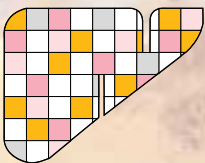


# A Guide for Patients with Liver Diseases including Guidelines for Nutrition



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# A Guide for Patients with Liver Diseases including Guidelines for Nutrition

Nutrition and Dietetics in Cirrhosis  
of the Liver and other Chronic Liver  
Diseases

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# 1 Introduction

This booklet entitled “A Guide for Patients with Liver Diseases including Guidelines for Nutrition” owes its existence to the desire of many patients with chronic liver diseases who want to discover more about correct “eating and drinking” in their illness.

Our knowledge in the field of nutrition in chronic liver diseases, especially cirrhosis of the liver, has increased considerably in recent years. The purpose of this booklet is to give you new information and clear up any misunderstandings or wrong information.

Today, we recognize that there is no such thing as a “liver diet” suitable for all patients. Diet in cirrhosis of the liver is very important. Cirrhosis of the liver (liver shrinkage), which is characterized by the progressive destruction of liver cells, can be slowed by correct eating and drinking. In turn, the patient’s general health can be improved by correct eating and drinking.

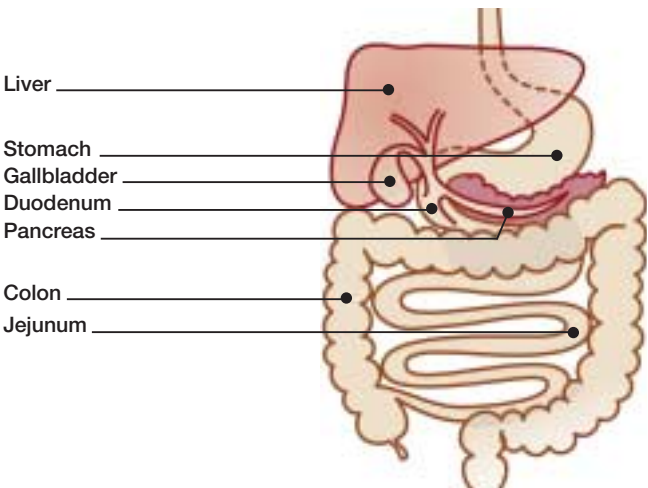
Diet in cirrhosis of the liver and other chronic liver diseases does not mean skimmed quark by the pound or a bland diet that is low in fat and lacking in taste! This booklet is designed to help you achieve a clearer understanding of the significance of diet in chronic liver diseases. You will find recipes, but that is not all. The recipes are preceded by a detailed and clear medical introduction and dietetic information. You should understand exactly why particular food ingredients and, hence, particular foods, are bad for you and for your liver. This booklet does not intend, nor is it capable of, replacing the qualified individual advice provided by your doctor, dietary counsellor or dietician.

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## 2 Liver function and nutrition

The liver weighs about 1.5 kg and is situated on the right side of the upper abdomen. The gallbladder lies in the hollow of the right lobe of the liver. The gallbladder is a “storage bin” for the bile produced by the liver. Bile is important for the digestion of fat. The gallbladder contracts in case of fatty diets and bile is secreted into the duodenum, the upper segment of the small bowel, into which the stomach contents are emptied.

The liver is the main metabolic organ in the body. In order to be able to carry out its metabolic functions, a great part of the blood pumped out by the heart is carried to the liver via the circulatory system. Studies have shown that about 1–1.5 liters of blood are transported to the liver every minute via the portal vein. The hepatic artery brings oxygen-rich blood to the liver while the portal vein transports nutrient-rich blood to the liver. The blood in the portal vein has already passed through the gastrointestinal tract and absorbed large amounts of nutrients. In the hepatic cells, the nutrients (proteins, carbohydrates,





and fat) and their building blocks (proteins = amino acids, carbohydrates = simple sugars and fats = fatty acids and glycerol) are processed further.

## **2.1 Functions of the liver**

As the main metabolic organ in the body, the liver has many different functions, including:

- Production of protein building blocks (amino acids), proteins (e.g. clotting factors, albumin), cholesterol and bile acids
- Regulation of the blood sugar level by production or use of glucose
- Production and supply of bile for digestion of fats
- The neutralization and elimination of waste products of the body's own metabolism and foreign substances such as drugs and environmental toxins
- Storage of nutrients (glycogen and sugar reserves), minerals (e.g. iron), or vitamins (e.g. vitamin B<sub>12</sub>)

### *2.1.1 Proteins*

The body required proteins for the maintenance of its structure, including the skeleton, tendons, connective tissue and for the cell membranes of every cell in the body. Proteins are also required for the performance of bodily functions, such as muscle contractions and the production of antibodies to defend against invading microbes. Protein means “the first” or “the most important”. This underlines the great importance of proteins to the organism. **Without protein, life is not possible.** Because the body constantly replaces “worn-out” protein and because protein cannot be stored long-term, human beings are dependent on an adequate daily intake of protein for optimum body functioning. Protein in the body is not normally used for gaining energy but is

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an important building substance. If it is burned for energy, 1 g of protein provides the body with 4 kilocalories (kcal).

### **High protein foods**

Eggs, poultry, fish, meat, milk, dairy products, sausage, pulses (especially soy beans and soy products).

### **Low protein or protein-free foods**

Sugar, oil, starch, butter, margarine, salads, fruit, vegetables.

In the small bowel, protein is broken down into short protein fragments (so-called oligopeptides) and into its smallest components, the amino acids, and carried to the liver. Using these amino acids, the liver synthesizes the body's own proteins and provides them to the organism. Some of these building blocks are passed on by the liver to other organs so that they can use them to make their own proteins (for example, muscle fibers in muscle).

## *2.1.2 Carbohydrates*

The main function of carbohydrates is as a fuel for the rapid supply of energy for the body. Carbohydrates are made of chains of different lengths which are formed by the splicing of simple sugars (monosaccharides). Glucose is one of these monosaccharides that represents the main energy source for all tissues. Fasting blood sugar levels in the healthy person range between 50 and 110 mg/dl (2.8–6.1 mmol/l) and increase two hours after ingesting 75 g of glucose to levels below 180 mg/dl (10.0 mmol/l). Higher values are evidence for diabetes mellitus. In quantitative terms, carbohydrates are the

most important nutrient for the human body. Roughage, which cannot be digested by the body, is also classed among the carbohydrates. Roughage is important for feeling satisfied, having good digestion and healthy intestinal flora. 1 g of carbohydrate provides the body with 4 kilocalories (4 kcal).

### **High carbohydrate foods**

Sugar, sweets, bread, flour, starch, fruit, potatoes, rice, pasta, oats, crispbread, milk, vegetables.

### **Low carbohydrate foods**

Butter, margarine, oil, meat, fish, poultry, eggs, sausage, cheese.

Acted upon by enzymes in saliva, the secretions of the pancreas and intestinal mucosal membrane, these carbohydrates are broken down into sugar building blocks, including monosaccharides such as glucose, galactose and fructose. They are absorbed by the bowel and carried by the circulation via the portal vein into the liver. Some of these carbohydrates are stored in the liver and the muscles in the form of glycogen, which represents a rapidly available source of energy. The remainder is returned to the blood and serves as the energy supply for the cells. If more carbohydrates are absorbed than are required by the body, they are changed into fat and stored in the adipose tissue.

### *2.1.3 Fats*

Fats principally serve the body as a concentrated source of energy and a component of cell membranes, as well as being a substrate for the production of messenger substances, such as those that affect inflammation. We also need fats for the absorption of fat-soluble vitamins

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(vitamins A, D, E and K). When the fat metabolism is disturbed (e.g. increased cholesterol) the total fat intake should be restricted. Moreover, fats of high quality should be preferred (e.g. olive, rapeseed, corn, thistle, sunflower or soy-bean oils, diet or sunflower margarine). Fish oil (omega-3-fatty acids) also has the effect of lowering the blood fat level (particularly the triglycerides). Of particular value are the simple unsaturated fatty acids, which are found especially in olive or rapeseed oil. Butter or other animal fats are not preferred, since they chiefly contain saturated fatty acids and cholesterol. 1 g of fat produces 9 kilocalories (kcal).

### **High fat foods**

Oil, lard, butter, margarine, mayonnaise, sausage, fast food, sweets, cheese, meat, cream, cakes.

### **Low fat or fat-free foods**

Fruit, vegetables, flour, bread, sugar.

Fats and fat-like substances (e.g. cholesterol) are absorbed in the small bowel, carried to the blood stream in the lymphatics and transported to the liver. The components of fat (fatty acids and glycerol) are broken down in the liver and transported to other organs, such as the muscles, as a source of energy. Excess fat is stored in adipose tissue. The liver ensures that fat is utilized and absorbed from the small bowel by the excretion of bile.

## *2.1.4 Vitamins*

A distinction is drawn between fat-soluble and water-soluble vitamins. The fat-soluble vitamins A, D, E and K are stored in the liver. The liver is also involved in metabolism of vitamins of the B group and vitamin K. Minerals, such as iron, are also stored in the liver.

### *2.1.5 The body's toxin laboratory*

Together with the kidneys, the liver is the detoxifying organ for the body. Toxins (poisons) produced by the body as a result of metabolism and those administered (medicines, harmful substances and alcohol) are rendered harmless in the liver and made ready for excretion through the bile (into the stool) or the kidney (through the urine).

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## 3 Chronic liver diseases

### 3.1 Fatty liver (*hepatic steatosis*)

Doctors speak of a fatty liver when the liver cells show fatty degeneration to over 50%. Normally their fat proportion is 5%. There are various forms of fatty liver, which react positively to dietary therapy:

- Hepatic lipomatosis (due to overnutrition [calories, fat, carbohydrates])
- Alcoholic fatty liver (caused by alcohol)
- Deficiency fatty liver (due to protein and calorie deficiency)
- Metabolic fatty liver (e. g. in diabetics)
- Uncertain causes (fatty liver in endemic sprue)

In addition, medications and other causes may lead to fatty liver and fatty liver inflammation (steatohepatitis). Fatty liver is frequent in Germany due to generalized faulty nutrition and overeating, with at least one quarter of the population being affected. Fatty liver does not initially restrict liver function, however, and, as a rule, has a low clinical significance, usually does not cause any complaints and can be fully reversed. It may, however, progress to an inflamed form, called steatohepatitis, which may be indistinguishable from the form caused by alcohol abuse. This disease, called non-alcoholic steatohepatitis (NASH), together with alcoholic steatohepatitis (ASH), may progress to liver cirrhosis.

Nutritional therapy of fatty liver is dependent on the cause:

- Hepatic lipomatosis    Weight reduction by means of fat-reduced diet, avoidance of high-sugar foods, including fructose and products containing it, exercise, abstinence from alcohol

- Alcoholic fatty liver      Abstinence from alcohol, adequate supply of energy, proteins, vitamins and trace elements
- Deficiency fatty liver      Adequate supply of energy and protein, abstinence from alcohol
- Metabolic fatty liver      Good blood sugar regulation, reduction of overweight, exercise, abstinence from alcohol

A fatty liver is particularly vulnerable and in danger of progressing to steatohepatitis in patients who fast and attempt drastic weight reduction. Hence, a more careful and long-term weight reduction is recommended. The success of dietary therapy is significantly improved by regular exercise. As in all liver diseases, alcohol should absolutely be avoided. Bland liver diet is neither advisable nor appropriate in the case of fatty liver.

Only recently have physicians turned their attention to endemic sprue as a cause of fatty liver and other liver damage. The causal mechanisms are not yet completely understood. Remarkable, however, is the success of gluten-free diets, which often result not only in resolution of intestinal symptoms but lead to complete reversal of liver changes. This is even more important in light of the fact that endemic sprue is one of the most common hereditary disease in European populations and is probably still not recognized in all cases.

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### 3.1.1 Alcoholic liver disease

In Germany, the most common cause for the development of cirrhosis of the liver is misuse of alcohol. When alcohol is misused over many years, liver damage and the probability of developing cirrhosis of the liver must be reckoned with. This includes an increased risk for developing hepatic cell carcinoma.

The risk of alcohol damage is significantly higher for women than for men. Even now – from the very young to the very old – Germans drink 20–25 g of pure alcohol per day on average.

#### *For men:*

A daily intake of 60 g alcohol (that is 1.5 liters of beer, 0.6 liters of wine, 0.5 liters of sparkling wine or 120 g of rum) over a prolonged period damages the liver.

#### *For women:*

A daily intake of 20–40 g alcohol (that is 0.5–1.0 liters of beer, 0.2–0.4 liters of red wine or 100–200 g of liqueur) over a prolonged period damages the liver.

Alcohol provides 7 kcal per gram:

The alcohol content of various alcoholic drinks per 100 ml (after F. Heepe, Dietetic Indications, Springer Verlag):

Malt beer	0.6–1.5 g	Light wine	5.5– 7.5 g
Small beer	1.5–2.0 g	Medium wine	7.5– 9.0 g
Whole beer	3.5–4.5 g	Strong wine	9.0–11.0 g
Strong beer	4.8–5.5 g	Fortified wine	11.0–13.0 g
Alcohol-free drinks	–0.5g	Sparkling wine	7.0–10.0 g
		Liqueur	20.0–35.0 g
		Spirits	32.0–50.0 g
		Rum	40.0–70.0 g



As soon as the first signs of liver damage or cirrhosis of the liver appear, consumption of alcoholic drinks should be cease. As a precaution, so-called alcohol-free drinks (e.g. alcohol-free beer, sparkling wine or wine) should also be avoided. It is important to note that foods (e.g. brandy beans) and some medications (drops) may also contain alcohol. Inform your doctor that you suffer from cirrhosis of the liver whenever you are prescribed a new medicine. In the case of all liquid medication, ask your pharmacist whether it contains alcohol. Renounce self-medication and avoid so-called restoratives (e.g. lemon-balm spirit or so-called “health tonics”), which often contain high-percent alcohol.

Bear in mind that the average daily intake of alcohol in the Federal Republic of Germany is nearly 30 g! Since reunification of the old and new Federal States, the Germans have become world champions in drinking alcohol. In Germany, about 20,000 people die of cirrhosis of the liver alone. **Cirrhosis of the liver is thus the fifth most common cause of death in our country.**

Alcohol steatohepatitis is a consequence of alcohol abuse but still represents a reversible transitional stage on the path from simple fatty liver to alcoholic liver cirrhosis. Severe acute alcoholic hepatitis is a life-threatening disease with high mortality. Crucial to therapy is an absolute abstinence from alcohol and institution of dietary therapy up to intensive therapy to prevent organ failure.

### ***3.2 Hepatitis – Inflammation of the liver***

Viral hepatitis (inflammation of the liver caused by different viruses) belong to the most common infectious diseases worldwide. There are acute and chronic forms. Some

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types of hepatitis can develop into liver cirrhosis. To date, the following causative agents have been identified for hepatitis:

- Hepatitis A Virus (HAV)
- Hepatitis B Virus (HBV)
- Hepatitis C Virus (HCV)
- Hepatitis D Virus (HDV)
- Hepatitis E Virus (HEV)

Hepatitis viruses can be transmitted orally through infected foods or beverages (especially hepatitis A and E) or parenterally (bypassing the digestive tract) through body fluids, such as blood, blood products and genital secretions (especially hepatitis B, C and D). Dietary therapy plays no important role in the treatment of viral hepatitis. Special forms of diet previously propagated and recommended (bland liver diet) have no value and should not be implemented. In the acute phase, it often helps to keep to a light normal diet, which is easy to digest. In the hospital, hepatitis patients are usually given a light standard diet excluding foods and beverages that are generally hard to digest.

A similar clinical picture, and one that, in the past, was difficult to distinguish from viral hepatitis, is due to inflammation caused by the body acting against itself. This entity is known as autoimmune hepatitis. Today, the diagnosis and therapy of autoimmune hepatitis are no longer as difficult. Autoimmune hepatitis is always a chronic hepatitis and carries the risk of progressing to cirrhosis. With regard to nutrition, the same general recommendations as in chronic viral hepatitis or liver cirrhosis apply. Only in the phase of treatment with prednisolone is it important for patients to watch for increased appetite with resulting weight gain and possible increase in blood sugar levels.

### **3.3 Primary biliary cirrhosis (PBC)**

Primary biliary cirrhosis is a rare, chronic liver disease whose rate of progression varies from case to case. It occurs mainly in women. The causes of this liver disease are not yet clear but it is assumed that an autoimmune disease causing damage of the smallest bile ducts and leading to cirrhosis is involved. The only generally accepted treatment at present is ursodeoxycholic acid (UDCA), a naturally occurring bile acid that is also present in human bile in small amounts. Ascites, esophageal varices or hepatic encephalopathy, frequent complications of liver cirrhosis, may develop in PBC as well. Patients affected with PBC also suffer from marked pruritus (itching) which generally disappears on treatment with ursodeoxycholic acid or which can be treated with drugs that bind bile acids in the bowel.

As the disease progresses, the disturbance of bile formation results in too little bile acid being produced for the digestion and absorption of normal dietary fats. With this loss of dietary fats in the stool (fatty stool or steatorrhea), patients experience an energy deficit, weight loss and inadequate absorption of the fat-soluble vitamins A, D, E and K, resulting in deficiency syndromes including night blindness, reduced sense of taste, weak bones and a tendency to bleed. Patients may be helped by means of the so-called MCT (medium-chain triglycerides) diet. In general, dietary therapy in patients with PBC is similar to that recommended for patients with cirrhosis due to other causes.

Frequently the fat soluble vitamins (A, D, E and K) are required at increased doses. If there is a loss of bone substance (osteopenia), vitamin D and calcium must be administered.

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### **3.4 Hemochromatosis**

This hereditary disorder leads to the absorption of abnormally large amounts of iron in the small bowel, resulting in an iron overload for the body and damage to the liver (cirrhosis), heart (heart muscle weakness), joints (damage to joint cartilage) and internal glands (diabetes). Iron is a vital trace element. A low-iron diet is practically impossible to maintain, since iron occurs in a great variety of foods. Today, no attempt is generally made to treat hemochromatosis with dietary measures. However, high-iron foods such as innards and large amounts of meat, sausage or cold cuts should be avoided.

### **3.5 Wilson's disease**

In Wilson's disease, the liver, due to an inherited defect, is unable to excrete excess copper, resulting in copper overload in the organism. This causes damage to the liver (cirrhosis) and the nervous system. Copper is a vital trace element. In an average diet we take in about 2–5 mg of copper per day. Patients with Wilson's disease should maintain a low-copper diet. Foods rich in copper, which should be avoided, are seafood, sea fish, innards, large amounts of meat and sausage, nuts, dried fruit, particularly raisins, mushrooms and cocoa. There is little copper in milk, dairy products, cheese, sugar, flour and fruit.

## **4 Liver cirrhosis – Final stage in chronic liver diseases**

Over 2 million people in the Federal Republic of Germany (old and new Federal States) suffer from chronic liver diseases. A total of 800,000 people in Germany suffer from cirrhosis of the liver.

Cirrhosis of the liver is defined as advanced, irreparable destruction of metabolically-active liver cells, transformation of the architecture of the blood vessels and increase in connective tissue. The connective tissue is unable to carry out the functions of the liver cells. The liver tissue becomes firm and shrinks, which is why cirrhosis of the liver is also known as “shrunken liver”.

This shrinking also affects the blood vessels, blocking the inflow of blood flowing in from the bowel through the portal vein (portal hypertension). This can lead to the formation of varicose veins in the esophagus (esophageal varices), ascites (accumulation of fluid in the abdomen) and disturbances of bowel function (e. g., reduced digestion of fats, bloating). Other complications of liver cirrhosis are malnutrition, frequent bacterial infections, brain dysfunction up to and including coma (hepatic encephalopathy) and hepatic cell carcinoma. The end point of advanced liver cirrhosis is either death or liver transplantation.

There are a large number of so-called “liver protection agents”. Up to now there is no proof that cirrhosis of the liver can be improved or cured. The positive effect of dietetic treatment, on the other hand, is well-established.

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*The causes of cell destruction are chronic inflammations of the liver (hepatitides) brought about by:*

- Alcohol abuse
- Non-alcoholic steatohepatitis (NASH)
- Viruses
- Chronic inflammation of the bile ducts (cholangitis)
- Medications
- Unknown causes (cryptogenic)
- Rare metabolic disorders (e. g. hemochromatosis, Wilson's disease)

In a small percentage of patients, no cause can be discovered and the term cryptogenic cirrhosis of the liver is used. Cryptogenic means that the cause of the illness is unknown.

*Two different degrees of severity of cirrhosis of the liver should be distinguished:*

- **Compensated form:**

With adequate detoxifying activity, no ascites and no hepatic encephalopathy. The diagnosis is made by ultrasound, laboratory studies, laparoscopy (using an endoscope to inspect abdominal organs) or biopsy (examination of a tissue sample using microscopic methods). In this stage, the patient often does not experience any symptoms at all or only uncharacteristic complaints.

- **Decompensated form:**

As a result of reduced liver function and portal hypertension, patients experience jaundice, readiness to bleed due to lack of clotting factors, ascites, edema, loss of muscle mass, bleeding esophageal varices, hepatic encephalopathy and progressive deterioration in laboratory test results and in the general condition state of the patient (weakness, fatigue).

## **4.1 Complications of liver cirrhosis**

### *4.1.1 Ascites*

Ascites means an increased collection of fluid in the free abdominal cavity. The blood flow through the liver is hindered by the increasing replacement of liver cells by connective tissue. This leads to an increase in the pressure in the portal venous flow which results in the transfer of fluid into the abdomen. The development of ascites is promoted by a shift in the protein content (lack of albumin) and disturbances in mineral and hormone metabolism. As a result of this lack of protein, fluid can cross more readily into the abdominal cavity. Migration of bacteria from the bowel can also promote the development of ascites. In cases of ascites patients must also be tested to see if they are getting enough energy and protein.

### *4.1.2 Digestive disturbances and intolerances*

The enlargement and hardening of the liver and the increasing pressure in the portal venous system affect the intestinal function. This may lead to disturbances in bowel motility, compromised barrier function of the intestinal mucosal membrane (migration of bacteria) and incomplete digestion (bloating, fatty stools). Patients may notice food intolerance, which may be characterized by the following symptoms:

- Sensation of fullness
- Loss of appetite
- Abdominal pain
- Bloating

The food intolerances differ between individuals. Use of dietary protocols and adherence to a light normal diet have proved their value in identifying the foods that cause intolerances. Foods which are often hard to di-

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gest are fatty, fried, raw and heavily seasoned foods as well as pulses, Sauerkraut and cabbage varieties (apart from cauliflower and kohlrabi). Patients with cirrhosis of the liver should obtain advice from a dietician about easily digestible foods.

#### *4.1.3 Malnutrition*

As the liver disease progresses, its effects on the nutritional status of the individual become more and more noticeable. Typically, patients will experience a loss of muscle mass and subcutaneous adipose (fat) tissue, while at the same time increasing the amount of water in the tissues (ascites, edema). Often, despite severe malnutrition, patients' weight will remain in the normal range due to drastic shifts in the composition of the body: A loss of valuable body mass (e.g. muscle) is replaced by an excess of useless ballast (ascites, tissue water). In many cases, this combined deficit in protein and energy intake is associated with deficiencies of vitamins and trace elements. Consequences of malnutrition include weakness, immune deficiencies and worsening of liver function.

#### *4.1.4 Esophageal varices*

As a result of increased pressure in the portal vein, the blood flow from the area of the portal vein seeks new outlets and we see the development of circulatory bypasses to relieve the pressure and the liver is bypassed by so-called collaterals. These develop from small, thin-walled vessels that otherwise carry only small amounts of blood. The vessels are now well perfused and filled to bursting. Circulatory bypasses are frequently formed around the stomach and the esophagus. They are similar to varicose veins in the legs and are termed esophageal



or fundal varices depending on location in the esophagus or upper end of the stomach. These vessels rupture easily and this can lead to dangerous bleeding. After large meals, the perfusion of the bowel increases, resulting in increased pressure within the esophageal varices. Hence, six small meals are better than three large ones in patients with esophageal varices.

#### *4.1.5 Changes in protein metabolism*

Patients with liver cirrhosis are more often in danger of losing body protein than are healthy persons. Because of their reduced glycogen reserves, patients with cirrhosis have reached a stage of hunger after 16 hours of fasting that occurs in healthy persons only after 36 hours of abstinence from food. Hence, after only a short period of fasting, the body turns to its own protein for energy production. Similarly, repeated bacterial infections and bleeding serve to increase this protein catabolism.

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Mouth

Esophagus

Liver

Stomach

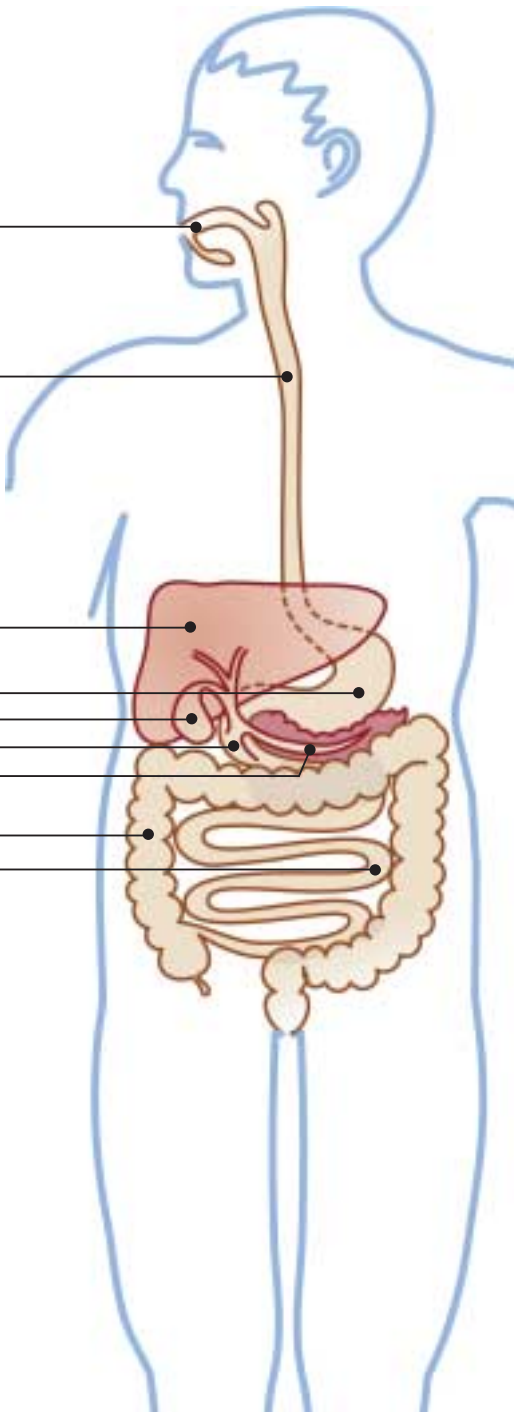
Gallbladder

Duodenum

Pancreas

Colon

Jejunum



This leads, on one hand, to worsening of pre-existing muscle atrophy and, on the other, to a build-up of waste products (such as ammonia) from the increased protein breakdown. Fasting may result in the development of neurologic changes (encephalopathy) and, hence, should be strictly avoided. Because patients with cirrhosis are still able to utilize dietary protein for the production of important proteins in the body, it is important to optimize protein intake and avoid protein-restricted diets.

Measuring ammonia levels in the venous blood of patients with cirrhosis is not helpful. Ammonia levels in cirrhosis are almost always elevated as the result of collateral circulation; these levels cannot be used as evidence for the extent of encephalopathy. Elevated ammonia levels without symptoms of hepatic encephalopathy do not justify protein restriction.

#### *4.1.6 Hepatic encephalopathy*

Worsening of liver function and the detour around the liver for a certain part of the blood leaving the intestine results in the bodies of patients with liver cirrhosis being overwhelmed with toxins that in healthy people would have been captured in the liver and made harmless. Patients with advanced liver cirrhosis therefore have higher blood concentrations of toxic breakdown products, such as ammonia, phenols, indoles and amines. This affects the function of the brain. As a result, patients are tired and slowed in their reaction time. They suffer from disturbances of concentration and coordination, which may become noticeable as writing disturbances, shaking and jerking of the hands (flapping tremor). Their ability to operate a motor vehicle may also be compromised. These toxic symptoms of the brain are called hepatic en-

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cephalopathy (from *hepar*, liver; *enkephalos*, brain; *pathos*, disease). This may progress to deep unconsciousness (hepatic coma).

#### 4.1.7 *Diabetes mellitus*

Half of those with cirrhosis of the liver also suffer from diabetes mellitus. Diabetics must pay particular attention to carbohydrates and obey special dietary rules. This group of patients must be given thorough and comprehensive advice by dieticians. Patients with cirrhosis of the liver and diabetes should get themselves a carbohydrate exchange table in addition to a table of nutritional values. For diabetics, the high-fiber diet recommended in cirrhosis of the liver is doubly important. Roughage slows down the rise in blood sugar after consumption of high carbohydrate meals.

#### 4.1.8 *Clotting factors*

If the liver fails, it can no longer produce and provide to the blood adequate amounts of clotting factors, resulting in an increased tendency to bleed.

This situation may also occur when, despite adequate remaining synthetic ability, the liver is unable to produce clotting factors due to a lack of vitamin K, a necessary nutrient. Vitamin K deficiency typically occurs when intestinal absorption of fats and fat-soluble vitamins (including vitamin K) is reduced. Normal absorption of fats requires bile, which is produced by the liver. In some situations, it may be necessary to give additional vitamin K either as an injection or as an infusion.

## 5 Diet in cirrhosis of the liver

The importance of correct diet in cirrhosis of the liver is unfortunately still underestimated. **A “liver-adapted” diet is just as important as medication.** Many patients leave hospital without first having been given advice by a dietician. Comprehensive individual dietary guidance for patients should be a matter of course because otherwise the patient cannot observe the correct diet.

As long as the liver fulfils its functions (compensated type of cirrhosis of the liver), no dietetic treatment is required. Patients should maintain a healthy diet, preferably taking six small meals distributed throughout the day, and absolutely avoid alcohol. In no case should protein intake be restricted, because, in doubtful cases, this will only be harmful. Daily protein intake should be about 1.2 g per kg body weight.

In decompensated liver cirrhosis, it is important to assure that the patient is getting the required amounts of nutrition. Often, due to poor appetite, rapid satiety (e.g. due to ascites), weakness and fatigue, dietary intake is inadequate. Also, the poor taste of hospital food or of a low-sodium diet may be culpable in patients not taking adequate nutrition. It is important to monitor patients to assure that they are actually consuming the food offered to them. If patients do not receive adequate nutrition in the course of ordinary eating, the use of products for artificial enteral nutrition should be considered.

There should be no automatic decision to put patients on a reduced protein diet even in decompensated liver cirrhosis.

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On the contrary, these patients, who are often affected by a significant protein and energy deficit, should actually be taking 1.5 g of protein per kg each day, or about 100–120 g of protein per day in most cases. This corresponds to an ordinary diet in healthy persons, with adequate amounts of fruit, vegetables, salads, whole grain products, potatoes, rice and pasta. Information on healthy diets can be obtained from the nutrition pyramid printed in the middle of this booklet (pages 36–37). The protein content of various foods can be found in dietary tables (e.g. GU Nährwerttabelle, Gräfe und Unzer Verlag).

Only in rare cases will it be necessary to reduce the daily amount of protein in the diet.

This is only in cases where hepatic encephalopathy is demonstrably triggered by high-protein meals but does not occur when protein intake is restricted. This is a sign of protein intolerance. It is uncommon for encephalopathy to be triggered in this way. More common triggers for encephalopathy are infections, bleeding, drugs, compromised renal function or constipation.

## **5.1 Principles of light normal diet**

The light normal diet has superseded the “organ-protecting diets” which for so long forced many patients with diseases of the liver, pancreas, stomach, gallbladder or intestine into dietary regimes that were too restrictive. The regimes were strict and provided no real help to patients. Patients with liver diseases are often given a light standard diet in hospital.

The light normal diet is a type of diet that serves only to avoid symptoms such as sensation of pressure or fullness, pain, nausea, bloating or diarrhea, which may occur after eating.

If you suffer from food intolerance, try to avoid all foods, which, in your own experience, are capable of producing symptoms. Individual advice provided by dieticians can help you in this. It does happen that certain patients cannot tolerate particular foods or dishes. This differs according to the individual. Lists of forbidden foods and of foodstuffs and dishes which are generally considered to be poorly-tolerated are not helpful and should not be disseminated. In determining what is permissible for you and what is less good, you will find it helpful to make use of dietary protocols. Write down what you eat and state whether and what symptoms you had afterwards. After a short period of time you will be able to produce your own list.

The following foods, which on statistical evaluation lead to intolerances in many patients, can serve as a starting point:

pulses	coleslaw	wine
cucumber (raw)	potato salad	spirits
white cabbage		strong tea/coffee
cabbage	hard-boiled egg	
paprika	Eisbein (pickled	nuts
pickled cabbage	knuckle of pork)	cream
(Sauerkraut)		raw fruit with
onions	highly seasoned,	a core or stone
savoy	smoked,	
mushrooms	deep-fried and	
leek	fatty foods	

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Avoid all foodstuffs that you cannot tolerate. Do not rely solely on books or booklets. You yourself must decide what suits you and what doesn't.

## **5.2 Indications for starting dietetic treatment**

Dietetic treatment becomes necessary when there are signs of malnutrition or adequate nutrition is no longer possible using ordinary means.

*Signs of malnutrition include:*

- Loss of muscle mass
- Loss of subcutaneous adipose tissue
- Increase in tissue water

Body weight and even the body-mass index (BMI) may erroneously remain normal because the loss of muscle and adipose tissue is masked by the accumulation of water in the form of ascites or edema. Physical examination by a physician is essential. Laboratory values such as the levels of albumin, prealbumin or cholinesterase are less useful for diagnosing malnutrition in liver cirrhosis, since they are already abnormal due to reduced liver function. If the actual degree of malnutrition must be measured, it is helpful to measure the body cell mass with a bioimpedance assay (BIA).

The experienced physician, however, requires no technical aides to determine whether or not a patient is malnourished: He makes the diagnosis based on questioning the patient and the results of his physical examination.





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*The measures in dietetic treatment are:*

- Assuring the adequate intake of protein and of the correct types of proteins
- Assuring an adequate supply of energy
- Increased dietary intake of fiber
- Administration of branched-chain amino acids
- Reduced intake of sodium
- Restriction of fluid
- Increased intake of potassium

*The goals of dietetic treatment are:*

- Preventing or remedying malnutrition
- Improving liver function
- Avoiding catabolic states (increased breakdown of the body's own protein), which may trigger hepatic encephalopathy
- Improving protein metabolism, especially in patients requiring reduced protein diets, by providing increased amounts of branched-chain amino acids
- Management of the formation of ascites and edema by a low sodium diet, fluid restriction, and a plentiful supply of potassium

### **5.3 Energy supply**

About 70% of patients with chronic liver diseases have a faulty diet. The immune system of malnourished patients is weakened. There may also be loss of the body's own protein, such as in catabolic states with breakdown of muscle mass. Here it should be remembered that, despite being underweight, there is an increased proportion of adipose tissue and a decreasing proportion of muscle mass. The affected patients have a paunch (fatty tissue, and/or ascites) and thin arms and legs (little muscle). The patients only seem to be well nourished. The causes of the poor state of nutrition lie in an imbalance between

increased energy needs (due to the liver disease and its complications) and inadequate energy intake (poor appetite, faulty diet). The energy requirement can be calculated using the following formula:

*Energy requirement*

Normal weight (height in centimetres minus 100) times 35 = energy requirement in kilocalories per day.

This calculation considers the energy content of all foods, including that of dietary protein, which is not primarily used as a source of energy.

*Example:*

A man of 174 cm requires about 2600 kilocalories while in hospital ( $174 - 100 = 74$ ,  $74 \text{ times } 35 = 2590 \text{ kcal}$ ). This diet should include from 90 g (compensated cirrhosis, good nutritional status) to 120 g (decompensated cirrhosis, malnutrition) of protein.

1 g of protein delivers 4 kilocalories (kcal) of energy.

This formula includes a safety factor. It is also applicable to overweight patients in whom no reduction in calories is to be recommended. Patients with decompensated cirrhosis of the liver ought not to lose weight (except with ascites). Besides fatty tissue, the body also breaks down protein and this aggravates hepatic encephalopathy. The breakdown of the body's own protein leads to an increase in the blood ammonia level. Energy supplementation in the decompensated form of cirrhosis of the liver, if necessary, can be done using fats or carbohydrates. Patients should not be prevented from using butter.

Energy supplementation prevents any body substances (e.g. muscle tissue) being used as a source of energy or to meet the protein requirement. When endogenous

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body substances are broken down there is an increase in levels of ammonia. This encourages the development of hepatic encephalopathy.

*Following are examples of foods suitable for energy supplementation:*

### **Maltodextrin 19**

Maltodextrin is a neutral tasting powder that is obtained from maize starch. 1 g of maltodextrin contains 3.8 kilocalories. Maltodextrin is easy for the body to utilize and is well tolerated. You can make use of it in cold and hot food, in sweet or spicy dishes. No more than 10 g of maltodextrin should be incorporated into 100 g of food. Maltodextrin 19 can be obtained from the pharmacist.

The use of these products is not suitable for **diabetics**. The increase in blood sugar following glucose or maltodextrin is extremely rapid. The products should only be used with careful monitoring of blood sugar and blood sugar correction where required.

### **Butter, margarine or oil**

At 9 kilocalories per gram, fat is the most energy-rich foodstuff. For example, a pudding can be cooked with cream instead of milk for energy enrichment. The caloric value of soups and sauces can easily be increased with butter or margarine. Mashed potato with cream and butter is a true calorie bomb. A limit is only placed on the use of fat when the patient has raised levels of fat in the blood; in that case, he should avoid butter and cream. Patients with cirrhosis of the liver, however, only rarely have increased levels of cholesterol. The amount of fat is also restricted when it is poorly tolerated.

### **Artificial foods (liquid diet/tube feeding)**

For patients who cannot eat adequately, there are industrially manufactured liquid diets which can be either taken by mouth or administered through a tube. This type of artificial nutrition is known as enteral nutrition. Taken by mouth, it differs from parenteral nutrition, which is administered by infusion into a vein.

These liquid diets cover the body's requirements for all essential nutrients and can be used as an exclusive source of nourishment. For patients with cirrhosis, liquid diets with higher caloric content (1.5 kcal/ml) are preferable to standard formulas (1.0 kcal/ml) because of their frequent edema or ascites. As a rule, these diets do not require any special formula, since the primary concern is to assure adequate energy and protein intake. Only in very rare cases, where reduced protein is truly necessary because the protein content of a standard diet causes encephalopathy, is it necessary to use liquid diets specially formulated for the needs of liver patients.

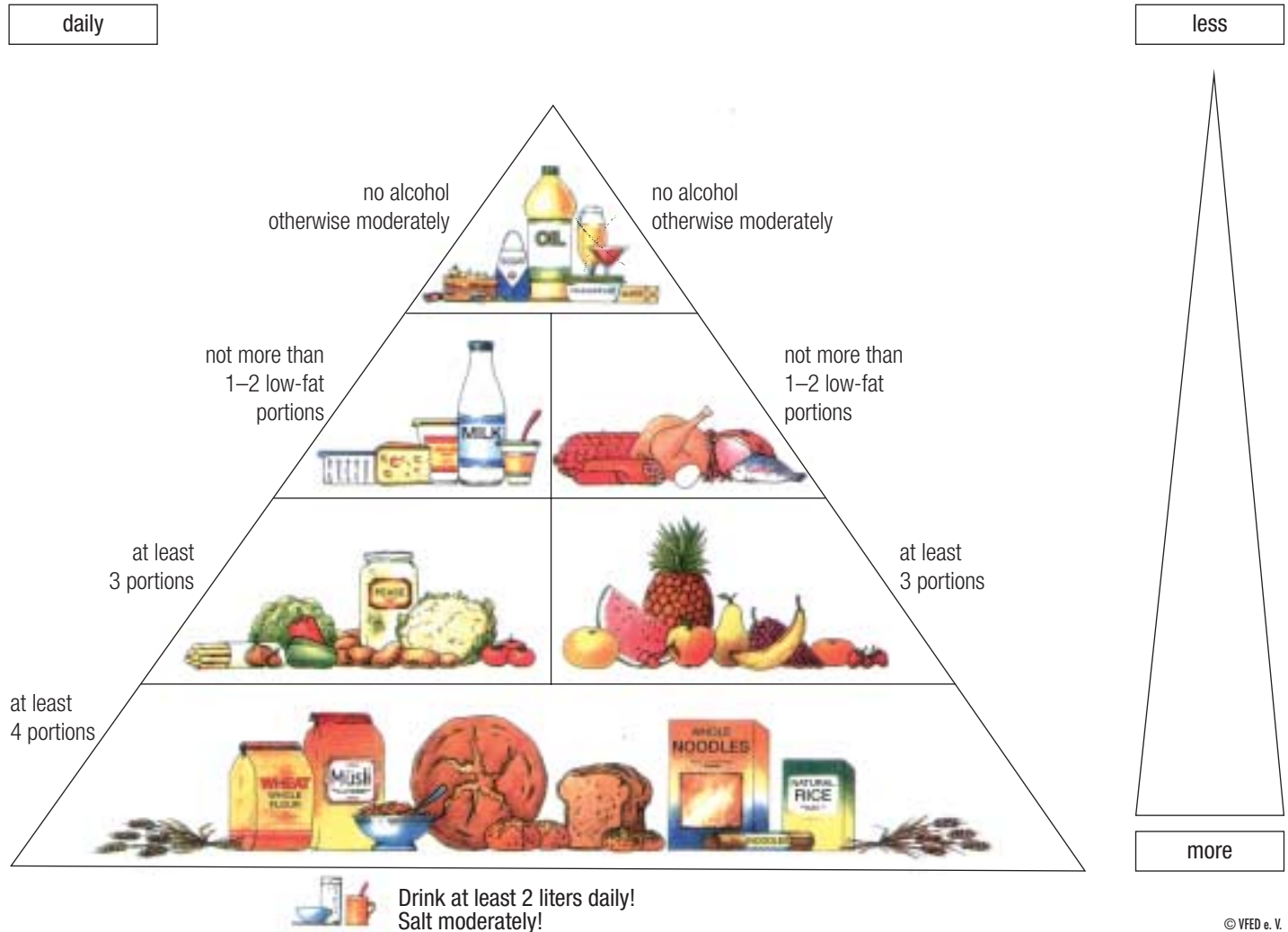
### **5.4 Protein and hepatic encephalopathy**

In the previous sections, we have mentioned several times that patients with liver cirrhosis are often affected by a loss of protein, leading to muscle loss, weakness and reduced immune functions (infections). Research has shown that patients with liver cirrhosis are very similar to malnourished persons in terms of their protein metabolism.

## The nutrition pyramid of the VFED e.V.

(VFED – Verband für Ernährung und Diätetik e.V.)

Choose wisely when it comes to eating and drinking



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Experts recommend the following protein intakes:

- 1.2 g of protein per kg body weight each day in compensated liver cirrhosis
- 1.5 g of protein per kg body weight each day in decompensated liver cirrhosis and malnutrition

In no case should one respond automatically to a diagnosis of liver cirrhosis with restriction in protein intake. It is not difficult to understand that a person with a protein deficient diet is further jeopardized by restrictions in protein intake.

Physicians are often concerned that an adequate protein intake may trigger hepatic encephalopathy. This fear is based on experiences with a very small group of problematic patients in whom protein intake at recommended levels does result in encephalopathy. This is the “protein dilemma” in which adequate protein is good for malnutrition but bad for encephalopathy and vice-versa. This dilemma, however, does not apply in 99% of patients with liver cirrhosis, in whom other triggers, such as infection, bleeding, drugs, renal failure, electrolyte imbalance and constipation, are present.

Beside the amount, the quality of protein also is important. This is especially true for all sick persons and very especially for persons with liver disease.

#### *5.4.1 Well tolerated and poorly tolerated protein*

Even though hepatic encephalopathy is rarely triggered by excess dietary protein, it is still important to distinguish between well and poorly tolerated proteins.

Proteins differ in terms of their chemical structure and exert different effects on brain function. For example, one gram of blood protein is much more likely to trigger hepatic encephalopathy than 1 g of vegetable protein. This is of importance because bleeding esophageal varices can result in large losses of blood into the stomach and bowel. This blood is then digested with the food in the small bowel and absorbed. This results in the formation of toxic breakdown products such as ammonia and to a more severe imbalance in the amino acid content of the blood. One result of this imbalance is a disturbance of the important synthesis of the body's own proteins.

An imbalance in amino acids does not occur solely after such bleeding, but actually occurs in all cirrhosis patients as a consequence of disturbed liver function and the detour of portal blood through the collateral circulation. Patients with cirrhosis are deficient in branched-chain amino acids (BCAA), but have an excess of aromatic amino acids (AAA). In addition, the amino acids glutamic acid, methionine and sometimes cysteine are elevated in the blood of cirrhotic patients.

### **BCAA**

Metabolism

- independent of liver function
- predominantly in the musculature
- useful for detoxification

Blood level reduced in cirrhosis

Useful in encephalopathy

### **AAA**

Metabolism

- dependent on liver function
- predominantly in the liver

Blood level increased in cirrhosis

Unfavorable in encephalopathy



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**Poor tolerance**

**Aromatic amino acids  
(AAA)**

**Blood**

**Meat/sausage**

**Fish/egg**

**Milk/dairy products**

**Vegetable protein**

**Branched-chain amino acids  
(BCAA)**

**Good tolerance**

Foodstuffs with a high BCAA content include milk and dairy products and vegetable foods. Meat and fish, on the other hand, have high AAA content. The BCAA (valine, leucine and isoleucine) counteract encephalopathy, while the AAA (tyrosine, phenylalanine) and methionine promote it. BCAA are metabolized independent of the liver predominantly in muscles and the level of waste products does not increase as a result of their being broken down. BCAA also inhibit the breakdown of protein in muscles.

**It is difficult to increase the dietary intake of BCAA significantly.**

This positive effect can be achieved by using special protein preparations (amino acid mixtures) containing a high proportion of BCAA. This is especially useful in patients on reduced-protein diets. In this situation, increasing the intake of BCAA can lead to turning protein metabolism from a negative balance to neutral or even a positive balance without an increased risk of hepatic encephalopathy.

Recent studies in Italy and Japan suggest that the intake of BCAA is helpful even in patients with liver cirrhosis who do not suffer from protein intolerance.

*Ask your physician or pharmacist about dietary foods with a high proportion of branched-chain amino acids or about drugs with BCAA.*

As you can see, all proteins are not created equal. Dietetic foods and drugs with a high proportion of branched-chain amino acids require a physician's prescription. As a rule, patients take 0.2 g of BCAA per kg body weight each day. A patient weighing 70 kg, therefore, would re-

quire 14 g of BCAA per day. When BCAA are prescribed, they are included in the daily protein intake. The intake of dietary protein is accordingly reduced or the amount of BCAA is subtracted from the difference between tolerated and recommended daily protein allowances.

**Example:**

71 kg patient ( $71 \times 1.2$ )	
Protein requirement	= 85.2 g
Tolerated protein amount	
= 0.8 g per kg body weight	
( $71 \times 0.8$ )	= 56.8 g
<hr/>	
Difference	= 28.4 g

About 30 g of BCAA must be taken daily.

Besides protein in blood, and to a decreasing degree, the proteins in meat, fish and eggs are unfavorable in hepatic encephalopathy, while, and to an increasing extent, the proteins in milk, dairy products and plants are considered favorable. Caution is required in purely vegetarian diets because, as a result of their high fiber content, nitrogen excretion in the stool is increased. This can result in a situation similar to a reduced-protein diet and can make the protein deficit even worse.

#### 5.4.2 Parenteral nutrition in coma

Coma, more specifically *hepatic coma*, is the most profound form of hepatic encephalopathy and is characterized by complete unconsciousness. In this situation, nutrition is usually provided by infusion directly into the circulatory system. This is called “parenteral” nutrition. The supply of amino acids is adapted to the liver. Energy requirements are supplied with carbohydrates and fats.

Enough energy must be supplied to prevent the breakdown of the body's own proteins. Carbohydrates are given as glucose. Parenteral nutrition also includes essential vitamins and minerals.

When the symptoms improve and the patient regains consciousness, it is important to return as soon as possible to the natural route of nutrition. On the first day of oral nutrition, protein intake should be 1 g per kg body weight and this increases over the next days to the recommended daily allowances. Reduced protein diets are justified only in the very rare cases of true protein intolerance. This must always be determined individually. In such cases, oral supplements with branched chain amino acids (BCAA) can be immediately started.

## **5.5 Carbohydrates**

Carbohydrates are the main source of energy for the body. Like fat they do not raise the levels of toxins in the body. 1 g of carbohydrate provides the body with 4 kilocalories (kcal). Foods that are rich in carbohydrate include: sugar, sweets, fruit, bread, foods made with flour, potatoes, milk and vegetables.

### *5.5.1 Roughage reduces the "toxin level"*

Non-digestible roughage is also classed among the carbohydrates. Roughage (or fiber) consists of those parts of vegetable foodstuffs that cannot be utilized by the human body. Roughage promotes digestion, slows the rise in blood sugar, reduces the level of cholesterol and improves the sensation of satiety. For patients with cirrhosis of the liver it is of particular importance that it binds toxins in the bowel. The intestinal transit time is

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also improved, reducing the amount of toxins formed. The food pulp moves more rapidly through the bowel and, as a result, smaller amounts of toxins are formed and absorbed. The danger of hepatic encephalopathy is reduced. A high-fiber diet often has side effects (bloating, sensation of fullness or abdominal pain). The body must gradually accustom itself to a high-fiber diet. It is important to take enough fluid because roughage needs to absorb water and bulk. If fluid is restricted, a high-fiber diet is not feasible. In that case, concentrates of roughage (e.g. *Plantago ovata* seed shells, bran products, oatmeal bran, pectin concentrates) cannot be used either. Foods that are high in fiber include: wholemeal bread, crispbread, fruit, vegetables, potatoes, brown rice, wholemeal noodles or bran.

#### *5.5.2 Use of lactulose preparations in cirrhosis of the liver*

Lactulose is an artificial disaccharide, which is made of fructose (fruit sugar) and galactose. Humans are unable to break down the sugar because they lack the required digestive enzyme. Lactulose reaches the large bowel intact where it is broken down to acetic acid and lactic acid by bacteria. Acidification of the bowel content changes the bacterial flora in the bowel. Bacterial digestion of protein is reduced. The production and absorption of ammonia and the formation of other toxins in the bowel is inhibited. The level of toxins in the patient falls and this leads to a decrease in symptoms of toxicity. The transit time for the unformed stool is reduced. As a result, toxins arising in the bowel or found in the stool are absorbed from the bowel in reduced amounts. Side effects such as bloating and diarrhea generally disappear after a short period of acclimatization.

Lactulose serves as a nutrient medium for bacteria in the bowel. These bacteria utilize nitrogen, which is thus not available for the production of ammonia. The less ammonia that is formed, the less that can be absorbed. The exact mode of action of lactulose is not yet known. Taking lactulose is harmless if you keep to the dose recommended by your doctor. Long-term use is also safe. Lactulose can be given as a deep enema or orally as a syrup or granulate. Lactulose granules have the advantage of tasting less sweet than lactulose syrup. The efficacy of lactulose preparations is extraordinarily good and they are often successfully used with protein-modified and protein-restricted diets to combat hepatic encephalopathy. Although lactulose is a carbohydrate, it does not need to be included in the calculation of carbohydrates for diabetics. The reason for this is that lactulose is not absorbed and thus cannot increase the blood sugar. The dose of syrup is usually 15–50 ml three times a day. For the granulate, the dosage is 6 g of lactulose 3–5 times a day. The dosage must be established by your physician on the basis of symptoms. The aim is to produce three soft stools a day.

In addition to lactulose preparations there are also preparations of lactitol. Lactitol is also a carbohydrate. Its mode of action is similar to that of lactulose.

## **5.6 Fats**

At 9 kilocalories (kcal) per gram, fat is the most energy-rich foodstuff. Fat does not increase toxic levels of ammonia in hepatic encephalopathy. It is used as a source of energy and as an energy store. The intake of animal fats should not be too high and the intake of vegetable fats should not be too low. In about 40% of the patients suffering from cirrhosis of the liver the digestion of fats is

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disturbed because of poor fat utilization and absorption. This may also affect the absorption of fat-soluble vitamins (A, D, E and K), which may lead to deficiency and must be supplemented parenterally in these patients.

In steatorrhea, special fat (MCT-fat) can be used. MCT is the abbreviation for **m**edium **c**hain **t**riglycerides. MCT-fats can be absorbed in the bowel even in the absence of bile acids and reach the body as a source of energy. MCT-fats do not naturally occur in foods.

MCT-fats can be obtained in the form of “Ceres MCT Diet Margarine” and “Ceres MCT Diet Cooking Oil” from the Union Deutsche Lebensmittelwerke (for the address, see the appendix). Special MCT fats, such as margarines, oil and special dietetic MCT foods such as soft cheese, hazelnut nougat creme etc., can also be obtained from the firm of Basis Diät GmbH.

It is important not to overheat “Ceres” fats. The MCT-fats produced by Basis Diät GmbH can be heated to higher temperatures than “Ceres” products and can be used for brief frying as well as for baking (for address, see appendix).

MCT fats must be administered using a phased increase in dose. If steatorrhea is present, the diet must be low in fat and the intake of fat must largely take the form of MCT fats.

If you have to have a MCT diet, you should discuss with your doctor or dietician how to obtain sufficient energy/calories despite the low-fat diet. Use of liver-specific liquid or tube diets is often of help here.

## **5.7 Minerals, vitamins and water**

### *5.7.1 Sodium*

In the treatment of ascites and edema, restriction of sodium and fluids constitutes the basis of treatment. Sodium most commonly occurs in table salt, known chemically as sodium chloride since it consists of sodium and chloride. In order to determine the sodium proportion of the table salt content, the table salt content must be divided by 2.5 (Example: 1 g table salt means 400 mg of sodium). To determine the table salt content from the sodium portion, the sodium portion is multiplied by 2.5 (Example: 400 mg sodium mean 1 g of table salt). Salt binds water in the body. The intake of salt is always accompanied by an increased thirst.

All patients with cirrhosis should, as a rule, be advised to use less salt in order to inhibit the development of ascites or edema.

#### *5.7.1.1 Sodium-defined diets:*

- Strict low sodium diet (1 g of table salt per day)
- Low sodium diet (3 g of table salt per day)
- Sodium-reduced diet (6 g of table salt per day)

As a rule, a low sodium or sodium-reduced diet is given in hospital. At home, a sodium-reduced diet has proved successful.

There are a whole series of low sodium and sodium-reduced products that will make it easier for you to stick to a sodium-reduced diet. Ask about them in your specialist shop, in the health food shop or at your pharmacist.



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A **strict low sodium diet** can only be maintained for a short period of time with inpatient care in hospital. It is possible to achieve a loss of up to 500 ml of edema fluid per day with a strict low sodium diet. Special low sodium products (e.g. bread) are a component of this type of diet. With such dietary restrictions, a varied diet is not possible. In many hospitals, a fruit-rice diet, which is extremely low in sodium, rich in potassium and low in protein is given for a few days to get rid of edema.

In the hospital, a **low sodium diet** is often prescribed to get rid of edema. This diet requires the use of low sodium foods and the use of special low sodium products. As a rule, the sodium content of animal products is high. For example, 1 liter of milk contains 1.2 g of table salt.

At home only a sodium-reduced diet is feasible in the long term. This basically entails doing without all high sodium foodstuffs and the addition of salt to foods.

### *High sodium foods*

Particularly high in sodium are: ready-to-serve meals, salt herrings, pre-prepared salads, tinned vegetables, fast food, Matjes (young) herring, sausage, cheese, ready-made soups, ready-made sauces and snacks.

### *High sodium foods*

		sodium content
100 g	Emmental cheese	450 mg
100 g	hard cheese	1520 mg
100 g	mayonnaise	702 mg
100 g	caviar	1940 mg
100 g	Matjes (young) herring	2500 mg
100 g	pickled herring	5930 mg
100 g	corned beef	833 mg
100 g	cervelat sausage	1260 mg
100 g	bacon	1770 mg
100 g	mustard	1307 mg

### *High salt products*

Adherence to a sodium-reduced diet is made easy by the use of predominantly fresh or deep frozen products. You should avoid use of tinned vegetables, ready-made soups and ready-to-eat meals. Remember that soup seasonings (e.g. Maggi), garlic salt, herbal salt, glutamate, soya sauce, beef cubes, seasonings, mustard and ketchup also contain large quantities of sodium.

### *Herbs instead of salt*

In order to make your food tasty, liberal use of herbs and spices is recommended. The taste of foods can also be improved with garlic, leeks, onions, tomatoes, low sodium mustard or horseradish. Wholegrain products have a more intense taste than products made with white flour. Salt substitutes can also help but these are often said to have a soapy taste.



### *5.7.1.2 Low sodium mineral waters*

Mineral water labels include an analysis of the sodium content. Mineral waters up to 100 mg sodium per liter are permitted. Inexpensive mineral waters are often low

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in sodium. Low sodium mineral waters are defined as those containing less than 20 mg sodium per liter.

*Low sodium mineral waters:*

Adelholzer  
Apollo Quelle (Apollo Spring)  
Bad Brückenauer  
Brückenauer Wernarzer Brunnen  
Contrex  
Kloster Quelle (Kloster Spring)  
Marco Heilwasser (Marco mineral water)  
Mathildenquelle  
Perrier  
Rietenauer Heiligenthalquelle  
Rietenauer Kneipp-Quelle  
Sinziger Mineralwasser  
St. Linus  
Volvic  
Wildungen Reinhardsquelle

*5.7.1.3 High sodium mineral waters*

You should avoid high sodium mineral waters with more than 500 mg sodium per liter if you are keeping to a low sodium diet.

*High sodium mineral waters:*

Aachener Kaiserbrunnen  
Apollinaris  
Bad Mergentheimer Karlsquelle  
Bad Mergentheimer Wilhelmsquelle  
Brohler Sprudel  
Fachinger, Staatl. (state-certified)  
Kaiser Friedrich Quelle (Kaiser Friedrich Spring)  
Romina Friedrichsquelle  
Selters, Staatl. (state-certified)

### *Low sodium foods*

There are a whole series of low sodium and sodium-reduced products which will definitely make it easier for you to keep to a sodium-reduced diet. Your dietician will be pleased to advise and assist you concerning the products and where to get them (often the health food shop).

### *5.7.2 Potassium*

Salt substitutes generally contain potassium in place of sodium compounds. In addition to an improvement in taste, they have the advantage of the high potassium content. A potassium-rich diet is particularly important for patients who take diuretics to get rid of fluid, as potassium deficiency can otherwise occur.

Particularly rich in potassium are all types of vegetables (particularly cabbage, potatoes, herbs, tomatoes, spinach, tomato pulp, mushrooms and chanterelles), fruit (particularly avocado, apricots, bananas, fruit juices and dried fruit).

When edema is present, fluid intake must be restricted so that a potassium-rich diet must often fall through. Potassium-rich foods also contain a lot of water.

### *5.7.3 Vitamins and minerals*

Patients with cirrhosis of the liver often show a deficiency in minerals (zinc, iron, calcium, potassium) and vitamins (A, D, E, K, folic acid, B<sub>1</sub>, B<sub>2</sub>, B<sub>6</sub>, B<sub>12</sub>). Because supplementation in the form of tablets, capsules or drops is much easier than the demonstration of a deficiency,

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some experts recommend the pragmatic solution of simply prescribing these preparations.

Many patients with cirrhosis of the liver suffer from a marked zinc deficiency. When zinc is given in tablet form, hepatic encephalopathy often improves. Particularly good here are zinc tablets containing organic zinc compounds such as zinc histidine, which are more reliably absorbed from the bowel than inorganic zinc salts.

#### *5.7.4 Supply of fluid*

A restriction in the amount of fluid drunk is only required if the level of sodium in the blood is too low or in case of edema or ascites. The amount consumed should be reduced to 500–1000 ml. When the fluid intake is low, only drinks that quench the thirst should be chosen. Milk, mixed drinks, sweetened soft drinks or teas, and high sodium mineral waters are not appropriate. Mineral water, which is also used to supply the calcium requirement, is thirst quenching.

In all other cases the amount of liquid taken should be 1.5–2 liters, as with healthy people.

#### *High calcium mineral waters:*

Kloster-Quelle (Kloster Spring)

Marco Heilwasser (Marco Mineral Water)

Rietenauer Heilwasser (Rietenauer Mineral Water)

Rietenauer Kneipp Quelle (Rietenauer Kneipp Spring)

Steinsieker Mineralwasser

Mineral water is considered rich in calcium if it contains more than 150 mg of calcium per liter. The calcium content is written on the bottle label. Some kinds of mineral

water contain even more than 500 mg of calcium per liter and thus play an important part in meeting calcium requirements.

It is important to note that many foodstuffs have a high water content (e.g. fruit, vegetables, soups, yoghurt, milk or pudding). The daily fluid intake and body weight in ascites should be carefully recorded.

### **5.8 Soft diet**

The importance of soft or strained foods in the prophylaxis of variceal bleeding has not been proven. What is certain is that sufficiently small, careful chewed and well moistened foods are better tolerated and more efficient. In all disorders of the esophagus, you should consider the temperature (lukewarm is best, avoid very hot or very cold) and aggressiveness (acid, hot spices) of the food.

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## 6 Summary

There are no absolute restrictions in diet for patients with liver disease and in general they should follow the principles of a balanced, healthy diet. It is essential, however, that patients with diseases of liver absolutely avoid alcohol in any form.

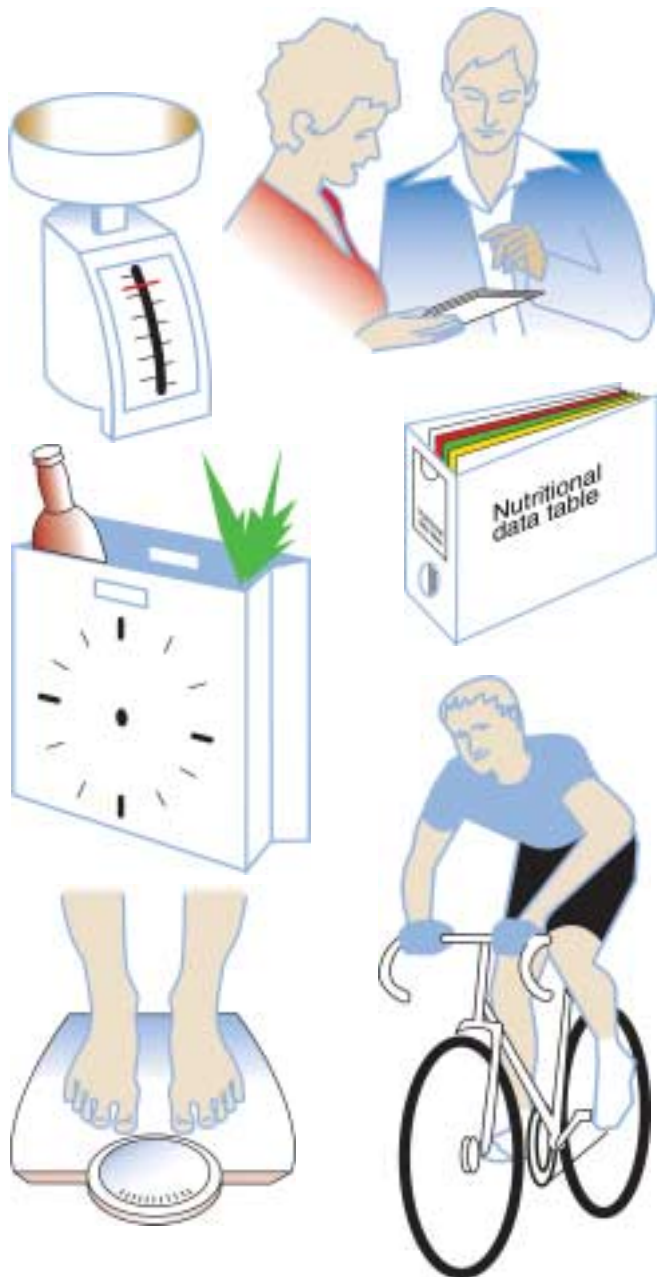
Liver patients with advanced disease are threatened as their disease progresses with malnutrition, which can be addressed with the following measures:

- Adequate caloric intake (35 kcal per kg body weight daily)
- Adequate intake of protein (1.2–1.5 g per kg body weight daily)
- Adequate intake of vegetable fiber or roughage
- Regular exercise to maintain muscle mass
- Timely addition of enteral dietary supplementation
- Timely addition of branched-chain amino acids

The implementation of these recommendations demands from each patient a fundamental change in his lifestyle and required much motivation and cooperation. This is easier the more one understands the reasons for these recommendations and their purpose. This is the purpose of this brochure and we hope that it proves helpful to these patients.

It cannot and should not replace the consultation of your physician and dietician. Interaction with these professionals should start during your hospitalization and continue on an outpatient basis after discharge.

## Your tools for correct nutrition





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## 7 Appendix

### 7.1 Personal levels of tolerance

My protein restriction: \_\_\_\_\_

My calorie requirement: \_\_\_\_\_

Calorie requirement:

Normal weight times 35 for moderate activity

My normal weight: \_\_\_\_\_

(Dry weight without ascites)

Normal weight: height in centimeters minus

100 = normal weight in kilograms

Fluid: \_\_\_\_\_

CE (carbohydrate equivalent) –

distribution for diabetics

Total CE-amount: \_\_\_\_\_

Notes:

## 7.2 Protein exchange table

### Protein exchange table

*10 g of protein are contained in:*

#### Milk and dairy products

- 285 ml buttermilk
- 300 ml cow's milk (3.5% fat)
- 300 g yoghurt (3.5% fat)
- 360 g sour cream

#### Cheese and eggs

- 35 g Emmental (45% fat by dry weight)
- 35 g hard truckle cheese (ca. 1% fat by dry weight)
- 40 g Edam (45% fat by dry weight)
- 40 g Gouda (45% fat by dry weight)
- 45 g Brie (50% fat by dry weight)
- 45 g Camembert (30% fat by dry weight)
- 50 g Camembert (45% fat by dry weight)
- 55 g Camembert (60% fat by dry weight)
- 70 g Soft cheese (45% fat by dry weight)
- 75 g Low-fat curd cheese (skimmed)
- 80 g Whole egg (equals about 1½ class 4 or 5 eggs)
- 90 g Double cream cheese (60% fat by dry weight)
- 90 g Quark (40% fat by dry weight)

#### Fats and oils

- 900 g mayonnaise
- 1430 g butter
- 5000 g margarine

*Edible oils do not contain protein.*

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## **Fish, seafood and fish products**

50 g	trout
50 g	sardines in oil
60 g	grilled herring
60 g	herring (fillet)
60 g	cod
60 g	crabs
60 g	plaice
70 g	eel
70 g	herring in tomato sauce
70 g	caviar (mock, real caviar 40 g)

## **Poultry**

45 g	chicken (breast)
50 g	turkey (breast)

## **Veal**

50 g	fillet
50 g	joint
50 g	cutlet

## **Beef**

45 g	mince
45 g	tatаре (minced steak)
50 g	fillet
50 g	roast beef

## **Pork**

50 g	chop
50 g	cutlet
50 g	liver
55 g	fillet
60 g	mince
85 g	Eisbein (pickled knuckle)
243 g	back bacon

## Sausage

35 g	ham (uncooked, without fatty coat)
50 g	mince (half and half)
50 g	ham (cooked)
55 g	salami (German)
60 g	cervelat
60 g	liver sausage (lean)
65 g	ham sausage
75 g	black pudding
75 g	(meat) sausage
75 g	frankfurter
80 g	smoked sausage
80 g	liver sausage (full fat)
80 g	Bologna sausage (Brunswick)
80 g	mortadella
85 g	meat loaf

## Cereals, bread and baking materials

80 g	oats
90 g	wheat
95 g	wheat flour (type 405)
135 g	natural rice (uncooked)

*Starches and blancmange powders contain small traces of protein.*

75 g	average for noodles (uncooked)
100 g	average for muesli
100 g	rusk
135 g	average for bread

## Pulses, nuts and seeds

35 g	sunflower seeds (shelled)
40 g	peanuts (roasted)
40 g	average for pulses
50 g	pistachios
55 g	almonds
75 g	hazel nuts

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## **Vegetables and mushrooms**

All types of vegetable and mushroom are relatively low in protein and, as a rule, contain no more than 2–3% of protein so that 200 g portions seldom contain more than 5 g of protein. Peas are the most high protein vegetable. 200 g of peas contain 11.6 g of protein. Vegetables and mushrooms contain large amounts of roughage.

## **Fruit and fruit products**

All types of fruit and fruit products (e.g. jams or fruit juices) are low in protein and, as a rule, contain no more than 0.3–3% protein so that a piece of fruit (150 g) seldom contains more than 3 g of protein. Fruit contains large amounts of roughage.

### 7.3 Dietary timetable

Meal	Food	Quantity of food (in grams)	Protein content
<b>Breakfast:</b>			
<b>Snack (elevenses):</b>			
<b>Lunch:</b>			
<b>Snack (tea):</b>			
<b>Evening meal:</b>			
<b>Snack (supper):</b>			
<b>TOTAL PROTEIN CONTENT:</b>			<b>GRAMS</b>

## 7.4 Overview of foods

The following summary is intended to give you a brief overview of suitable and less suitable foods. This summary should under no circumstances take the place of your tools (protein exchange table, diet plan, scales and a table of nutritional values).

Food group	+ suitable	- less suitable	Remarks
Meat (including game), offal and fish	Fatty varieties contain less protein!	Preserved e.g.: young (Matjes) or pickled herring, caviar	P, Na
Sausages	High fat varieties of sausage contain less protein!	Highly salted e.g. ham, salami, Buendner Fleisch [dry cured beef]	P, Na
Milk and milk products	High fat varieties of cheese contain less protein!	Highly salted. Large quantities of quark are bad!	P, Na, F
Fats	All types. There is no objection to butter! A larger amount of fat meets the energy requirement.	Salted fat for spreading	Na
Eggs and poultry	High fat varieties of poultry contain less protein!	Highly salted	P
Vegetables	<b>All varieties within the limits of fluid tolerance.</b> Roughage lowers the level of toxins!	Salted preserves and highly salted vegetables (e.g. olives). Soya and soya products contain a lot of protein!	F, Na

Food group	+ suitable	- less suitable	Remarks
Potatoes	<i>All varieties.</i> Roughage lowers the level of toxins!	Highly salted potatoes, crisps, chips	Na
Fruit	<b><i>All varieties within the limits of fluid tolerance.</i></b> Roughage lowers the level of toxins!		F
Nuts	In normal quantities. Roughage lowers the level of toxins!	Highly salted e.g. peanuts	Na
Cereals and bread	All sorts. Roughage lowers the level of toxins!		P, Na
Sugar and sweets	In normal amounts	Sweets with a lot of sugar, protein, ice-cream, chocolate or cheesecake. Look at the table of nutritional values!	P
Drinks	<b><i>Within the limits of fluid tolerance</i></b>	Alcoholic drinks of all types are strictly forbidden!	F, A
Herbs, salt and spices	Herbs and spices	Salt and all salt-containing products e.g. ready made sauces	Na
Ready-to-cook meals	Pay attention to the analysis on the packet!	Highly salted	Na, P

Key:

A = alcohol, P = protein, F = fluid, Na = sodium



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## Methods of preparation

The following is a list of the types of cooking that are generally well tolerated (+) and those that are less well tolerated (-).

- + boiling, baking, steaming, microwave, baking bag (plastic tube for baking in the oven), chicken brick (Römertopf), aluminium foil, and pressure cooker
- frying, roasting, stewing

Please note that this advice may not be appropriate to the same extent for all patients. If you can tolerate roast meat (in the framework of protein tolerance!), then of course you can eat it. Try out and see what you can tolerate and what you can't. But do not try everything at once. As a rule, methods of cooking that require a lot of fat (e.g. frying), or which result in a lot of production toxins (e.g. grilling), are poorly tolerated.

## 7.5 80 g Protein diet

Note: This diet plan is adequate for a daily protein allowance of 1.2 g per kg body weight in a 67-kg person and an overall caloric requirement of 35 kcal per kg body weight. It serves as an example of nutrition in the stage of compensated liver cirrhosis.

<b>Breakfast</b>	<b>Protein content (g)</b>
50 g mixed bread (1 slice)	3.5 g
50 g wholemeal bread (1 roll)	4.2 g
20 g fat for spreading (e.g. butter)	0.1 g
25 g jam/marmalade	0.5 g
30 g meat paste (e.g. Bologna sausage)	6.6 g

Coffee or tea

<b>Snack</b>		<b>Protein content (g)</b>
150 g	yoghurt with fruit (1 small bowl)	5.8 g
<b>Lunch</b>		
200 g	potatoes, rice or noodles	4.2 g
250 g	vegetables (e.g. green beans)	6.0 g
100 g	meat (e.g. Wiener Schnitzel)	14.3 g
10 g	cooking fat (e.g. soy oil)	0.0 g
120 g	blancmange/flummery	3.5 g
<b>Afternoon</b>		
100 g	fruit cake (1 largish piece)	2.5 g
<b>Evening meal</b>		
50 g	wholemeal bread (1 slice)	3.8 g
50 g	mixed bread (1 slice)	3.5 g
20 g	fat for spreading (e.g. butter)	0.1 g
30 g	meat paste (e.g. liver sausage)	10.1 g
40 g	Camembert (40% fat)	8.1 g
100 g	raw salad with yoghurt dressing	3.2 g
<b>Supper</b>		
140 g	fruit (e.g. 1 pear)	0.7 g

*Analysis:*

2400 kilocalories, protein: 80.7 g, fat: 105 g, carbohydrate: 260 g, fiber: 32 g, sodium: 2.6 g, potassium: 3.3 g.

Note for **diabetics**: replace sugar and honey with saccharin and use diabetic jam.

CE – distribution:

4.5 CE, 1 CE, 4 CE, 3 CE, 4 CE, 1 CE (total: 17.5 CE).

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## 7.6 60 g Protein exchange diet, sodium-reduced

Note: This diet plan is **not** adequate for achieving a daily protein allowance of 1.2 g per kg body weight for a 67-kg person and a total caloric intake of 35 kcal per kg body weight. It can be used as an example of a diet to be used in the very rare instances of true protein intolerance. The daily protein deficit of 20 g must be substituted with a corresponding amount of BCCA.

<b>Breakfast</b>		<b>Protein content (g)</b>
50 g	bread roll (1 roll)	3.9 g
50 g	mixed bread (1 slice)	3.5 g
20 g	fat for spreading (e. g. butter)	0.1 g
25 g	jam/marmalade	0.5 g
5–6 g	protein (see protein exchange table)	
for example:		
30 g	corned beef	5.6 g
	coffee or tea with milk and sugar	0.4 g
<b>Snack</b>		
130 g	fruit/stewed fruit (e. g. 1 apple)	0.3 g
<b>Lunch</b>		
250 g	vegetables (e. g. carrots)	2.5 g
200 g	potatoes, rice or noodles	4.2 g
10 g	cooking fat (e. g. soya oil)	0.0 g
15 g	protein (see protein exchange table)	
for example:		
75 g	pork cutlet	15.6 g
150 g	fruit/stewed fruit	0.9 g
20 g	cream (30% fat)	0.5 g

**Afternoon**

5–6 g protein (see protein exchange table)

for example:

150 g	cream yoghurt with fruit	5.6 g
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**Evening meal**

100 g	mixed bread (2 slices)	7.0 g
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20 g	fat for spreading (e.g. butter)	0.1 g
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5–6 g protein (see protein exchange table)

for example:

50 g	double cream cheese with herbs	5.5 g
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100 g	vegetables (as salad, e.g. tomatoes)	1.0 g
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10 g	oil (as dressing, e.g. sunflower oil)	0.0 g
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**Supper**

130 g	fruit/stewed fruit (e.g. 1 banana)	1.4 g
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*Analysis:*

2450 kilocalories, protein: 58.6 g, fat: 115 g, carbohydrate:

270 g, fiber: 34 g, sodium: 1.9 g, potassium: 3.7 g.

Note for **diabetics**: replace sugar and honey with saccharin and use diabetic jam.

CE-distribution:

4.5 CE, 1 CE, 5 CE, 1 CE, 4 CE, 1 CE (total: 16.5 CE).

Note for patients who are not suffering from ascites: season sparingly with salt.

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## 7.7 Sources of information material

Dietary counseling:	Deutsche Gesellschaft für Ernährungsmedizin (DGEM) DGEM Infostelle Dipl. oec. troph. Brigitte Herbst Olivaer Platz 7 D-10707 Berlin Germany Tel.: ++49 (30) 8891-2852 Fax: ++49 (30) 8891-2839 E-mail: infostelle@dgem.de www.dgem.de
	Deutsche Gesellschaft für Verdauungs- und Stoffwechselkrankheiten (DGVS) Geschäftsstelle Olivaer Platz 7 D-10707 Berlin Germany Tel.: ++49 (30) 8877-4784 Fax: ++49 (30) 8877-4786 E-mail: info@dgvs.de www.dgvs.de
Carbohydrate exchange table:	VFED e.V. St. Franziskus-Krankenhaus Morillenhang 27 D-52074 Aachen Germany Tel.: ++49 (241) 507300 Fax: ++49 (241) 507311 E-mail: info@vfed.de www.vfed.de

- Referrals for independent dieticians: VFED e.V.  
Tel.: ++49 (2 41) 50 73 00  
www.vfed.de
- Maltodextrin 19: SHS-Gesellschaft für klinische Ernährung mbH  
Postfach 30 61  
D-74020 Heilbronn  
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Fax: ++49 (71 31) 58 30 71
- Nutricomp Hepa: B. Braun Melsungen AG  
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## Further information for patients with liver diseases:



**Primary biliary cirrhosis (PBC)**  
**Primary sclerosing cholangitis (PSC)**  
Author: U. Leuschner  
36 pages (U82e)

This brochure can be ordered **free of charge** from Falk Foundation e.V. or the local Falk partner.

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