

Diets in oncology

While **nutritional factors** may play a role in cancer **prevention** and development, the contribution of dietary measures in **cancer treatment** is **controversial**. Any dietary changes can generally be achieved in patients only to a very limited extent. This is also true for serious diseases such as obesity. In contrast, the patient is willing to make changes at the time of cancer onset. Many alternative dietary therapies take advantage of this. In the past, withdrawal diets, diets with excessive calorie intake and various non-standard procedures have been considered. Glucose overdose has been considered in the past as a factor that could induce regressive changes in tumors and aid treatment. However, this approach has not proved effective. It makes no sense to become a vegetarian or to take any kind of ritually modified diet when cancer develops.

Tumours and malnutrition

The main measure today is the **prevention of malnutrition**. Tumour malnutrition is common in cancer patients and represents an important negative prognostic factor, according to some even more serious than the stage of the cancer. Malnutrition weakens cellular immunity, delays healing and also slows down reparative responses after treatment. Malnutrition is also associated with muscle weakness, poorer mobility, and the subsequent development of thrombosis and infectious complications. PIF (proteolysis inducing factor) produced by some tumours is probably responsible for muscle wasting.

Cancer patients tend to have **anorexia** (induced, for example, by certain interleukins), **depression** with reduced food intake, the **catabolic effect of the tumour** and other reasons for malnutrition. Anorexia and mucosal involvement can also be a consequence of chemotherapy and radiotherapy. Cachexia from gastrointestinal involvement is usually referred to as secondary. Weight loss of more than 10% of weight in 6 months is considered critical.

The need for nutritional intervention can be quantified, for example, with the PG-SGA (patient generated subjective global assessment of nutritional status) questionnaire. Also, patients undergoing bone marrow transplantation have a poorer prognosis if they weigh less than 95% of ideal weight (the risk is also higher if they weigh more than 110-120% of ideal body weight). Administration of glutamine at a dose of around 0.5-0.6 g/kg of weight is likely to have an effect on reducing complications and survival. According to the observation of the development of complications and survival, full dietary enteral or parenteral nutrition is an important measure in successful bone marrow transplantation. Similarly, nutritional support during radiotherapy and cancer surgery is important.

In other cancer patients, however, **glutamine administration** has not had the expected effect. The issue of so-called **immunonutrition** in patients with carcinomas is highly debated. "Immunomodulatory" diets with polyunsaturated fatty acids, arginine, glutamine, ornithine and alpha-ketoglutarate have not been shown to have a convincing effect. After administration of omega-3 fatty acids, laboratory evidence of improved T-helper/T-suppressor ratio and even longer survival have been sporadically observed. Also, in the field of cancer surgery, papers that have not demonstrated an effect of these so-called immunomodulatory diets are prevalent.

Weight loss is most typical for gastric, pancreatic and oesophageal cancers, and to a lesser extent for colorectal, bronchogenic and oral cavity cancers. Pathogenetic factors include nausea, anorexia nervosa, inflammation of the mucous membranes of the digestive tract, including mycoses and toxic effects of therapy, dry mouth and reduced secretion of digestive juices, diarrhoea or centrally mediated anorexia, and occasionally constipation due to lack of exercise and inadequate food intake.

From a dietetic point of view, the following measures may be tried:

- intake of small portions of food;
- intake of softer food;
- cooler food is suitable;
- fluid intake outside mealtimes;
- spicier and more colourful food is recommended;
- limiting fatty foods and a predominance of sweeter foods and protein is preferable;
- sufficient fibre is recommended;
- artificial food supplements such as sipping are appropriate.

If adequate oral intake cannot be maintained, enteral or parenteral nutrition is in order.



Smoking increases risk of lung cancer



Lack of exercise and increased alcohol intake increase breast cancer risk

- Enteral nutrition is preferable when the gastrointestinal tract is at least partially functional. It protects the intestinal mucosa and preparations with glutamine, arginine, MCT fats and polyunsaturated fatty acids are used. For example, Nutridrink or Nutridrink fibre are optimal supplements to enteral nutrition and diet in cancer patients. Supplementing energy intake with 1-3 drinks per day of 300 kcal is reasonable. It is usually successful for the patient to receive half the energy from Nutridrink and half the energy from a regular diet.
- Parenteral nutrition does not have major benefits, it is only given when oral intake is not possible. We already follow the classical all-in-one system and prefer to start with smaller doses up to 1000 kcal per day. It is reported that MCT fats are not utilized by the tumor tissue. However, there is probably no objective evidence of the usefulness of branched-chain amino acids or MCT fats in cancer treatment, nor is there any reason to administer high doses of glycidides. A slight increase in amino acid doses to 1-2 g/kg of weight per day at a dose of up to about 40-50% of the administered energy is appropriate. Glycidides are usually given at 3-4 g/kg of weight. There is no convincing evidence for the advantages or disadvantages of administering fat emulsions; it is sensible to administer them at a rather lower dose.

Recently, the supplementary **administration of synthetic gestagens** seems to be a very important measure to influence appetite and malnutrition. Bristol-Myers Squibb's Megace, according to several prospective randomized trials, improves quality of life. There is an increase in weight, improved appetite, mental and physical fitness. Administration is possible in tablets and oral suspension.

Cancer and obesity

Currently, **overweight** and **lack of exercise** are considered to be one of the most important **risk factors** for cancer, which may be both a cause and a consequence of obesity. Lack of physical activity is likely to be a risk factor for colon, breast and lung cancer, or sufficient physical activity is a protective factor for these diseases. The association between physical activity, overweight and consumption of certain foods is currently the subject of a number of epidemiological studies.

Many studies have shown that **obese men** are **more likely** to develop **colon, rectal and prostate cancer**. **Obese women** have an increased risk of most **gynaecological cancers and gallbladder tumours**. The rising relative risk of cancer death as a function of percentage overweight is in the following older table by Garfinkel, who followed 750,000 patients of both sexes for 12 years. Men who were 40% overweight had a cancer risk 1.3 times higher than controls and women 1.5 times higher. These figures are lower than the same risks for coronary heart disease (men 1.95 and women 2.07) and for diabetes (women 5.19 and men 7.90).

Overview of cancer risks				
Percentage overweight	10-19%	20-29%	30-39%	≥ 40 %
Men				
colon, rectum	-	-	1.53	1.73
prostate	-	1.37	1.33	1.29
Women				
endometrium	1.33	1.85	2.30	5.42
uterus (non-specific)	-	1.81	1.40	4.65
cervix	-	1.51	1.42	2.39
ovary	-	-	-	2.63
gall bladder	1.59	1.74	1.80	3.58
breast	-	-	-	1.53

Obesity, especially morbid obesity, is **associated with both hormone-dependent and hormone-independent cancers**. The risk of breast cancer appears to be relatively low in the obese. In fact, the incidence is affected by combining premenopausal and postmenopausal tumors into one table. Interestingly, pre-menopausal obesity reduces the risk of breast cancer. Women who are obese in adolescence are relatively protected from cancer until the transition; women who gain weight and become obese in adulthood have a high postmenopausal risk. Thus, after the transition, the risk of breast cancer is significantly higher for the obese than for the lean. The mechanism for the higher incidence of hormone-dependent cancers is debated. The proliferative effect may be due to higher estrogen levels (among others from the conversion of steroids to estrogens in adipose tissue) and relative hyperestrogenemia with lower levels of sex hormone binding globulin. According to Bruning, both the amount of free estrogens and the reduction of SHBG correlate with the waist-hip ratio. Phytoestrogens, estrogenic substances of plant origin, may also play a role in the pathogenesis of gynecological tumors. It is clear from intervention studies that even modest weight loss significantly reduces the risk of tumours. This effect is comparable to the reduction in the risk of diabetes and greater than the reduction in other risks such as cardiovascular risks. A small weight loss of 5-10% is sufficient to reduce cancer risk and further reduction does not change the risk. Williamson followed 43,000 American women who had never smoked. Over 12 years, he assessed the incidence of cancer. With a 10% reduction in weight, the risk of cancer fell by 50%. The relationship between colorectal cancer and obesity is interesting. According to this author, the risk of colorectal cancer peaks at a body mass index (BMI) of around 30 and is about 4 times higher. The risk is therefore already present in patients with a BMI between 25-30, i.e. in overweight patients. At higher BMIs, the risk decreases.

In recent years, the **relationship between kidney cancer and obesity, hypertension, diabetes** and reduced physical activity has also been convincingly demonstrated. One of the hypotheses tested in experimental animals is the increased peroxidation reaction in the proximal tubules of the kidney in the obese. A controversial protective role may then be played by antioxidants in the diet. For every unit of BMI (about 3 kg in an average tall individual), the risk of kidney cancer increases by 7%.

Tumours - prevention

Diet is very often implicated in the development of cancer. However, proving that diet induced a tumour in a particular individual is virtually impossible.

- The **products** of frying and deep-frying are undoubtedly **carcinogenic**, so it is not only the diet that is important, but also its modification. Diet usually has potentiating, stimulating or protective effects and is almost never a major risk factor for cancer. Probably genetic disposition plays a major role and diet acts as a facilitating factor. According to reports from the World Health Organisation, diet contributes to about 30% of cancer. It is logical to assume that diet is more important in the development of gastrointestinal cancers than in cancers in other locations, although there is some association in some cancers, for example of the respiratory tract. It should be noted, however, that there is as yet no evidence that any diet or dietary pattern can clearly prevent the development of cancer.
- **Consumption of sufficient fruit and vegetables** is considered to be a highly protective factor for most cancers.
- Smoking and alcohol consumption are **risk factors for upper gastrointestinal tract tumours**, as well as for **laryngeal tumours**, and their effect is multiplied. Any other risk or protective factors cannot then apply much, as both smoking and alcohol are very potent carcinogens.
- **The protective effect** of fruit and vegetable consumption is probably on oesophageal cancer. Tumours of these sites are more common in the obese.
- The incidence of **gastric cancer** is decreasing worldwide, but it is still the third most common cancer in the world. Until recently, *Helicobacter pylori* infection was considered the dominant risk factor. Current knowledge suggests that *H. pylori* is neither the only nor a necessary condition for the development of gastric cancer. However, patients with this chronic infection have a higher risk of developing gastric cancer, and it is in these patients that the positive effect of increased fruit and vegetable consumption is most often demonstrated. Cereal fibre has no convincing protective effect on gastric tumours.
- The incidence of **colorectal cancer** varies widely around the world. The most important protective factor is the consumption of vegetables or fruit. Meat consumption, smoking and alcohol, especially beer, increase the incidence of colon cancer. In addition to vegetable consumption, the use of non-steroidal anti-inflammatory drugs, hormone therapy and physical activity have a protective effect. This tumour is also significantly more common in overweight and partially obese people. The incidence is very high in type 2 diabetics. In a Czech study, it was shown that the risk of type 2 diabetes is 3-4 times higher than that of non-diabetics.
- Also, for **liver and pancreatic cancers**, the protective effect is mainly due to the consumption of leafy vegetables, whereas a slightly increasing risk of these cancers is associated with a high consumption of legumes (the higher the consumption, the higher the risk). The importance of protective fruit and vegetable consumption as a group for liver cancer has not been demonstrated. In pancreatic cancer, as in other cancers where cigarette smoke is a significant risk factor, the beneficial effects of antioxidants contained in fruit and vegetables may apply. Pancreatic tumours are significantly more common in type 2 diabetics and have recently been shown to be more common in type 1 diabetics.
- **Lung cancer**, the most common cancer in the world and one of the cancers for which we know the most important risk factor, is also positively influenced by antioxidants contained mainly in fruit. The incidence of laryngeal cancer can be positively influenced by sufficient consumption of both fruit and vegetables. The greatest positive protective effect has been demonstrated for broccoli, including in the Czech population.
- Globally, a sufficiently **high consumption of vegetables** could reduce the incidence of stomach cancer by up to 50% (about 28% in Europe) and colorectal cancer by up to 29% (about 18% in Europe). The importance of fruit and vegetable intake for bronchogenic cancer has been calculated as follows: the risk of a man who has smoked for 40 years is about 16 times higher than that of a non-smoker. This risk is 19% lower for those in the top quintile of vegetable intake (average 286 g/day) compared with the lowest quintile (103 g/day), and 31% lower for fruit in the highest quintile (325 g/day) compared with the lowest quintile (46 g/day). Thus, in total, fruit and vegetable intake can reduce the incidence of bronchogenic carcinoma by 13 %. Conversely, administration of high doses of betacarotene has been shown to be carcinogenic not only in bronchogenic carcinoma but also in alcoholics in general. Paradoxically, beta-carotene may have a pro-oxidant effect. The administration of other antioxidant vitamins (C, E) has not yet led to convincing results.
- The **ATBC study** suggested that the risk of prostate cancer may be reduced by up to 40% by administering vitamin E. More recently, it has been shown that regular intake of multivitamin mixtures increases the risk of prostate cancer. For breast cancer, it is convincing that regular intake of fruit and vegetables can reduce the risk by up to 60%. Even more convincing is the evidence that regular alcohol intake increases the risk of breast cancer. The influence of diet during childhood is very likely, with longer breastfeeding reducing the risk; higher fat intake (even polyunsaturated fatty acids) is a significant risk factor for later cancer.
- Currently, epidemiological studies also confirm the importance of **excessive salt intake** as a risk factor for gastric cancer, not only free salt but also, for example, in the form of food preservatives. Therefore, a high intake of canned vegetables that are pickled in brine or directly in salt is not a protective but a risk factor.
- **Meat consumption** is a risk factor that has long been discussed. Current studies confirm the likely risk of eating red meat, but even more so smoked, cured meat, including sausages. Also, the cooking of meat plays a very important role in the prevention of both gastric and colorectal cancer. In both cases, grilling, barbecuing and sausage making, or the curing and preserving of meat, are considered to be risky. The mechanism of

action is likely to be different. However, it is known that heterocyclic amines and polycyclic aromatic hydrocarbons are formed in meat prepared at high temperatures (frying, grilling, etc.). Both of these groups contain a number of substances with carcinogenic or potentially carcinogenic effects. Of course, the darker the meat ('burnt') and the more juice/fat from the meat consumed, the higher the risk or the higher the content of dangerous substances, at least if we compare the consumption of well-done and rare steaks.

- **Canned meat** is likely to contain excessive amounts of salt or some other substances - smoked meats e.g. smoke fumes etc. Epidemiological studies in recent years have shown that red meat consumption is a real increased risk of certain cancers: grilled meat increases the frequency of colon adenomas and stomach cancer, and fried meat is associated with lung cancer, although this risk factor is very weak compared with smoking.
- **White meat consumption** appears to have a protective effect on breast cancer. However, this conclusion does not apply to fish consumption. There is a lack of valid epidemiological studies on fish consumption and especially on fish preparation, since fish can also be prepared in a risky way (grilling, frying, smoking, salting, etc.).
- **Milk and dairy products** are a large group of potentially protective foods. However, reliable results from repeated large-scale epidemiological studies are still lacking; preliminary results suggest a protective effect of calcium and vitamin D; on the other hand, high milk consumption leads to activation of IGF-1 (insulin-like growth factor 1), a potential risk factor for cell growth.
- **Consumption of acrylamide**, a substance produced during the cooking of potatoes and cereals, is a major cancer risk. It is present in high concentrations in gingerbread, dark biscuits, chocolate, cocoa and coffee extracts, as well as in potato chips. It is produced by the Maillard reaction, which is analogous to the glycation of haemoglobin and produces a delicious flavour in baked foods from pastries to meat. In addition to being carcinogenic, the substances formed can be atherogenic and nephrotoxic. The longer the food is baked, the more of this substance is produced.
- The question of the quality and **quantity of fat intake** and its role in the genesis of bowel tumours is very complex. The risk curve is S-shaped. Intake of around 40% of energy in fat has a high risk (typical Western diet) and intake of around 10-20% has a relatively low risk (e.g. traditional Japanese diet). Saturated fats, such as the fat in beef and lard, pose a greater risk. In contrast, monounsaturated oils, such as olive oil, have little risk. The relationship to n-6-polyunsaturated fatty acids is controversial. According to some, n-6-polyunsaturated fatty acids appear to have a suspected carcinogenic effect in mammary cancer and, less perhaps, colon cancer. The incidence of colorectal cancer is reduced in animal models by n-3-unsaturated fatty acids contained in fish fats and oils. This positive effect may be due to increased production of prostaglandins, which reduce the stimulatory effect on cell growth.

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