

## Energy stores in man, their composition and measurement

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The components of body-weight in a normal adult male are indicated diagrammatically in Fig. 1; roughly 42 kg water, 12 kg protein, 12 kg fat and 0.5 kg glycogen would be found in a normal male weighing 70 kg. The remainder of weight not accounted for by these four components (3.5 kg in this example) is mostly bone mineral, with small quantities of peptides, nucleotides and other compounds which, although they may be vitally important metabolically, are negligible as components of the energy stores of the body.

Complete combustion of the body indicated in Fig. 1 would yield about 660 MJ (158 Mcal), of which two-thirds would come from the fat. In an obese person weighing 100 kg the amount of fat in the body might well be trebled, and the energy content of the whole body would then be more than doubled.

This paper is about the composition and measurement of energy stores in man. Passmore (1965) made a distinction between 'stores' and 'reserves' thus: 'A store is an accumulation of something, which can be used in a time of emergency, whose loss in no way diminishes the ability of the body to do work and to meet stress and strain. A reserve may be defined as material available for use in an emergency, but

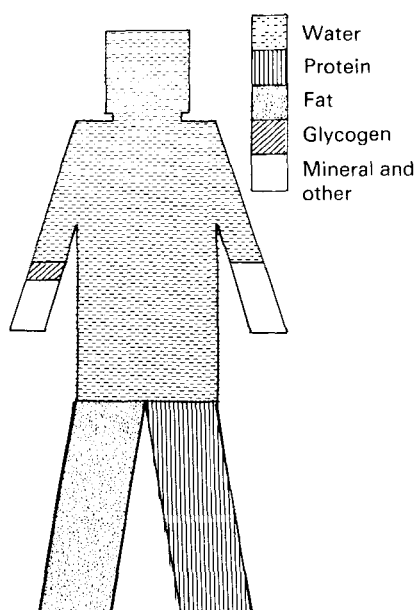


Fig. 1. Diagrammatic representation of the components of body-weight in a normal 70 kg adult male (Garrow, 1981).

whose loss is attended with some impairment of health or of physiological function'. The energy content of the body is the sum of the fat, protein and carbohydrate; it is convenient to consider first how these components can be measured in living subjects, and then to ask what proportion of the body content can be regarded as a store, or reserve, as defined above.

#### *Methods for measuring total body fat*

*Weight-for-height.* Usually, people who exceed the weight-for-height standards set by life insurance companies are overweight because they have an excess of fat. However, there are obvious exceptions. Some American football players were rejected for military service because they were overweight, but Behnke *et al.* (1942) were able to show that the excess weight was muscle, not fat. Conversely, Lesser *et al.* (1971) showed that 'normal' body-weight did not necessarily mean normal fat, especially in women over the age of 60 years; several subjects had 30–45% of body-weight as fat, despite having a body-weight which fell in the normal range. Thus weight-for-height is not always a reliable indicator of adiposity.

*Fat-soluble gases.* Since many anaesthetic gases, and rare gases such as xenon and krypton, are much more soluble in fat than in water it should be possible to measure body fat by observing the uptake of these gases. This has been tried (Hyttén *et al.* 1966; Lesser *et al.* 1971) but the method is unsatisfactory for clinical use because it takes many hours breathing in a closed-circuit apparatus to achieve an equilibrium concentration of the tracer gas. All the practical methods for measuring fat therefore rely on some assumed relationship between the variable measured and the fat content of the body.

*Skinfold thickness.* The simplest and least expensive technique is to measure the thickness of skinfolds at four sites; biceps, triceps, subscapular and suprailiac. Tables relating these measurements to total body fat have been published by Durnin & Womersley (1974). The main disadvantage of this technique is that it requires an experienced observer to obtain reproducible measurements of skinfold thickness in any subject, and in very obese subjects it may be impossible to do so.

*Total body water.* A less direct approach is to measure the total water content of the body, by observing the volume of dilution of a known dose of water labelled with tritium (Sheng & Huggins, 1979), deuterium (Halliday & Miller, 1977) or the heavy isotope of oxygen  $^{18}\text{O}$  (Schoeller *et al.* 1980). If it is assumed that the fat-free tissues of the body have a constant proportion of water, namely 73% (Pace & Rathbun, 1945), then the weight of fat-free tissue can be calculated, and subtraction from body-weight gives the weight of fat. Unfortunately water is not 73% of the weight of all fat-free tissues: if all the fat is extracted from a sample of skin, water makes up about 69.4% of the weight of the remainder, and if fat is extracted from adipose tissue about 88% of the residue is water. Other tissues give intermediate values. It is obvious that the water content of the fat-free tissues of the body cannot be constant as weight changes; with 10% gain or loss in weight it

is very unlikely that there will be 10% gain or loss of both skin and adipose tissue. Since tissues of different water content contribute differently to the weight change it is not possible that the water content of the mixture of all fat-free tissues should remain constant. With increasing adiposity the water content of fat-free tissues tends to increase, so the assumption of a constant water content leads to an underestimate of the fat content of obese subjects.

*Total body potassium.* A similar estimate of body fat can be made on the assumption that fat-free tissue has a constant K content. All K has a natural radioactive tracer,  $^{40}\text{K}$ , which makes it possible to measure total body K by detecting the high-energy radiation coming from this isotope. However, similar reservations apply to the assumption of a constant K content in fat-free tissue, as were discussed above for water. It appears that the K content of fat-free tissue tends to decrease with increasing obesity (Colt *et al.* 1981) so the assumption of constant K in fat-free tissue leads to an overestimate of body fat in obese subjects. Since this is the opposite error to that obtained with water estimates a combination of both water and K measurements yields a better estimate of total body fat than either measurement alone (Bruce *et al.* 1980).

*Conductivity.* The electrolyte solutions in lean tissue conduct electricity well, while fat does not. Thus, a measurement of whole-body conductivity yields an estimate of body fat. This has been successfully applied to farm animals (Domermuth *et al.* 1976) and is being developed for use with human subjects.

*Body density.* The specific gravity of human fat at body temperature is 0.90, while that of mixed fat-free tissues is about 1.10. Thus, a measurement of whole-body specific gravity will also give an estimate of body fat. This technique was pioneered by Behnke *et al.* (1942) and has been much used, with modifications, by other workers. The technical difficulty is to obtain a reliable measurement of the volume of the tissues of the subject. Usually this is done by observing the apparent weight loss when the subject is weighed first in air and then in water. However, it requires a trained and cooperative subject to submerge completely and remain motionless while the measurement is made. Furthermore, the water is displaced by air trapped in lungs and gut, as well as by the tissues of the subject. The residual air in lungs can be measured by a washout technique, but gas in gut is difficult to measure accurately. This problem has been largely overcome by using a plethysmograph in which the subject stands immersed in water up to neck level, and the volume of air around the head, and in the lungs and gut, is measured by a pneumatic method (Garrow *et al.* 1979).

*Validation of methods.* With experimental animals the accuracy of methods for estimating body composition can be checked by direct chemical analysis, but this is clearly impractical with human subjects, so alternative methods of validation must be found. If subjects are kept in a metabolic ward for several weeks, with careful monitoring of food intake and energy expenditure throughout this period, it is possible to make quite an accurate estimate of the change in body fat by calculating energy balance, and in body protein by nitrogen balance. If measures of body composition are applied at the beginning and end of the balance period the

change in estimated body composition should check with the results of the balance studies. This test was applied to nineteen obese patients by Garrow *et al.* (1979). At the beginning of the balance period the patients weight  $97.55 \pm 19.81$  kg, (mean  $\pm$  SD) and their fat content was estimated to be  $45.63 \pm 14.50$  kg by density,  $48.07 \pm 13.88$  kg by K and  $47.09 \pm 13.85$  kg by total body water determinations. During the balance period they lost  $5.43 \pm 1.83$  kg, and of this the estimates of fat loss were as follows, by energy balance ( $2.77 \pm 0.71$ ), N balance ( $2.69 \pm 1.23$ ), change in density ( $2.83 \pm 2.32$ ), change in water ( $2.37 \pm 2.38$ ) and change in total body K ( $2.90 \pm 3.54$ ). The agreement in mean fat loss by the different methods indicates that the constants assumed for calculating fat loss from the original data were at least plausible. The variation about this mean is in part due to true individual variation, but this true variation must be the same in each case, because the tests were all applied to the same nineteen subjects. The increasing SD on going from energy balance  $\rightarrow$  N balance  $\rightarrow$  density  $\rightarrow$  water  $\rightarrow$  K must therefore indicate increasing errors in the methods when used to estimate changes in body fat. It can be calculated that the SD due to measurement error on a single estimate of body fat by density is about 2.2 kg, by water about 2.3 kg and by K about 3.5 kg (Garrow *et al.* 1979).

#### *Methods for measuring total body protein*

*Neutron activation.* The methods discussed previously—density, water and K—for measuring body fat also give estimates of fat-free mass, and hence of body protein, which is about 20.5% of fat-free mass. However there is a more direct method for measuring total body N. If the subject is irradiated with neutrons, N (and other elements) become transiently radioactive (Hill *et al.* 1978; Cohn *et al.* 1980). This induced radiation can be detected by a system similar to that used for measuring  $^{40}\text{K}$ . Hill *et al.* (1978) found a coefficient of variation of 8.5% between estimates of fat-free mass derived from neutron activation, and from measurement of skinfolds.

#### *Measurement of total body glycogen*

Glycogen can be demonstrated histologically in liver and muscle, but there is no method for measuring the body content during life. The glycogen in liver can be estimated from the glucose production after administration of a large dose of glucagon (Radziuk, 1979). In normal subjects the glycogen content varies from one muscle to another (Hultman, 1967), so it is not possible to estimate total muscle glycogen from a single muscle biopsy. In post-mortem material glycogen may be absent from muscle, since it is destroyed by glycolytic enzymes after death. Olsson & Saltin (1970) manipulated the glycogen content of muscle by a programme of exercise and diet, and demonstrated that there was an increase in body water of about 3–4 g for each g glycogen stored.

This very labile glycogen:water pool causes confusion when attempts are made to relate weight change to energy imbalance. In the long run, with large changes in body-weight, each kg weight change is equivalent to about 30 MJ (7.2 Mcal) since

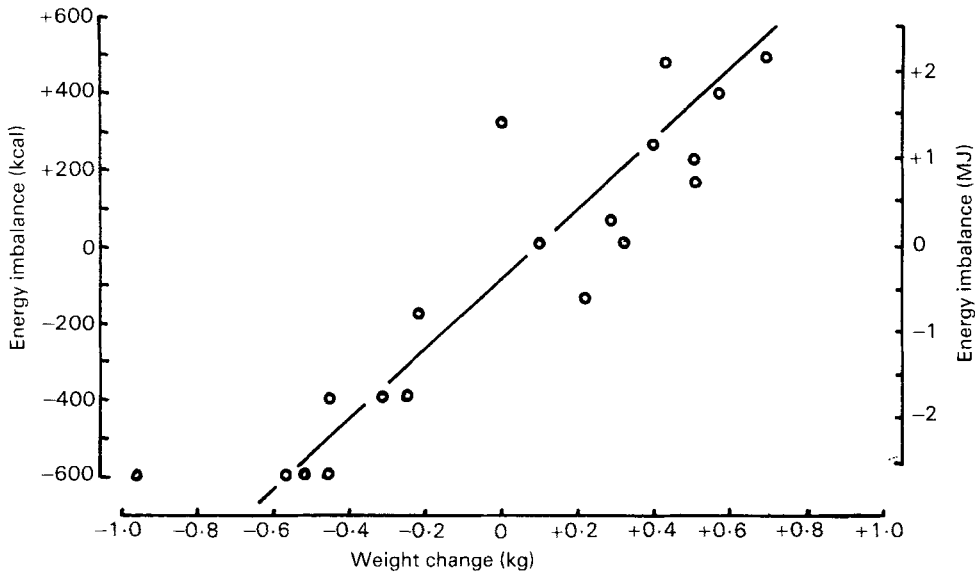


Fig. 2. Weight change measured serially in one subject, related to minor degrees of energy imbalance. The regression line suggests that the weight change is related to changes in a glycogen:water pool, with each g of glycogen binding 3 g of water (Garrow, 1974).

changes in adipose tissue are involved (Garrow, 1974). However, small energy imbalances can be met by changes in the glycogen:water pool, which has an energy value of only about 4.2 MJ (1 Mcal) per kg. This is indicated in Fig. 2 which relates weight change to small changes in energy balance in one subject. Even with very careful measurements of energy balance and weight change under standard conditions there is considerable scatter about the regression line, but there is a significant tendency for weight change and energy imbalance to be related in a manner which suggests that the weight change reflects changes in the glycogen:water pool.

#### *Energy stores or reserves?*

Having determined the body content of fat, protein or glycogen the problem remains to estimate what proportion of this total body content can be lost without loss of physiological efficiency (the 'stores' by the definition of Passmore, 1965) and how much more can be lost without causing death (the 'reserves'). In the case of glycogen the question is unanswerable, because this energy store is so small, labile and difficult to measure. As the marathon runner exhausts his glycogen reserves his running efficiency decreases, but we do not know how completely the body can be depleted of glycogen.

In the case of fat and protein we have better data. About one-third of the adult population of this country, and of the US, carry more than the optimum amount of fat, so they certainly have an energy store which could be lost without any impairment to health—indeed their health would probably improve. The evidence

for this statement is that lowest mortality in the experience of the life insurance companies, and in the survey conducted by the American Cancer Society (Lew and Garfinkel, 1979), is associated with a Quetelet's index ( $W/H^2$ ) between 20 and 25 (Garrow, 1981). The same range includes the physique of very successful athletes, whether men (Khosla, 1978) or women (Wilmore *et al.* 1977). Probably 20% of the adult population have at least 20 kg fat (Garrow, 1981), and could afford to lose 10 kg. This represents an energy store of 376 MJ (90 Mcal), or at least a month's energy requirements.

Some authors believe that there is no such thing as a reserve of protein; 'every molecule is performing a vital function, . . .' (Blackburn & Kaminski, 1980). This can hardly be true. The nineteen obese patients studied by Doré, Hesp, Wilkins and Garrow (unpublished results) initially weighed 104.5 kg, of which about 54 kg was fat-free tissue. When their body-weight was reduced to 73.7 kg their fat-free tissue had decreased to about 46 kg, so the loss of body protein was about 2 kg. There was no evident impairment of health after weight loss, so we must believe that these obese patients had a store of about 2 kg of expendable protein, in addition to the vast store of excess fat.

To assess reserves of fat and protein it is necessary to estimate the minimum amount compatible with life. Passmore (1965) suggests that 2.5 kg of fat and 8.6 kg protein is about the minimum required by an adult. Obviously these are estimates which it is difficult to verify experimentally. In the bodies of children who died of malnutrition there was gross wasting of protein. The protein remaining was about 40–50% collagen, instead of only about 27% in well-nourished children (Picou *et al.* 1966). Thus it is necessary to consider both the quantity and type of protein in these reserves. Similarly, the fat 'reserves' of a fatally malnourished child may not be metabolically accessible; three children with hardly any remaining subcutaneous fat had severe fatty infiltration of the liver, so that on postmortem analysis more than 30% of total body fat was recovered from the liver (Garrow *et al.* 1965). Thus it is not possible to make precise estimates of the fat and protein reserves in normal people. It is reasonable to expect that an adult, starting with the body composition shown in Fig. 1, would probably survive the gradual loss of 9.5 kg of fat, or of about 4.5 kg of protein.

#### REFERENCES

- Behnke, A. R., Feen, B. G. & Welham, W. C. (1942). *J. Am. Med. Ass.* **118**, 495.  
Blackburn, G. L. & Kaminski, M. V. (1980). In *Practical Nutritional Support*, pp. 166–189 [S. J. Karran and K. G. M. M. Alberti, editors]. Tunbridge Wells: Pitman Medical.  
Bruce, A., Andersson, M., Arvidsson, B. & Isaksson, B. (1980). *Scand. J. clin. Lab. Invest.* **40**, 461.  
Cohn, S. H., Vartsky, D., Yasumura, S., Sawitsky, A., Zanzi, I., Vaswani, A. & Ellis, K. J. (1980). *Am. J. Physiol.* **239**, E524.  
Colt, E. W., Wang, J., Stallone, F., van Itallie, T. B. & Pierson, R. N. Jr. (1981). *Am. J. clin. Nutr.* **34**, 367.  
Dormeruth, W., Veum, T. L., Alexander, M. A., Hedrick, H. B., Clarl, J. & Eklund, D. (1976). *J. Anim. Sci.* **43**, 966.  
Durnin, J. V. G. A. & Womersley, J. (1974). *Br. J. Nutr.* **32**, 77.

- Garrow, J. S. (1974). *Energy balance and obesity in man*, p. 362. Amsterdam: North Holland Publ. Co.
- Garrow, J. S. (1981). *Treat Obesity Seriously: a Clinical Manual*, p. 264. Edinburgh: Churchill Livingstone.
- Garrow, J. S., Fletcher, K. & Halliday, D. (1965). *J. clin. Invest.* **44**, 417.
- Garrow, J. S., Stalley, S., Diethelm, R., Pittet, Ph., Hesp, R. & Halliday, D. (1979). *Br. J. Nutr.* **42**, 173.
- Halliday, D. & Miller, A. G. (1977). *Biomedical Mass Spectrometry* **4**, 82.
- Hill, G. L., Bradley, J. A., Collins, J. P., McCarthy, I., Oxby, C. B. & Burkinshaw, L. (1978). *Br. J. Nutr.* **39**, 403.
- Hultman, E. (1967). *Scand. J. clin. Lab. Invest.* **19**, 209.
- Hytten, F. E., Taylor, K. & Taggart, N. (1966). *Clin. Sci.* **31**, 111.
- Khosla, T. (1978). *Br. J. Sports Med.* **12**, 97.
- Lesser, G. T., Deutsch, S. & Markofsky, J. (1971). *Metabolism* **20**, 792.
- Lew, E. A. & Garfinkel, L. (1979). *J. chron. Dis.* **32**, 563.
- Olsson, K. E. & Saltin, B. (1970). *Acta Physiol. Scand.* **80**, 11.
- Pace, N. & Rathbun, E. N. (1945). *J. biol. Chem.* **158**, 685.
- Passmore, R. (1965). In *Human Body Composition, Approaches and Applications*, p. 121 [J. Brozek, editor]. Oxford: Pergamon Press.
- Picou, D., Halliday, D. & Garrow, J. S. (1966). *Clin. Sci.* **30**, 345.
- Radziuk, J. (1979). *Can. J. Phys. Pharm.* **57**, 1196.
- Schoeller, D. A., van Santen, E., Peterson, D. W., Dietz, W., Jaspan, J. & Klein, P. D. (1980). *Am. J. Clin. Nutr.* **33**, 2686.
- Sheng, H-P. & Huggins, R. A. (1979). *Am. J. clin. Nutr.* **32**, 630.
- Wilmore, J. H., Brown, C. H. & Davis, J. A. (1977). *Ann. N.Y. Acad. Sci.* **301**, 764.