

# Diabetic food

The so-called **Diabetic food** can usually be found in supermarkets in so-called diabetic corners. This is an outdated concept. Foods labeled as "diabetic" usually contain energy value and carbohydrates, fats, in full, so we must include them in the value of the diet. They are not necessary for diabetics and may not be sought after. Diabetologists all over the world agree that these foods are inappropriate. The diabetic should choose a rational antisclerotic diet freely. He should avoid foods unsuitable for diabetics. **Suitable foods are mainly low-fat meats, low-fat dairy products, fresh fruits and vegetables.**

The concept of food for diabetics leads to a higher consumption of these foods. There are no sweets for diabetics and there are no cans or compotes and other foods suitable for diabetics. The intake of fresh fruits and vegetables is optimal. For others, the effort is to choose foods that do not lead to a high rise in blood sugar after a meal. More or less, these are foods with a so-called low glycemic index. In overweight type 2 diabetics, it is then appropriate to eat low-energy light foods, resp. drinks.



Poučování veřejnosti o orientaci v etiketě potravin, která informuje o obsahu cukru v potravině

## Diabetic food supplements

Although there are many theories that could justify the administration of vitamins or antioxidants in diabetes, their clinical effect has never been proven. Many studies with vitamin E have even turned out to be negative. The diet of a diabetic should undoubtedly include, in particular, **sufficient vegetables and a reasonable amount of fruit.** These substances are a natural source of antioxidants and some vitamins. Furthermore, it should be **varied**, it should include **dairy products**. Recently, supplements called vitamins for diabetics can be found in pharmacies. They cannot help the diabetic, and considering that the diabetic diet is expensive, it is a pity that diabetics spend money on something ineffective. Even the administration of unsaturated fatty acids is not metabolically justified. Because omega fatty acids have been shown to have cardiological effects (reduced mortality) and we know that virtually every type 2 diabetic has latent ischemic heart disease, dietary supplements with omega-fatty acids in the appropriate ratio of omega-3 and omega-6 are fully indicated. In many countries, it is even already covered by insurance companies (Solvay's Omacor).

In **diabetics type 2** with **present Atherosclerosis and** endothelial dysfunction, the intake of coffee and green tea, **is undoubtedly beneficial , where effects suppressing endothelial dysfunction have been demonstrated.**

## Glycemic index

Carbohydrates enter the bloodstream at different rates depending on the type of food and the type of carbohydrate present. As a result, the blood sugar (blood sugar level) can reach high levels after a meal, which are clearly harmful or insignificant. **If the sugar in the blood rises slowly, it is enough to be gradually transferred to the cells and therefore does not contribute to the development of diabetic complications** (atherosclerotic changes, damage to the eyes and kidneys). This property is called the **glycemic index - GI** in food . Every food containing sugars has its own GI.

In 1977, Jenkins described a reduction in postprandial glycemia after food fortification with viscous fiber or with less heat treatment. The decrease in postprandial glycaemia (postprandial glycaemia) was explained by an increase in food viscosity, intestinal fermentation, but also by the physical composition of the food. In 1982, in the journal Diabetologie, the same author described that foods rich in carbohydrates in the form of low-fiber starch have a different effect on postprandial glycemia in diabetics, but also in healthy individuals. These differences were explained by Jenkins at different rates of digestion. Other studies looking at glucose uptake by bolus or continuous food (sipping) of food or at different frequencies of food intake showed that the variations were probably more due to different glucose resorption.

**Postprandial glycemia**, the level of glycemia in healthy individuals and diabetics is a significant risk factor for ischemic heart disease. **it depends on two main factors:**

1. individualitě nemocného (inzulinová senzitivita, individuality of the patient (insulin sensitivity, beta-cell function, gastrointestinal motility, physical activity, digestion, absorption, utilization, oxidation of food intake, and daily variations of these factors);
2. food intake (amount, state, biological source and rate of digestion of polysaccharides, amount of sugars, fats, proteins, fiber, acidity of food and method of food preparation).

**The glycemic index of a diet is defined as the ratio of the area under the ascending part of the postprandial glycemia (postprandial glycaemia) curve of the test diet, which contains 50 g of carbohydrates and the standard diet.** The standard diet was initially 50 g of glucose, later glucose was replaced by white bread containing 50 g of carbohydrates, because the bread had less effect on gastric motility.

Despite a number of critical remarks, GI is becoming part of dietary recommendations in the treatment of diabetics. **Foods with a lower glycemic index are a prevention of diabetes**, but in diabetics the effect is very dependent on the total dose (load) of sugar taken (glucose load). The patient often reduces foods with a higher glycemic index, but then eats more foods with a lower glycemic index and the positive effect disappears.

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Glycemic indices of food related to glucose							
Foodstuff	GI	Foodstuff	GI	Foodstuff	GI	Foodstuff	GI
peanuts	14	low fat yoghurt	14	cooked peas	22	cherries	22
fructose	23	grapefruit	25	red boiled beans	27	whole milk	27
dried apples	29	boiled lentals	30	boiled beans	31	dried apricots	31
soy milk	31	boiled beans	33	low fat yoghurt with sugar	33	pear	38
apple	38	plums	39	unsweetend apple juice	40	cooked` spaghetti	41
peach	42	roasted muesli	43	pudding	43	lentil soup	44
orange	44	cooked macaroni	45	pineapple juice	46	grapes	46
lactose	46	sugar-free orange juice	48	sugar-free grapejuice	48	cooked green peas	48
chocolate	49	boiled carrots	49	oatmeal cooked with water	49	boiled oatmeal	49
pasta with cheese cooked	50	vanilla milk ice cream	50	kiwi	52	potato chips	54
banana	55	fruit compote	55	sterile sweetcorn, dried	55	muesli natural	56
white long grain rice	56	apricot	57	whole wheat pita bread	57	pizza cheese and tomatoes	60
cream ice cream	61	new boiled potatoes	62	old boiled potatoes	63	coca-cola	63
bean soup	64	raisins	64	Rye bread	65	pineapple	66
pea soup	66	croissant	67	whole wheat bread	69	white bread	70
mashed potatoes	70	corn chips	72	watermelon	72	rice	72
french fries	75	cornflakes	84	old baked potatoes	85	gluten free bread	90
baguette	95	glucose	100	candied fruit	103	maltodextrin	105

## Postprandial glycemia

Postprandial glycemia is clearly a significant factor. **It increases the risk of atherosclerosis** (eg heart attack) in diabetics and non-diabetics. It can be reduced by eating foods with a lower glycemic index, appropriate treatment of diabetes (well-chosen insulin or antidiabetics that can affect postprandial glycaemia, such as glinides). However, the specific glycemia achieved is decisive. As mentioned above, the total carbohydrate load is important and it is therefore not certain that a food with a lower glycemic index will always cause a smaller increase in postprandial glycaemia. Many authors also question the importance of glycemic indices, but they are definitely educational. The patient chooses more suitable foods. Postprandial glycaemia is affected by many individual factors, such as the rate of gastric emptying, food biting and bowel function.

## Hypoglycemia

Hypoglycemia is a **low blood sugar (< 3,2 mmol/l)**. It is a feared complication of diabetes treatment that needs to be prevented. Other causes of hypoglycaemia, such as insulin-producing tumors (insulinoma) or dumping syndrome, are rare.

The most common side effect with insulin treatment is hypoglycaemia. Iatrogenic-induced hypoglycemia is currently the most common acute complication of diabetes. We speak of chronic hypoglycemia when frequent, usually mild hypoglycemia recurs. Hypoglycemia is actually an arbitrarily determined biochemical laboratory value of glycemia lower than 3.5 mmol / l in capillary blood. The most common cause of hypoglycaemia is skipping, delaying or small amounts of food or increased physical exertion. Under these circumstances, the usual dose of insulin (or a sulphonylurea antidiabetic) is excessive and hyperinsulinemia causes hypoglycaemia. The risk of hypoglycaemia increases with decreased renal function and alcohol consumption, especially if food intake is reduced (hepatic glycogen depletion and inhibition of hepatic gluconeogenesis).

**acording to the severity of hypoglycemia we distinguish between asymptomatic, symptomatic mild** (the patient can handle it himself) **or symptomatic severe** (requires the intervention of another person). **The most severe degree of hypoglycemia is coma.**

### Clinical symptoms

**clinical signs of hypoglycemia** are caused by an adrenaline counter-regulatory reaction (they restore glycaemia by stimulating the release of glucose by the liver) and a lack of glucose in the brain (brain neuroglycopenia). Adrenergic symptoms predominate in the sudden onset of hypoglycemia, and symptoms caused by an increase in counter-regulatory hormones (especially glucagon and adrenaline) alert you to hypoglycemia in time. Mild hypoglycaemia is most often manifested by adrenergic symptoms (tremor, sweating, tachycardia, hunger, nervousness), very frequent hunger or faint tiredness or restlessness. If the onset of hypoglycaemia is slow, central symptoms are in the foreground (general weakness, decreased mental performance, lack of concentration, nausea, convulsions). In severe hypoglycemia, the diabetic may be confused, aggressive, and the environment may appear drunk. Severe hypoglycaemia may cause loss of consciousness.

## Mild hypoglycemia

**correction of mild hypoglycemia**, which they usually recognize quickly, patients usually manage themselves. They ingest fast-absorbing carbohydrates, which they should always carry with them, for example in the form of sugar cubes, concentrated juice or cola. About 15 grams of carbohydrates (3 sugar cubes, a glass of juice) are usually enough to normalize glycemia. To avoid delayed hypoglycemia, a diabetic must eat a meal that contains complex carbohydrates (preferably a slice of bread or croissant) after ingesting rapidly absorbable carbohydrates. Foods high in fat (such as chocolate) are unsuitable because they can slow down the digestion and absorption of carbohydrates. If the hypoglycaemia is so severe that the patient cannot swallow or cooperate, glucose or intramuscular glucagon must be given intravenously. Intravenous administration of 60 ml of 40% glucose (24 g) is usually sufficient to correct clinical hypoglycaemia, in children and adolescents, even smaller amounts will suffice. However, the diabetic must always eat after adjusting the state of consciousness. All diabetics should be provided with a card with basic information about their disease, including treatment. In unconscious patients, who do not know whether they are diabetic, the immediate determination of glycemia on a glucometer will decide on the next step. Physical signs (damp skin, normal skin turgor, superficial respiration, tachycardia, normal or high blood pressure) will help in the orientation whether it is a hypoglycemic or hyperglycemic coma. If the cause of the unconsciousness in a diabetic person is not clear, we must never give insulin as first aid. If hypoglycaemia is the cause of unconsciousness, insulin will make the condition much worse. Therefore, it is not a mistake in such a case to administer glucose, which is a causal treatment for hypoglycemia and will not significantly worsen the condition with existing hyperglycemia. To correct hypoglycemic coma, we repeatedly administer 60 ml of 40% glucose intravenously. If promptly corrected, the patient should be given an infusion of glucose, sometimes with corticosteroids.

## Hypoglycemic coma

**treatment of a deep prolonged hypoglycemic coma**, induced, for example, by treatment with sulfonylureas, antidiabetics, or in post-stroke conditions, may take days or longer. Rarely, neurological defects may be permanent after hypoglycaemia. Due to the possible consequences of prolonged hypoglycaemia, any hypoglycaemia must be treated immediately.

In some diabetics, the counterregulatory glucagon and later the adrenaline response to hypoglycemia (due to autonomic neuropathy and metabolic adaptation of brain cells to hypoglycemic states) is lost during diabetes. Patients lose the ability to recognize the usual warning signs of hypoglycaemia and suffer from so-called unexpected hypoglycaemia. In unexpected hypoglycemia that result from impaired perception of hypoglycemia, the diabetic is more at risk for possible complications of severe hypoglycemia. In the case of impaired consciousness, vomiting may occur, aspiration may occur, neurological focal symptoms and the consequences of hypokalaemia, which occurs when potassium is transferred from the extracellular space to the cells, may occur.

Patients who are more at risk of hypoglycaemia (for example, in an intensified insulin regimen) are equipped with kits containing 1 mg of glucagon. An informed relative or co-worker can then administer glucagon intramuscularly to a diabetic in severe hypoglycemia. This will increase blood glucose within 15-30 minutes and the patient can then correct the hypoglycaemia with food. Administration may be complicated by vomiting and headache, especially in children. It loses its effect with repeated use. Following parenteral administration of glucose (or glucagon), severe hypoglycaemia usually resolves. The most severe hypoglycemia is accompanied by unconsciousness (hypoglycaemic coma). A patient with insulin-induced hypoglycemia can also be treated on an outpatient basis; diabetics with antidiabetic-induced hypoglycemia should almost always be hospitalized.

## References

### Related Articles

- Diet in type 1 DM
- Diet in type 2 DM
- Diet therapy
- Special diabetic diets

### Source

- SVAČINA, Štěpán. *Dietologie a klinická výživa* [online]. [cit. 2012-03-10]. <<https://el.lf1.cuni.cz/p66466615/>>.