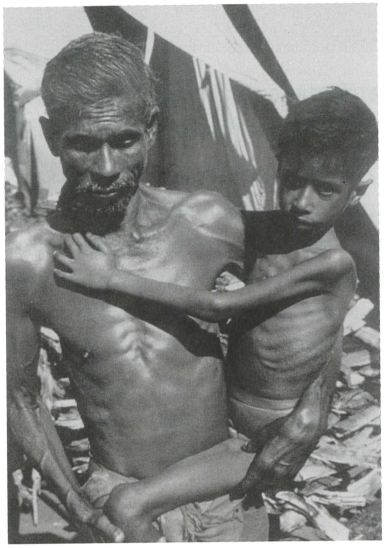


▲ Figure 6

- weight gain is associated with arteries becoming stiffer and narrower which can raise blood pressure.

Hypertension can also be caused by high salt intake. Circulating salt has an osmotic effect.

Effects of starvation

Starvation can lead to breakdown of body tissue.

Starvation occurs due to the severe lack of intake of essential and non-essential nutrients. In the absence of dietary intake of energy sources, the body will first access glycogen stores. However, if no glucose is available, the body will break down its own muscle tissue to utilize the resulting amino acids as energy sources. The amino acids are sent to the liver where they are converted to glucose. This results in a loss of muscle mass. In figure 6, the child is suffering from marasmus. His thin limbs indicate that muscle tissue has been broken down as an energy source by his body.

Anorexia

Breakdown of heart muscle due to anorexia.

The medical term anorexia means reduced appetite. Anorexia nervosa is a psychiatric illness, with causes that are complex. It involves voluntary starvation and loss of body mass. The amounts of carbohydrate and fat consumed are too small to satisfy the body's energy requirements, so protein and other chemicals in the body are broken down. There is wasting of muscles, resulting in loss of strength. Hair becomes thinner and can drop out. The skin becomes dry and bruises easily. A fine growth of body hair tends to develop. Blood pressure is reduced, with slow heart rate and poor circulation. In females, infertility is another common consequence, with no ovulation or menstrual cycles.

As body weight in a person with anorexia falls, not only is skeletal muscle digested, but heart muscle deteriorates. To some degree, the skeletal muscle mass reduces disproportionately faster than the cardiac mass. Lack of protein, electrolytes and micronutrients may result in the deterioration of muscle fibres. The lack of dietary intake also alters the electrolyte balance; i.e., concentrations of calcium, potassium and sodium. Both skeletal muscle and cardiac muscle do not contract normally under these circumstances. There is often reduced blood pressure, a slower heart rate and reduced heart output in patients.

Data-based questions: Changes in heart dimensions in patients with anorexia

The data in figure 7 shows the dimensions of various structures in subjects with normal diet and in patients with anorexia.

1 Calculate the percent change in the mean dimensions of
   a) the left ventricle
   b) the left ventricle wall
   c) the left atrium
   d) the base of the aorta. [5]

2 Identify the part of the heart with the largest decrease in dimension due to anorexia. [1]

3 Suggest what might be the symptoms of this change in the affected patient. [3]

		Left ventricle	Left atrium	Base of aorta	Ventricle wall
Normal	mean	47 mm	29 mm	27 mm	9 mm
	range	(35–57)	(19–40)	(20–37)	(6–11)
Anorexia	mean	38 mm	26 mm	21 mm	8 mm
	range	(38–44)	(17–34)	(18–26)	(6–9)

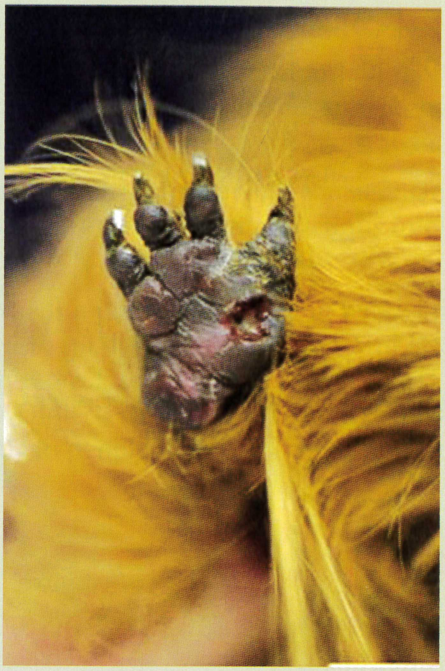
▲ Figure 7

The guinea pig as a model organism for studying scurvy

Falsification of theories with one theory being superseded by another: scurvy was thought to be specific to humans, because attempts to induce the symptoms in laboratory rats and mice were entirely unsuccessful.

In 1907, two scientists, Holst and Frolisch, published a research paper on their success in developing an animal model for the study of scurvy. They caused scurvy by feeding guinea pigs (*Cavia porcellus*) with whole grains. They cured scurvy in the guinea pigs through dietary modification including feeding fresh cabbage and lemon juice. The ideas within their paper were somewhat unpopular with the scientific community as the concept of nutritional deficiencies was unheard of at the time. The use of the term "vitamin" did not begin until later.

Their animal model allowed for the systematic study of the factors that led to the scurvy, as well as the preventive value of different substances. Substituting guinea pigs for pigeons, an animal model that had been used in beriberi research, was a lucky coincidence, as the guinea pig was later shown to be among the very few mammals capable of showing scurvy-like symptoms, while pigeons, as seed-eating birds, were later shown to make their own vitamin C and could not develop scurvy.



▲ Figure 8 Dermatitis in a guinea pig fed exclusively on rabbit pellets. This is one of a number of symptoms of scurvy seen in guinea pigs with the disease

Phenylketonuria

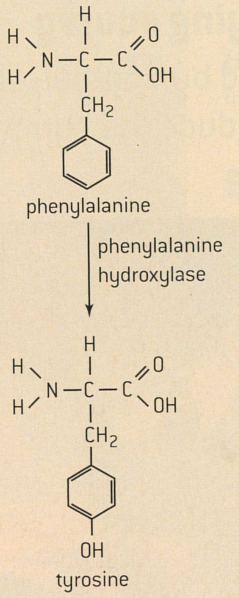
Cause and treatment of phenylketonuria (PKU).

Phenylketonuria (PKU) is a genetic disease. It is caused by mutations of a gene coding for the enzyme that converts phenylalanine into tyrosine.

The mutations produce alleles of the gene that code for enzymes unable to catalyse the conversion reaction. Only one normal allele is needed for satisfactory conversion of phenylalanine to tyrosine, so this allele is dominant. The symptoms of PKU only occur in individuals with two recessive mutant alleles. Phenylalanine then accumulates in the body and there can also be a deficiency in tyrosine.

The consequences of PKU are potentially very serious. The high phenylalanine levels cause reduced growth of head and brain, with mental retardation of young children and severe learning difficulties, hyperactivity and seizures in older children. Other consequences are a lack of skin and hair pigmentation.

PKU babies are unaffected at birth because the mother's metabolism has kept phenylalanine and tyrosine at normal levels. This gives an opportunity for early diagnosis and treatment. A test should be carried out at about 24 hours after birth, by which time blood phenylalanine concentrations will have started to rise. Treatment involves eating a diet low in phenylalanine for the rest of the person's life. Meat, fish, nuts, cheese, peas and beans can only be eaten in small quantities. Tyrosine supplements may be needed. If a suitable diet is rigorously adhered to, the harmful consequences of PKU can be avoided.



▲ Figure 9 Synthesis of tyrosine from phenylalanine

▲ Figure 10 A new born baby being tested for PKU using the Guthrie test

Vitamin D deficiency

Lack of Vitamin D or calcium can affect bone mineralization and cause rickets or osteomalacia.

Vitamin D is needed for calcium absorption from food in the intestines, so the symptoms of vitamin D deficiency are similar to those of calcium, with children developing the skeletal deformities known as rickets. Vitamin D does not fit the definition of a vitamin very well, as it can be synthesized in the skin. This only happens when sunlight, or another light source containing ultraviolet light with wavelengths in the range 290–310 nm, strikes the skin. If teenagers and adults spend enough time outside, with their skin uncovered, no vitamin D is required in the diet. Children, pregnant women and elderly people are recommended to eat 10 µg per day, to supplement the amount made in their skin. There are few dietary sources of vitamin D. Oily fishes including herring, mackerel, sardines and tuna are rich sources. Eggs and liver also contain some, and certain foods such as margarine and milk are artificially fortified with vitamin D.

Ultraviolet light has some harmful consequences, including mutations that can lead to skin cancer. Melanin in the skin intercepts and absorbs light, including the ultraviolet wavelengths. Dark skins therefore give good protection against cancer, but they also reduce vitamin D synthesis. In indigenous human populations, skin colour balances the twin risks of vitamin D deficiency and cancer or other damage due to ultraviolet light. After population migrations there can be problems. In the 1970s immigrants with dark skin from the Indian subcontinent living in the United Kingdom started to show symptoms of vitamin D deficiency. Immigrants from northern Europe with light skin living in Australia were found to have high rates of malignant melanoma. Australians with light skin were then advised to stay out of bright sunlight, cover their skin or apply sun-block creams.

Blood cholesterol and heart disease

Cholesterol in blood as an indicator of the risk of coronary heart disease.

Cholesterol is a normal component of plasma membranes in human cells, but nevertheless it has developed a reputation for being a harmful substance. This is because research has shown a correlation between high levels of cholesterol in blood plasma and an increased risk of coronary heart disease. Advice is often given to minimize dietary cholesterol intake, but it is not certain that this will actually lower the risk of coronary heart disease (CHD), for a variety of reasons.

- Much research has involved total blood cholesterol levels, but only cholesterol in LDL (low-density lipoprotein) is implicated in CHD.
- Reducing dietary cholesterol often has a very small effect on blood cholesterol levels and therefore presumably has little effect on CHD rates.
- The liver can synthesize cholesterol so dietary cholesterol is not the only source.
- Genetic factors are more important than dietary intake and members of some families have high cholesterol levels even with a low dietary intake.
- Drugs can be more effective at reducing blood cholesterol levels than reductions in dietary intake.
- There is a positive correlation between dietary intake of saturated fats and intake of cholesterol, so it is possible that saturated fats, not cholesterol, cause the increased risk of CHD in people with high cholesterol intakes.

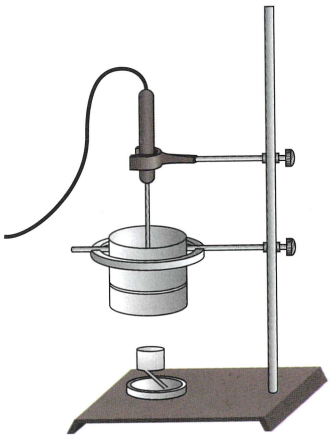
Calorimetry

Determination of the energy content of food by combustion.

The determination of the energy content of a substance is called calorimetry. Figure 11 shows an experimental set-up for a simple calorimeter. It is based on the knowledge of the specific heat capacity of water. It takes 4.186 J of heat energy to raise the temperature of 1 gram of water by 1 degree Celsius.

Q = mass of water × specific heat capacity × change in temperature

The apparatus consists of a thermometer to detect the change in temperature and a vessel containing a known mass of water (1 ml of water has a mass of 1 g). The sample to be tested for its energy content is ignited and placed below the container containing the water and the temperature change is noted.



▲ Figure 11

Activity

Using the experimental results below, estimate the energy content of the nut per gram.

Sample data:

Mass of nut = 0.60 g

Volume of water = 25 ml

Initial water temperature = 20 °C

Final water temperature = 65 °C