Table 15.4: Copper content of foods

Foods High in Copper (to be avoided)	Foods Low in Copper (desirable)
Meat: organ meats, lamb, pork, fish, liver and shellfish	Eggs
Milk: chocolate, cocoa and soyamilk, tofu	All other dairy products
Pulses: Beans Peas and lentils	Bread and pasta from refined flour, rice, sweet potatoes
Cereals: Bran containing cereals, soyaflour	All other vegetables including tomatoes
Vegetables: Mushrooms and juice	All other fruits (including jams, jellies) Lemonade and fruit flavoured beverages
Fruits: Dried fruits, dates, raisins and prunes	
Brewer's yeast	

Now, let us learn about the nutritional management of the last stage of liver disease, which is called hepatic encephalopathy or hepatic coma.

15.3.3 Dietary Recommendations for Hepatic Encephalopathy (HE) or Hepatic Coma

The nutritional management goals for hepatic coma include:

- reduction in protein intake to a minimum in order to decrease amount of ammonia produced,
- correcting plasma amino acid profile, and
- prevention of catabolism of tissue protein.

There is no general treatment. The diet needs to be individualized. The dietary recommendations include:

Calories: A 1500 to 2000 Kcal diet is recommended to prevent breakdown of tissue protein for energy. It is provided chiefly in the form of carbohydrates. It can be given by parenteral or tube feeding if needed. Carbohydrates help to build up liver glycogen reserves and have a protective role in the healing process.

Carbohydrates: An increase in carbohydrates in the diet is recommended because it is the main source of energy and thus spares the protein. It promotes glycogen repletion, which improves with carbohydrate adequacy. It also prevents hypoglycemia.

Proteins: It has not yet been proved that severe protein restriction improves the mental state of the patient in hepatic encephalopathy. Unnecessary protein restriction may only worsen body protein losses and therefore must be avoided. More than 95% of cirrhotic patients can tolerate mixed protein diets. The protein intake may begin with 0.2 g/ kg IBW /day. If the patient remains asymptomatic for a week it may gradually be increased by 10-15 g per week, and then 20-40 g and gradually to 0.5 g/kg IBW per day as indicated in Table 15.1 above.

Research postulates that vegetable proteins and caesin may improve mental status compared to animal protein. Vegetable based diets are lower in AAAs and higher in BCAAs than meat based diets. The potential advantage of vegetable protein is that it is low in methionine and ammoniogenic amino acids. The BCAA are desirable supplements in liver disease. These amino acids are metabolized by the muscles independent of the liver to provide energy, other amino-acids or small nitrogenous compounds and help in obtaining a positive nitrogen balance. Vegetables proteins are rich in BCAA. The common food sources rich in BCAA include dairy products and red meat. Whey protein and egg protein supplements are among the other sources.

Experts agree that BCAA enriched formulas should be indicated for patients with encephalopathy who do not tolerate standard proteins. Both enteral and parenteral BCAA supplement formulas are available commercially and could be used, if required.

An increase in BCAA helps in a number of ways. These include:

- enhances the uptake of AAA by muscles.
- increases protein synthesis in muscles.
- increases hepatic protein synthesis.
- reduces the cerebral AAA levels by competing for a common transport system across the blood brain barrier.

Fats: Fats require restriction, as diseased liver cannot metabolize fats. Substitution with MCT is recommended as they do not require bile salts and micelle formation for absorption and are readily taken up by the portal route.

Vitamins: Increase in intake of B-complex vitamins such as folate, thiamin, B_{12} and vitamin C is recommended as these vitamins act as coenzymes in various metabolic reactions.

Sodium: Depending on the state of the patient, a restriction of 2 g/day along with use of diuretics is recommended.

Fluid: Hyperaldosteronism is associated with liver failure, which results in increased renal sodium exchange for potassium. This urinary potassium loss further gets aggravated by diuretic therapy. There is an evident fluid retention. Thus, depending on the patients' state of hydration, urine output, presence of oedema and diuretic therapy, the fluid intake should be decided and recommended.

With this we end our study on the nutritional management of liver diseases. Try to recall what you have learnt so far by answering the questions given in check your progress exercise 2.

Check Your Progress Exercise 2

1. What dietary advice would you give to a patient suffering from viral hepatitis?

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2. Which is the last stage of liver disease? Discuss why BCAA are preferred over AAA? Also identify the food sources rich in BCAA.

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3. What is Wilson's disease? List at least five food items that must be avoided.

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4. Mention three foods that you would avoid in liver cirrhosis.

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