled type I diabetes is associated with elevated plasma lipids, but adequate insulin therapy usually restores lipid levels to normal. People with type I diabetes who are treated with insulin generally have plasma cholesterol, VLDL cholesterol and triglyceride concentrations similar to those of the general population of the same age and sex (Kern P, 1987). Although not all studies agree, it appears that blood glucose control may directly influence the levels of several plasma lipid components. Qualitative abnormalities such as changes in a density of lipoprotein composition may exist even when the usual clinical measurements of plasma lipids are normal (Dunn FL, 1992). Evidence that dietary fat and the development of atherosclerosis are linked is controversial and there is little sign that a reduction in dietary fat would reduce atherosclerotic disease. Epidemiological studies from Japan are often quoted and in other populations a fall in cardiovascular morbidity has coincided with alterations in eating habits. (Nattras M., 1996) The dietary contents of polyunsaturated / saturated fatty acids should optimally correspond to 1.2: 1.0 ratios.

The primary goal regarding management of dietary fat is to decrease the intake of total fat, saturated fat, and trans-fatty acids (Franz MJ et al, 2002). Monounsaturated fatty acids (MU-FA) and polyunsaturated fatty acids (PUFA) can be used as substitutes to keep lipid intake within recommended ranges or to improve the lipid profile.

Polyunsaturated fatty acids increase the production of lipid peroxides; some experimental studies considered their influence on occurrence of certain malignancies. They are rich in omega-6 fatty acids from sunflower oil and corn; moderate intake of these fatty acids lowers LDL cholesterol, while high intake lowers HDL cholesterol. Omega-3 fatty acids are found in deep sea blue fish (herring, mackerel, tuna and salmon). Their utilization from fish meat is more effective than the utilization from pharmacological supplements. The recommended intake of fish oil is 1.5-2.0 g / day. Omega-3 fatty acids moderately lower total cholesterol, significantly reduce triglyceride levels and reduce platelet aggregation, blood pressure and cardiovascular risk. However, they may increase hepatic glucose production, thus increasing blood glucose and hemoglobin A1-c levels. Therefore, in patients with impaired carbohydrate metabolism, the use of fish oil is not recommended; instead, blue sea fish meals should be taken 2-3 times a week.

Monounsaturated fatty acids are found in olive oil, walnuts and sesame. Studies have shown that application of these fatty acids for one month reduces insulin requirements and improve insulin sensitivity; decreases blood glucose and triglyceride levels and arterial blood pressure as well. The Mediterranean diet, with increased use of olive oil-rich omega-9 fatty acids, reduces LDL cholesterol without affecting HDL cholesterol. Its intake improves insulin sensitivity, blood glucose and triglyceride levels and blood pressure. These mechanisms explain the reduction in coronary heart disease in Mediterranean countries. Monounsaturated fatty acids are more resistant to lipid peroxidation than the polyunsaturated fatty acids and less involved in the pathogenesis of atherosclerosis.

Trans-isomers of polyunsaturated fatty acids formed during the hydrogenation process may have potentially harmful effects. These trans-isomers are formed during solidification of vegetable oils. They are found in margarine and pastries, and intake of these foods increases LDL cholesterol and lowers HDL cholesterol. Therefore, in the UK and some European countries the intake of these trans-isomers in the amounts more than 5 g / day is not recommended. A consideration was given on intake of long chain omega-3 fatty acids. Epidemiological research studies have shown that prolonged use of concentrated fish oil may have an anti-atherogenic effect thanks to the high omega-3 acid contents (A. Simopoulos, 1991).

The existing nutritional recommendations of the European Association for the Study of Diabetes (EASD) and American Diabetes Association (ADA) on dietary composition promote greater flexibility in the proportions of energy derived from carbohydrate and from monounsaturated fat (MUFA). MUFA are promoted as the main source of dietary fat because of their lower susceptibility of lipid peroxidation and consequent lower atherogenic potential (Kratz M., et al. 2002).

In diabetes, cholesterol intake is limited to a maximum of 300 mg daily, in order to slow down the development of atherosclerotic process. This means that cholesterol-rich foods (brain, egg yolk) should be avoided, and lean meat (veal, beef, chicken) or fish should be used instead; instead of whole milk a milk containing 2.8% fat.

#### 6. Proteins

Proteins are an essential nutrient, necessary for normal growth and development in childhood. Adequate protein ingestion is critical in normal muscle development. Proteins are an essential source of nitrogen.

The recommended intake is 15% of total caloric daily intake in older children and 20% in younger. The intake of proteins per kilogram of body weight should be higher in infants, children and adolescents in comparison to adults in order to support growth and development. The daily requirements are about 1.5g/kg for preschool children for and somewhat less -1g/kg for the children in school age -e.g., until the period of rapid growth during puberty, when the requirements increase again. (Stepanović R. et al 1991)

In diabetes variation in dietary protein may influence metabolic control by altering gluconeogenic substrate availability as well as insulin and contra-regulatory hormone secretion (Nuttall FQ., 1983).

Only in poorly controlled diabetes or in a period of recovering from ketoacidosis, the amount of protein should be greater than 2g/kg. The most important sources of protein are meat, fish and egg whites, but proteins are also represented in foods rich in carbohydrates (legumes, bread and cereals)

Excessive protein intake has also been implicated in the pathogenesis of diabetic renal disease and restricting its intake may retard the progression of nephropathy (Brenner BM., et al 1982)

The caloric mixture should comprise approximately 55% carbohydrate, 30% fat and 15% protein. A daily intake should be divided in 6-7 meals; breakfast and lunch should be represented with 20% of total caloric needs, dinner with 30% and each snack should contain 10% of daily calorie inputs. Each meal should be taken at certain time during the day with no major or frequent deviations. A bed time snack is considered an essential part of the regimen. This is necessary to prevent nocturnal hypoglycemia. The bed time snack includes at least 7-8g of protein, the amount equivalent to that in a meat or a milk exchange (7 to 8g). (Rudolph AM et al 1996)

#### 7. Fiber

Dietary fibers may be divided into soluble (found in fruits, oats, barley, legumes and root vegetables) and insoluble (found in wheat, wheat bran, grains and some vegetables). Soluble fibers are mainly hemi-cellulose compounds that bind water, bile acids and build sequestered forms with monosaccharides and disaccharides; thus, they partially inhibit the action of digestive enzymes and slow down the process of absorption of food by increasing the time of intestinal passage. This action lowers postprandial glycemia and total cholesterol levels. These fibers also reduce the levels of lipids, cholesterol, LDL cholesterol and body mass. Soluble fiber supplements such as pectin, fiber from carob can improve metabolic regulation. Insoluble dietary fibers have little effect on blood glucose and no effects on lipids, but may increase satiety and inhibit hunger, and thus induce positive effects in obese patients with diabetes (Dimitrijevic-Srećković V., 2002).

#### 8. Micronutrients: Vitamins and minerals

By complying with a proper and balanced diet, people with diabetes daily take adequate amount of vitamins and minerals, so there is usually no need for pharmacological supplements. The exceptions are some trace elements-copper, selenium and magnesium.

Individuals on weight –reducing diet, strict vegetarians patients with poor metabolic control and patients in critical care environments require special attention and assessment. A supplementation therapy containing vitamins and minerals is most often needed in these cases. In animal models it was shown that chromium deficiency was often associated with elevated blood glucose, cholesterol and triglyceride levels (Schrroeder HA, 1966; Wolscroft J et al,

1977). While serum zinc levels are generally lower in people with diabetes, zinc replacement is only suggested to be of benefit in helping to heal venous leg ulcer (Hoolbook T., et al 1979). There may be a need for magnesium replacement in patients with poor glycemic control or who are on diuretics. Magnesium depletion has been associated with decreased insulin sensitivity, which may improve with oral supplementation (Beaugerie L et al, 1990).

Some studies indicated that magnesium is a novel factor implicated to the pathogenesis of the diabetic complications. Magnesium plays a fundamental role as a cofactor in various enzymatic reactions of energy metabolism. Magnesium is a cofactor in cell membrane glucose - transporting mechanisms, as well as in various enzymes in carbohydrate oxidation. It is also involved, at multiple levels, in insulin secretion, binding and activity. Magnesium deficit has been described in patients with type I diabetes. Hypomagnesemia can also be the cause or a result of diabetes complications. If it is followed by diabetes, osmotic dieresis may play a role in the mechanisms responsible for magnesium deficiency. Magnesium loss may be linked to the development of diabetes complications via a reduction in the rate of inositol transport and its subsequent intracellular depletion that might enhance the development of complications. Magnesium is also taking part as a cofactor in many enzymes which are involved in lipid metabolism. Magnesium administration could decrease triglyceride, cholesterol and LDL cholesterol levels and also increase HDL cholesterol (Soltani N., 2011).

#### 9. Sodium

The American Heart Association recommends that sodium intake should not exceed 3000 mg/day, while other authors (National High Blood Pressure Education Program, 1993) recommend not more than 2400mg/day. Individuals with mild to moderate hypertension should ingest no more than 2400mg of sodium daily (or less than 6 g/day of sodium chloride). A study performed in mildly hypertensive subjects with diabetes on moderate dietary sodium restriction showed a reduction of approximately 20 mmHg in systolic blood pressure. A difference in diastolic blood pressure was not achieved (Dodson PM., et al 1989).

Routine supplementation with antioxidants, such as vitamins E and C and carotene, is not advised because of lack of evidence of efficacy and concern related to long-term safety.

#### 10. Recommended foods

A dietary intake of dark bread, rye, whole-meal bread, oats and barley flakes, porridge of maize flour, rice, spaghetti, potatoes, beans and lentils as a substitute for bread is recommended. Also, use of all kinds of vegetables (legumes, root and leafy vegetables) in the amount of 400-500 g/d (250g boiled, 250g fresh). All kinds of fruits are allowed, except for grapes, figs, prunes. The amount of fruit should be 500 g per day, divided into several installments. In addition, fresh fruit has an advantage over the pressed juice, since it is rich in dietary fibers.

Component	Comment	
Protein	Not"/> 1g per body weight	
Total fat	< 35% of energy intake	
Saturated+trans-unsaturated fat	<10% of energy intake	
n-6 Polyunsaturated fat	<10% of energy intake	
n-3 Polyunsaturated fat	Eat fish, especially oily fish, once or twice weekly. Fish oil	
cis-Monounsatureted fat	supplements-not recommended 10-20% ( 60-70%) energy intake	
Total carbohydrate	45-60%	
Sucrose	Up to 10% of daily energy requirements, provided it is eaten within the healthy diet. Those who are overweight or who have hypertriglyceridemia should consider using not-nutritive sweeteners where appropriate	
Fiber	No quantitative recommendation  Soluble fiber"/> has beneficial effect on glycaemic and lipid metabolism  Insoluble fiber"/> no direct effect on glycaemic and lipid metabolism but its high satiety content may benefit thos trying to lose weight and its advantageous to gastrointestinal health	
Vitamins and anti-oxidants	Encourage foods naturally rich in vitamins and anti- oxidants. With exception of some patients e.g. malnourishment, cancer etc. there is no evidence for the use supplements and some evidence that some are harmful	
Salt	Approx 6 g sodium chloride per day	

**Table 2.** (Connor H. et al. 2003)

The diet may contain dairy products - a skim milk, buttermilk, yogurt with 3.2% milk fat, cream, sour cream, butter, fatty cheeses and cheese. Lean meat (chicken, turkey, veal, beef, lamb, horse, deer, fish-river and sea), and cured meats from chicken, horse sausages, and sardines drained of oil are also recommended. Pork, duck, goose, sheep meat, fatty fish (catfish, perch, carp) should be avoided. The recommended intake is two poached eggs a week (in people with high cholesterol and triglyceride levels intake of yolk is not allowed). The use of vegetable fats is preferable (olive and sunflower oil) (Srećković V. Dimitrijevic, 2002).

The intake of starch should be provided by eating bread, grains, cereal, pasta, and starchy vegetables like corn and potatoes. They provide carbohydrate, vitamins, minerals, and fiber. Whole grain starches are healthier because they have more vitamins, minerals, and fiber.

The UK DAFNE Project (Dose Adjustment For Normal Eating) includes adults with type 1 diabetes participating in a group program. It is a kind of a group skills-based training with the aim to provide knowledge of flexible insulin meal-by-meal adjustments in order to match the carbohydrate content in a free diet. The groups consist of adult participants with type 1 diabetes of>2 years duration, without advanced complication, with HBA1c levels of 7.5-12%. DAFNE involves attending a 5-day training course plus a follow-up session around 8 weeks after the course and yearly half-day top-up sessions. The structured teaching program is delivered to groups of 6-8 participants and covers topics including carbohydrate estimation, blood glucose monitoring, insulin regimens, hypoglycemia, illness and exercise. A significant reductions in HbA1-c were found after 6 (-1.0%) and 12 months (-0.5%) of intervention, respectively. Also, a significant improvement in quality of life and well-being and satisfaction with treatment scores was registered (Nutrition Sub-Committee of the Diabetes Care Advisory Committee of Diabetes UK, 2003).

Exchange	Calories	Grams of	Grams of Protein	Grams of Fat
		Carbohydrate		
Bread/starch	80	15	3	Trace
Fruit	60	15	-	-
		Meat		
Lean	55	7	3	-
Medium fat	75	-	7	3
High fat	100	-	7	8
Milk				
Skim	90	12	8	Trace
Low fat	120	12	8	5
Whole	150	12	8	8
Fat	45	-	-	5
Vegetable	25	5	2	-

**Table 3.** Nutrient composition of the exchanges

## 11. Nutrition recommendations for controlling diabetes complications

If there was a family history of hypercholesterolemia (total cholesterol >240 mg/dl) or a family cardiovascular event before age of 55 years, or if family history was unknown, a fasting lipid profile should be performed on children >2 years of age soon after diagnosis (after glucose control has been established). If family history is not of concern, then the first lipid screening should be considered at puberty (≥10 years). All children diagnosed with diabetes at or after puberty should have a fasting lipid profile performed soon after diagnosis (after glucose control has been established

Initial therapy should consist of optimization of glucose control and MNT using a Step 2 AHA diet (American Heart Association, 2010 Dietary Guidelines) aimed at a decrease in the amount of saturated fat in the diet. People diagnosed with type 1 diabetes in childhood have a high risk of early subclinical (Krantz JS et al, 2004, Järvisalo MJ et al, 2004, Haller MJ., 2004), and clinical (Orchard TJ., et al. 2001) CVD. Although intervention data are lacking, the AHA categorizes children with type 1 diabetes in the highest cardiovascular risk group and recommends both lifestyle and pharmacologic treatment for those with elevated LDL cholesterol levels (Kavey RE et al, 2006; McCrindle BW et al, 2007). Initial therapy should be with a Step 2 AHA diet, which restricts saturated fat to 7% of total calories and restricts dietary cholesterol to 200 mg/day. Data from randomized clinical trials in children as young as 7 months of age indicate that this diet is safe and does not interfere with normal growth and development

A proper nutrition and physical activity is essential for the prevention of arterial hypertension in diabetic patients. Hypertension, which is predictive of progression of micro- as well as macrovascular complications of diabetes, can be prevented and managed with interventions including weight loss, physical activity, moderation of alcohol intake, and diets such as DASH (Dietary Approaches to Stop Hypertension). The DASH diet emphasized fruits, vegetables, and low-fat dairy products; included whole grains, poultry, fish, and nuts; and was reduced in fats, red meat, sweets, and sugar-containing beverages Treatment of high-normal blood pressure (systolic or diastolic blood pressure consistently above the 90th percentile for age, sex, and height) should include dietary intervention and exercise aimed at weight control and increased physical activity, if appropriate(Chobanian AV., et al. 2003, Sacks FM., et al. 2001, Appel LJ., et al. 2006).

If target blood pressure is not reached with 3–6 months of lifestyle intervention, pharmacologic treatment should be considered (Standards of Medical Care in Diabetes—2012)

In individuals with diabetes and macroalbuminuria, reducing protein from all sources to 0.8 g/kg body wt/ day has been associated with slowing the decline in renal function (Franz MJ., et al 2002, Hansen H., et al 2002); however, such reductions in protein need to maintain good nutritional status in patients with chronic renal failure (Meloni C., et al 2002)

Although several studies have explored the potential benefit of plant proteins in place of animal proteins and specific animal proteins in diabetic individuals with microalbuninuria, the data are inconclusive. (Wheeler ML., et al 2002)

Medical nutrition therapy is important in preventing diabetes, managing existing diabetes, and preventing, or at least slowing, the rate of development of diabetes complications (Nutrition Recommendations and Interventions for Diabetes, 2008).

#### 12. Nutritional management of exercise and sport

A regular physical activity improves glucose tolerance, increases muscle mass, reduces body fat mass and increases the number of insulin receptors and glucose entry into cells. A blood flow to the muscle and expansion of capillary space are stimulated during physical activity, which allows a better flow of insulin to muscle cells. (Zergollern Lj., et al., 1994) Regular

physical exercise improves insulin resistance and reduction of triglyceride levels and increases total HDL and HDL2 concentration; it also helps in lowering blood pressure. It reduces mortality in type I diabetes and may reduce HbA1c up to 0.7% in type II diabetes (Ha TKK. et al., 1998).

The U.S. Department of Health and Human Services' Physical Activity Guidelines for Americans (U.S. Department of Health and Human Services, 2008), suggest that adults over age 18 years have up to 150 min/week of moderate-intensity, or 75 min/week of vigorous aerobic physical activity, or an equivalent combination of the two. In addition, the guidelines suggest that adults also do muscle-strengthening activities that involve all major muscle groups two or more days per week. Studies included in the meta-analysis of effects of exercise interventions on glycemic control (Boulé NG. et al., 2001). The DPP lifestyle intervention, which included 150 min/week of moderate intensity exercise, had a beneficial effect on glycemia in those with prediabetes. Therefore, it seems reasonable to recommend that people with diabetes try to follow the physical activity guidelines for the general population.

In patients who are in poor metabolic control, vigorous exercise may precipitate ketoacidosis because of the exercise-induced increase in the counter-regulatory hormones. On the other hand, there is an excessive amount of insulin in the body, the hepatic glucose production in response to excess muscle consumption could be insufficient, and symptoms of hypoglycemia might ensue.

When people with type 1 diabetes are deprived of insulin for 12–48 h and becomeketotic, exercise can worsen hyperglycemia and ketosis (Berger M, Berchtold P., et al., 1977); therefore, vigorous activity should be avoided in the presence of ketosis. However, it is not necessary to postpone exercise based simply on hyperglycemia, provided the patient feels well and urine and/or blood ketones are negative.

In individuals taking insulin and/or insulin secretagogues, physical activity can cause hypoglycemia if medication dose or carbohydrate consumption is not altered. For individuals on these therapies, additional carbohydrate should be ingested if pre-exercise glucose levels are <100 mg/dl (5.6mmol/l). Hypoglycemia is rare in diabetic individuals not treated with insulin or insulin secretagogues, and no preventive measures for hypoglycemia are usually advised in these cases.

It is therefore desirable that every person with diabetes planning or having physical exercise carry a glucose or sacharose preparation in form of tablets, jelly or candy or sugar in the form of cubes, candy, juice or soda. If hypoglycemias occur frequently, the entire dose insulin should be reduced by 10-15%.

The enhanced energy expenditure resulting from exercise increases the need for additional protein which should be met by increased consumption of nutritionally balanced diet. A small amount of additional protein may be required for muscle growth resulting from chronic physical conditioning.

Physical activity can acutely increase urinary protein excretion. However, there is no evidence that vigorous exercise increases the rate of progression of diabetic kidney disease, and

there is likely no need for any specific exercise restrictions for people with diabetic kidney disease (Mogensen CE., 2002).

#### 13. Conclusion

In conclusion, dietary recommendations for diabetic patients should be based on healthy eating recommendations suitable for all children and adults. Generally, the ideal diet for the normalization of glycemic control in people with diabetes has not yet been designed. In fact, there may be as many diets for diabetes as people with diabetes, and they could be based on the many individual manifestations, presentations, and complications of the disease. Individuals on insulin therapy need to eat at consistent times synchronized with the time-actions of insulin, monitor blood glucose levels, and adjust insulin doses for the amount of food usually eaten.

Medical nutrition therapy is important in preventing diabetes, managing existing diabetes, and preventing, or at least slowing, the rate of development of diabetes complications. No nutrition recommendations can be made for the prevention of type 1diabetes at this time. Although there are insufficient data at present to warrant any specific recommendations for the prevention of type 2 diabetes in youth, interventions similar to those shown to be effective for prevention of type 2 diabetes in adults are likely to be beneficial.

The caloric mixture should comprise approximately 55% carbohydrate, 30% fat and 15% protein. A flexible dietary instruction based on the food pyramid and low-glycemic index choices achieved significantly better glycemic control than more traditional dietary advice.

Several prospective observational studies have shown that the overall glycemic index and glycemic load of the diet, but not total carbohydrate content, are independently related to the risk of developing type 2 diabetes. An intake of simple carbohydrates with high fiber foods - such as complex grains, vegetables and fruit slows the intestinal absorption. It also reduces postprandial hyperglycemia and lowers serum cholesterol levels.

The primary goal regarding dietary fat is to decrease the intake of total fat, saturated fat, and trans-fatty acids. Monounsaturated fat should be promoted as the main source of dietary fat because of their lower susceptibility to lipid peroxidation and consequent lower atherogenic potential. Moderate intake of omega 3 and omega-9 fatty acids lowers LDL cholesterol.

If there was a family history of hypercholesterolemia or a family cardiovascular event before age of 55 years, or if family history was unknown, a fasting lipid profile should be performed on children >2 years of age soon after diagnosis (after glucose control has been established). All children diagnosed with diabetes at or after puberty should have a fasting lipid profile performed soon after diagnosis (after glucose control has been established. AHA categorizes children with type 1 diabetes in the highest for cardiovascular risk and recommends both lifestyle and pharmacologic treatment for those with elevated LDL cholesterol levels.

Proteins are an essential nutrient, necessary for normal growth and development in childhood. The recommended intake is 15% of total caloric daily intake in older children and 20% in younger children. The daily requirements are about 1.5g/kg for preschool children for and somewhat less -1g/kg for the children in school age -e.g., until the period of rapid growth during puberty, when the requirements increase again. Only in poorly controlled diabetes or in a period of recovering from ketoacidosis, the amount of protein should be greater than 2g/kg. Variable dietary proteins may influence metabolic control by altering gluconeogenic substrate availability as well as insulin and contra-regulatory hormone secretion. Excessive protein intake has also been implicated in the pathogenesis of diabetic renal disease. In individuals with diabetes and macroalbuminuria, reducing protein from all sources to 0.8 g • kg body wt-1 • day-1 has been associated with slowing the decline in renal function.

Hypertension can be prevented and managed with interventions including weight loss, physical activity, moderation of alcohol intake, and diets such as DASH (Dietary Approaches to Stop Hypertension. 2006). If target blood pressure is not reached with 3–6 months of lifestyle intervention, pharmacologic treatment should be considered. Insoluble dietary fibers have little effect on blood glucose and no effects on lipids, but they may increase satiety and inhibit hunger, thus inducing positive effects in obese diabetic patients.

By complying with a proper and balanced diet, people with diabetes take adequate amount of vitamins and minerals, so there is usually no need for pharmacological supplements. Zinc replacement is only suggested to be of benefit in helping to heal venous leg ulcer.

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#### References

- [1] American Diabetes Association. Nutrition principles and recommendations in diabetes (Position Statement). Diabetes Care 2003: 26(Suppl. 1): S51–S61.
- [2] Appel LJ, Brands MW, Daniels SR, Karanja N, Elmer PJ, Sacks FM: Dietary approaches to prevent and treat hypertension: a scientific statement from the American Heart Association. 2006, Hypertension 47:296–308
- [3] Augustin L: Dietary glycemic index and glycemic load in breast cancer risk: a case control study. Ann Oncol 12:1533–1538, 2001

- [4] Ball SD, Keller KR, Moyer-Mileur LJ, Ding YW, Donaldson D, Jackson WD: Prolongation of satiety after low versus moderately high glycemic index meals in obese adolescents. Pediatrics, 2003: 111: 488-494
- [5] Beaugerie L, Flourie B, Marteau P, Pellier P, et al. Digestion and apsorption in the human intestine of three sugar alcohols. Gastroenterology 99: 717-723, 1990.
- [6] Berger M, Berchtold P, Cüppers HJ, DrostH, Kley HK, Müller WA, Wiegelmann W, Zimmerman-Telschow H, Gries FA, Krüskemper HL, Zimmermann H. Metabolic and hormonal effects of muscular exercise in juvenile type diabetics. Diabetologia 1977; 13: 355–365
- [7] Boulé NG, Haddad E, Kenny GP, Wells GA, Sigal RJ. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. JAMA 2001; 286: 1218-1227
- [8] Brand-Miller, Hayne S, Petocz P, Colagiuri S. Low-Glycemic Index Diets in the Management of Diabetes. A meta-analysis of randomized controlled trials. Diabetes Care 2003: 26: 2261–2267.
- [9] Brand-Miller J, Hayne S, Petocz P, Colagiuri S. Low-Glycemic Index Diets in the Management of Diabetes. A meta-analysis of randomized controlled trials. Diabetes Care 2003: 26: 2261–2267
- [10] Brand-Miller JC, Thomas M, Swan V, Ahmad ZI, Petocz P, Colagiuri S: Physiological validation of the concept of glycemic load in lean young adults. J Nutr. 2003: 133: 2728-2732
- [11] Brenner BM, Mezer TW: Dietary protein intake and the progressive nature of kidney disease: the role of hemodynamically mediated glomerular injury in the pathogenesis of progressive glomerular sclerosis in aging renal ablation and intrinsing renal disease. N eng. J. Med 307:652-659, 1982
- [12] Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ: The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA 289:2560–2572, 2003
- [13] Colombani PC. Glycemic index and load-dynamic dietary guidelines in the context of diseases.PhysiolBehav. 2004: 83(4): 603–10
- [14] Connor H., Annan F., bunn E et al., The implementation of nutritional advice for people with diabetes. Diabetic Medicine 2003, Vol 20 p. 786-807
- [15] Dimitrijević- Srećković V.: Dijetetskolečenje Tip I tip II dijebetesa, Dijabetes mellitus, inovacije znanja,2002: 57-65
- [16] Dodson PM, Beevers M, Hallworth R, et al. Sodium restriction and blood pressure in hypertensive type 2 diabetes: randomized blind controlled and crossover studies of

- moderate sodium restriction and sodium supplementation. Br Med J 298: 226-230, 1989
- [17] Dunn FL: Plasma lipid and lipoprotein disorders in IDDM. 1992. Diabetes 41 (Suppl. 2): 102-106, 1992
- [18] Foster-Powell K, Holt SH, Brand-Miller JC.International Table of Glycemic Index and Glycemic Load Values. Am J Clinical Nutrition 2002: 76: 5–56.
- [19] Franceschi S, Dal Maso L, Augustin L, Negri E, Parpinel M, Boyle P, Jenkins DJ, La Vecchia C: Dietary glycemic load and colorectal cancer risk. Ann Oncol 12:173–178, 2001)
- [20] Frany J. Marion, Horton S. Edwards, Bantle P John, et al. Nutrition Principles for the Management of Diabetes and Related Complications; Diabetes care, 17, No5, 1994, 490-518
- [21] Franz MJ, Bantle JP, Beebe CA, Brunzell JD, Chiasson JL, Garg A, Holzmeister LA, Hoogwerf B, Mayer-Davis E, Mooradian AD, Purnell JQ, Wheeler M: Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. Diabetes Care, 2002: 25: 148–198
- [22] Franz MJ, Bantle JP, Beebe CA, Brunzell JD, Chiasson JL, Garg A, Holzmeister LA, Hoogwerf B, Mayer-Davis E, Mooradian AD, Purnell JQ, Wheeler M: Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. Diabetes Care 25: 148–198, 2002
- [23] Gilbertson H, Brand-Miller J, Thorburn A et al. The effect of flexible low Glycemic index dietary advice versus measured carbohydrate exchange diets on glycemic control in children with type 1 diabetes. Diabetes Care 2001: 24: 1.
- [24] Grundy SM, Balady GJ, Criqui MH, Fletcher G, Greenland P, Hiratzka LF, Houston-Miller N, Kris-Etherton P, Krumholz HM, LaRosa J, Ockene IS, Pearson TA, Reed J, Smith SC Jr, Washington R: When to start cholesterol-lowering therapy in patients with coronary heart disease: a statement for healthcare professionals from the American Heart Association Task Force on Risk Reduction. Circulation 1997: 95:1683–1685
- [25] Ha TKK, Lean MEJ. Technical Review.Recommendation for the nutritional menagment of patient with diabetes. Eur. J ClinNutrituion 1998; 52: 647-481
- [26] Haller MJ, Samyn M, Nichols WW, et al. . Radial artery tonometry demonstrates arterial stiffness in children with type 1 diabetes. Diabetes Care 2004; 27: 2911–2917
- [27] Hansen HP, Tauber-Lassen E, Jensen BR, Parving HH: Effect of dietary protein restriction on prognosis in patients with diabetic nephropathy. Kidney Int 62:220–228, 2002
- [28] Hoolbook T, Lanner E: serum zinc and healing of leg ulcer.1979, Lancet 2: 708-712

- [29] Institute of Medicine: Dietary Reference Intakes: Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. Washington, DC, National Academies Press, 2002
- [30] Järvisalo MJ, Putto-Laurila A, Jartti L, et al.. Carotid artery intima-media thickness in children with type 1 diabetes. Diabetes 2002; 51: 493–498;
- [31] Jennie Brand-Miller, Susan Hayne, Peter Petocz, Stephen Colagiuri Low-Glycemic Index Diets in the Management of Diabetes, A meta-analysis of randomized controlled trial, Diabetes Care, vol. 26 no. 8, 2003 : 2261-2267
- [32] KAUFMANN FR. Surveillance and treatment for CVD in children with diabetes.ADA, Docnews 2005.
- [33] Kavey RE, Allada V, Daniels SR, et al. American Heart Association Expert Panel on Population and Prevention Science, American Heart Association Council on Cardiovascular Disease in the Young, American Heart Association Council on Epidemiology and Prevention, American Heart Association Council on Nutrition, Physical Activity and Metabolism, American Heart Association Council on High Blood Pressure Research, American Heart Association Council on Cardiovascular Nursing, American Heart Association Council on the Kidney in Heart Disease, Interdisciplinary Working Group on Quality of Care and Outcomes Research. Cardiovascular risk reduction in high-risk pediatric patients: a scientific statement from the American Heart Association Expert Panel on Population and Prevention Science; the Councils on Cardiovascular Disease in the Young, Epidemiology and Prevention, Nutrition, Physical Activity and Metabolism, High Blood Pressure Research, Cardiovascular Nursing, and the Kidney in Heart Disease; and the Interdisciplinary Working Group on Quality of Care and Outcomes Research: endorsed by the American Academy of Pediatrics. Circulation 2006; 114: 2710-2738
- [34] Kern P: Lipid disorders in diabetes mellitus, MT Sinai J Med, 1987: 54: 245-252,
- [35] Kliegman M Robert, Behrman E Richard et al. Nelson Textbook of Pediatrics, The Endocrine system, Type 1 Diabetes mellitus ,2007: 2404-2425
- [36] Krantz JS, Mack WJ, Hodis HN, Liu CR, Liu CH, Kaufman FR. Early onset of subclinical atherosclerosis in young persons with type 1 diabetes. J Pediatr 2004; 145: 452– 457;
- [37] Kratz M., Cullen P., kannenberg F., Kassner M., et al. Effect of dietary fatty acids on the composition and oxidizability of low density lipoprotein. Europian Journal of Clinical Nutrition 2002 56: 72-81
- [38] Lean, M.E. J. And james, W.P.T. Prescription of diabetic diets in the 1980s. Lancet, i, 723-725.
- [39] Liu S, Willett WC, Stampfer MJ, Hu FB, Franz M, Sampson L, Hennekens CH, Manson JE: A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. Am J ClinNutr 71:1455-1461, 2000

- [40] McCrindle BW, Urbina EM, Dennison BA, et. Al.American Heart Association Atherosclerosis, Hypertension, and Obesity in Youth Committee, American Heart Association Council of Cardiovascular Disease in the Young, American Heart Association Council on Cardiovascular Nursing. Drug therapy of high-risk lipid abnormalities in children and adolescents: a scientific statement from the American Heart Association Atherosclerosis, Hypertension, and Obesity in Youth Committee, Council of Cardiovascular Disease in the Young, with the Council on Cardiovascular Nursing. Circulation 2007; 115: 1948–1967
- [41] Meloni C, Morosetti M, Suraci C, Pennafina MG, Tozzo C, Taccone-Gallucci M, Casciani CU: Severe dietary protein restriction in overt diabetic nephropathy: benefits or risks? J RenNutr 12:96–101, 2002
- [42] MogensenCE: Nephropathy. In Handbook of Exercise in Diabetes. 2nd ed. Ruderman N, Devlin JT, Kriska A Eds. Alexandria, VA, American Diabetes Association, 2002, p. 433–449
- [43] National Hight Blood Pressure Education Program: Working Group Report on Primary Prevention of Hypertension. Bethesda, MD, U.S. Department of Health and Human Service, National Institutes of Health 1993, p.14
- [44] Nattras M. Malims clinical diabetes:1996: Diet in Diabetes7: 117-132
- [45] NICE. Type 1 diabetes diagnosis and treatment of type 1 diabetes in children and young people, 2004: (www.nice.org.uk/pdf/type1diabetes).
- [46] Nutrition Recommendations and Interventions for Diabetes ;A position statement of the American Diabetes Association ;American Diabetes Association, Diabetes Care January 2008 vol. 31 no. Supplement 1 S61-S78
- [47] Nutrition Subcommitte of the Diabetes Care Advisory Committee of Diabetes UK; The Implementation of nutritional advice for people with diabetes, Diabetes Medicine, 20: 786-807, 2003
- [48] Nuttall FQ: Diet and the diabetic patient. Diabetes Care, 1983 6: 197-207,
- [49] Orchard TJ, Forrest KY, Kuller LH, Becker DJPittsburgh Epidemiology of Diabetes Complications Study. Lipid and blood pressure treatment goals for type 1 diabetes: 10-year incidence data from the Pittsburgh Epidemiology of Diabetes Complications Study. Diabetes Care 2001; 24: 1053–1059
- [50] Rudolph A.M. Hoffman J.I.E, Rudolph C.D, Rudolph's Pediatrics, Diabetes mellitus in children 1996,22:1803-1818
- [51] Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR, Miller ER III, Simons-Morton DG, Karanja N, Lin PH: Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet: DASH-Sodium Collaborative Research Group. N Engl J Med, 2001: 344:3–10,

- [52] Salmeron J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, Stampfer MJ, Wing AL, Willett WC: Dietary fiber, glycemic load, and risk of NIDDM in men. Diabetes Care 20:545-550, 1997
- [53] Salmeron J, Manson J, Stampfer M, Colditz G, Wing A, Willett W: Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA 1997:277:472-477
- [54] Salmeron J, Manson JAE, Stampfer MJ, Colditz GA, Wing AL, Jenkins DJ, Wing AL, Willett WC: Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA, 1997: 277: 472–477,
- [55] Schrroeder HA: Chromium deficiency in rats: a syndrome stimulating diabetes mellitus with retarded growth. J. Nutr: 88: 439-445, 1966
- [56] Simopoulos A: Omega-3 fatty acid in health and disease and in growth and development. Am J Clinnatur: 1991: 54:438-463
- [57] SoltaniNepton, Prevention of Diabetes Complications; Type I Diabetes; complications; 2011, 16: 353-366
- [58] Stamler J, Vaccaro O, Neaton JD, Wentworth D for the Multiple Risk factor Intervention Trial Research Group: Diabetes, other risk factors, and 12-yaer cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial, Diabetes Care, 1993:
- [59] Standards of Medical Care in Diabetes 2012, Diabetes Care, vol. 35, sup. S11-S63
- [60] Stepanović R., Nestorović B. I sar. : Ishrana u Pedijatriji :Ishranadeceoboleleodšećernebolesti . Medicinskaknjiga Beograd-Zagreb 1991.,218-221
- [61] Stern L, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams M, Gracely EJ, Samaha FF: The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. Ann Intern Med 140:778–785, 2004
- [62] Thom L.DiabetesAssociates.Nutritional management of diabetes., 1993 Mar;28(1): 97-112.
- [63] U.S. Department of Health and Human Services: 2008 Physical Activity Guidelines for Americans.[articleonline],2008.Availablefrom http://www.health.gov/paguidelines/guidelines/default.aspx. Accessed December 2010.
- [64] Wheeler ML, Fineberg SE, Fineberg NS, Gibson RG, Hackward LL: Animal versus plant protein meals in individuals with type 2 diabetes and microalbuminuria: effects on renal, glycemic, and lipid parameters. Diabetes Care 25:1277–1282, 2002
- [65] Wolever TMS, Mehling C: Long-term effect of varying the source or amount of dietary carbohydrate on postprandial plasma glucose, insulin, triacylglycerol, and free fatty acid concentrations in subjects with impaired glucose tolerance. Am J ClinNutr. 2002, 76: 5-56

- [66] Wolscroft J. Barbosa J: Analysis of chromium induced carbohydrate intolerance in the rat. J. Nutr.107: 1702-1706, 1977.
- [67] Yu-Poth S, Zhao G, Etherton T, Naglak M, Jonnalagadda S, Kris-Etherton PM: Effects of the National Cholesterol Education Program's Step I and Step II dietary intervention programs on cardiovascular disease risk factors: a meta-analysis. Am J Clin Nutr1999: 69:632–646
- [68] ZergollernLj., Reiner Ž. I sar. Pedijatrija 2, 1994: Dijabetes mellitus 17: 1319-1330



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