Learning Objectives

- To know the components of energy expenditure in human beings;
- To understand the flow of energy in the biosphere;
- To understand the concept of energy intake and expenditure in humans;
- To know the methods for measurement of energy expenditure;
- To be able to define how energy intake influences energy expenditure.

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Key Messages

- Energy demanding processes in humans are covered by energy from foodstuffs or the body’s energy reserves of carbohydrates, fat and protein;
- Total energy expenditure consists of resting energy expenditure (REE), diet induced energy expenditure (DEE), and energy spent on activity (AEE);
- Activity induced energy expenditure (AEE) - is the most variable part of energy expenditure;
- Indirect calorimetry is the most exact method to measure energy expenditure;
- REE is dependent mainly on fat-free body mass, but it is influenced by many factors such as disease or inflammatory activity, hormonal status or drug treatment;
- Positive energy balance is a necessary condition for growth and development as well as for healing processes and muscle gain during rehabilitation;
- Positive energy balance without physical activity leads to development of obesity in the adult.
1. Definition of Energy Expenditure

All living organisms expend energy on their living activities. Most of the energy on the Earth comes from the Sun. This energy, mainly as UV light, is trapped during photosynthesis in thylakoids of green plants and then transformed in Calvin–Benson–Bassham (CBB) cycle to glyceraldehyde-3-phosphate (G-3-P); G-3-P is used to synthesize hexoses (glucose) that are then transformed into other metabolic substrates like complex carbohydrates, fats and proteins.

Animals utilise energy from plants for their growth and other energy demanding processes. The energy is released from the main energy substrates (carbohydrates, lipid and proteins) through the process of oxidation (mainly in mitochondria), and finally water, carbon dioxide and nitrogenous compounds (such as urea) are released (Fig. 1).

![Flow of energy in the biosphere](image)

2. Components of Energy Expenditure

Total energy expenditure is the sum of all energy demanding processes in the human organism. It consists of several components which are different in different clinical situations.

Total energy expenditure (TEE) consists of:
- resting energy expenditure (REE)
- diet induced energy expenditure (DEE)
- activity induced energy expenditure (AEE)

Resting energy expenditure (REE) – is the energy which is required for indispensable homeostatic functions:
- breathing
• heart function
• basic GI function
• intermediary metabolism (e.g. continual proteosynthesis and breakdown)
• maintaining of ion gradients across cell membranes
• thermogenesis
• energy necessary for growth (in growing children) or for regain of body mass (e.g. rehabilitation after catabolic disease)

During resting conditions almost 60% of REE is spent by the heart, kidneys, brain and liver, although these organs account for only 5% of body weight (1) (see Module 18.1).

REE is dependent mainly on fat-free body mass. However, REE can be influenced by factors like:
• Hormonal status
  – thyroid hormones increase REE
  – catecholamines increase REE
  – combined secretion of glucagon, epinephrine and cortisol increases REE (2)
• Disease processes
  – disease or trauma increase REE (15-100%)
• Adaptation processes
  – prolonged starvation decreases REE
• Drugs
  – sympathomimetic drugs increase REE
  – opiates, barbiturates, sedatives, β-blockers (3) and muscular relaxants decrease REE
• Age of subject
  – REE decreases with increasing age of the subject, due mainly to loss of lean mass
• Growth and body mass gain
  – Growing children spend some part of their REE on growth
  – During regeneration a fraction of the REE is necessary for anabolic processes

Diet induced energy expenditure (DEE) – Energy expenditure increases after meal ingestion or during administration of artificial nutrition (parenteral or enteral) in comparison with energy expenditure during fasting conditions. DEE is conventionally assumed to be 10% of TEE; however, its value is dependent on the thermic effect of specific substrates and on the rapidity of substrate administration. Especially high rates of administration of artificial nutrition can lead to substantial increases in energy expenditure. This can have negative effects (e.g. cardiovascular function in patients with heart failure). The thermic effect of nutrition (TEN) is defined as the increase in energy expenditure above basal fasting level divided by the energy content of the food ingested. It is usually expressed as a percentage of energy intake:

\[
\text{TEN} = \left( \text{REE after a meal} - \text{basic REE} \right)/\text{EI} \times 100
\]

TEN - thermic effect of nutrition
REE – resting energy expenditure
EI – energy intake

The thermic effect of nutrition is dependent mainly on food composition and metabolic pathways of particular substrates (4). It usually lasts for 5-10 hours after feeding.
Thermic effect of major energy substrates:
- Carbohydrates – 4-6%
- Lipids – 2-3%
- Proteins – 20-40% (proteins have higher TEN due to energy demanding processes connected with amino acid metabolism)

Activity induced energy expenditure (AEE) - is the most variable part of energy expenditure. It is dependent on physical activity during the day and also on physical capacity of special subject.
Examples of energy expenditure through physical exercise, in kcal/minute, for subjects of 60 kg and 80 kg are shown in Table1:

<table>
<thead>
<tr>
<th>Body weight</th>
<th>60 kg</th>
<th>80 kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mountain climbing</td>
<td>9.4</td>
<td>12.7</td>
</tr>
<tr>
<td>Swimming (breast stroke)</td>
<td>9.6</td>
<td>13.0</td>
</tr>
<tr>
<td>Ice Hockey</td>
<td>9.1</td>
<td>12.5</td>
</tr>
<tr>
<td>Handball</td>
<td>8.5</td>
<td>11.5</td>
</tr>
<tr>
<td>Horseback riding (galloping)</td>
<td>8.1</td>
<td>11.0</td>
</tr>
<tr>
<td>Basketball (practice)</td>
<td>8.1</td>
<td>11.0</td>
</tr>
<tr>
<td>Soccer</td>
<td>8.1</td>
<td>10.9</td>
</tr>
<tr>
<td>Golf</td>
<td>5.0</td>
<td>6.8</td>
</tr>
<tr>
<td>Tennis (recreational)</td>
<td>6.4</td>
<td>8.7</td>
</tr>
<tr>
<td>Weight training</td>
<td>5.0</td>
<td>6.8</td>
</tr>
<tr>
<td>Running: 3 min/km</td>
<td>17.1</td>
<td>23.1</td>
</tr>
<tr>
<td></td>
<td>5 min/km</td>
<td>12.5</td>
</tr>
<tr>
<td></td>
<td>7 min/km</td>
<td>8.0</td>
</tr>
<tr>
<td>Cycling:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Racing</td>
<td>10.0</td>
<td>13.5</td>
</tr>
<tr>
<td>15 km/h</td>
<td>5.9</td>
<td>8.0</td>
</tr>
<tr>
<td>9 km/h</td>
<td>3.8</td>
<td>5.1</td>
</tr>
</tbody>
</table>


Energy, which is necessary for all metabolic processes, activity growth and other is produced by oxidation of energy substrates (carbohydrates, lipids and proteins). During the oxidation process oxygen is consumed, and carbon dioxide, water and nitrogenous compounds (mainly urea) together with heat are released.

![Fig. 2 Oxidative processes in organisms](image-url)
3.1 Direct Calorimetry

Direct calorimetry is method based on measurement of the heat production. Heat released from the body is equivalent to energy expenditure. It can be measured in special devices which are called whole body direct calorimeters:

\[ \text{EE} = \Delta Q = V (T_2 - T_1) \]

Energy released form the body - $\Delta Q$ is equivalent to the total energy expenditure of the whole organism.

The direct calorimeter is quite a complicated device, which cannot be used in daily practice. Therefore, for the principal part of scientific work as well as for clinical practice indirect calorimetry is the only applicable method.

3.2 Indirect Calorimetry

In indirect calorimetry, energy production is based on knowledge of oxidative pathways of particular substrates. Energy expenditure is calculated from oxygen consumption and carbon dioxide production. Analyzers are connected to a ventilated hood, mouthpiece or special whole body chambers.
Ventilated hood (canopy)

Using indirect calorimetry we can measure:
- Oxygen consumption - \( \text{VO}_2 \)
- Carbon dioxide production - \( \text{VCO}_2 \)

The energy equivalent of \( \text{VO}_2 \) and \( \text{VCO}_2 \) is dependent on the quantities of carbohydrate (C), protein (P) and fat (F) oxidized. Protein oxidation (g) is calculated from nitrogen lost in urine and subsequently the following formula can be used for calculation of energy expenditure (7):

\[
\text{EE (MJ)} = 16.20 \ \text{VO}_2 + 5.00 \ \text{VCO}_2 - 0.95 \ P
\]

Energy expenditure can also be calculated either from oxygen consumption or carbon dioxide production (8).

Carbon dioxide production can be measured over a long term period utilising the **doubly labelled water method** (9).

The doubly labelled water method is a method of indirect calorimetry. The principle of the method is that after a loading dose of water labelled with the stable isotopes of \(^2\text{H}\) and \(^{18}\text{O}\), \(^2\text{H}\) is eliminated as water, while \(^{18}\text{O}\) is eliminated as both water and carbon dioxide. The difference between the two elimination rates is therefore a measure of carbon dioxide production. This method is used for long-term measurement of energy expenditure (usually 14 days).

**The Reverse Fick** method is used to measure oxygen consumption in ICU. This method is based on measurement of cardiac output (thermodilution) and the difference in oxygen concentration between arterial and mixed venous blood.

**Indirect calorimetry and oxidation of energy substrates: examples**

**Glucose oxidation**
1 mol glucose + 6 mol \( \text{O}_2 \) \( \rightarrow \) 6 mol \( \text{H}_2\text{O} \) + 6 mol \( \text{CO}_2 \)

**Fat oxidation**
1 mol palmitate + 23 mol \( \text{O}_2 \) \( \rightarrow \) 16 mol \( \text{H}_2\text{O} \) + 16 mol \( \text{CO}_2 \)
As we know that the product of protein oxidation is urinary nitrogen, we can calculate the oxidation of all macronutrients (carbohydrate, fat and protein) from oxygen consumption \( (V_{O_2}) \), carbon dioxide production \( (V_{CO_2}) \) and urinary nitrogen excretion \( (UN) \).

### 3.3 Estimation of Energy Expenditure

The most common approach to predict REE for an individual in clinical practice is to apply the Harris-Benedict equations.

- **Male:** \( REE = 66.5 + (13.8 \times \text{weight}) + (5.0 \times \text{height}) - (6.8 \times \text{age}) \)
- **Female:** \( REE = 655.1 + (9.6 \times \text{weight}) + (1.8 \times \text{height}) - (4.7 \times \text{age}) \)

These equations are based on sex, age, height and body mass, but do not take body composition into account. These formulae were developed in the 1920s; however, they are still commonly for estimation of energy expenditure. Since 1920 a lot of other formulae for REE calculation have been designed, however none of them is universally valid. Therefore only indirect calorimetry can be suggested as a “gold standard” for energy expenditure measurement (10, 11).

### 4. The Influence of Disease on Energy Expenditure

Acute and chronic illnesses frequently increase energy expenditure (12). This is due to the inflammatory reaction, increase in body temperature, shivering, or increased substrate cycling (futile cycles). The increase in body temperature leads to a rise in energy expenditure by 10-15% per degree C. The increase in energy expenditure during disease processes is also a result of increased sympathetic activity. Disease related increase in energy expenditure can be partially abolished by sympathetic blockade. The increase in energy expenditure after severe burns can be reduced by higher ambient temperature (the thermo-neutral zone for burns patients is over 30°C, compared to 28°C for normal subjects). In addition, energy intake influences energy expenditure during disease, showing that diet-induced thermogenesis also operates during illness (13). Thermo-neutral environments also decrease energy expenditure in ICU patients. The influence of critical illness on energy expenditure is described further in Module 18.1.

*Note: our thermoneutral zone is the ambient temperature at which we need to expend no additional energy in order to maintain our body temperature i.e. the ambient temperature at which our basal metabolic expenditure is minimal.*

### 5. Energy Intake and Energy Balance

In a stable non-growing organism the energy intake should balance energy expenditure. However whereas energy expenditure is a relatively continual process (with the constant REE part, and DEE and AEE in bouts according to food intake and body activity) energy intake is an intermittent process. Therefore, over short periods the energy balance of free-living subject may vary from positive to negative. However, in stable subjects the energy intake will be equivalent to energy expenditure over the long-term.

The main energy substrates:

- **Carbohydrates** – 4 kcal/g (glucose, maltodextrin, starch, glycogen);
- **Lipids** – 9 kcal/g (fat, lipid emulsion);
• **Proteins** – 4 kcal/g (meat, casein, whey protein, plant proteins).

Positive energy balance is associated with:

• Synthesis of glycogen (liver and muscle glycogen);
• Fat storage in adipose tissue – fat is also partially stored in non-adipose tissue like muscles and the liver;
• Storage of proteins – We have no genuine protein depots which are comparable to adipose tissue for storage of lipids. Therefore protein gain is always connected with growth, healing, or muscle gain. Hence, physical activity is a necessary condition for protein synthesis in skeletal muscles in adult patients.

Negative energy balance is associated with:

• Breakdown and oxidation of glycogen stores – body glycogen stores are exhausted within 24 hours;
• Lipolysis, release and oxidation of fatty acids;
• Protein breakdown and oxidation – a degree of protein breakdown is inevitable with a concomitant inflammatory process.

Growth, healing and recovery

In the growing organism (neonates, infants and children) a significant part of consumed energy substrates is devoted to growth and not oxidized. Then energy balance is positive. The most positive energy balance is apparent in neonates (14). Their daily energy expenditure is 50-60 kcal·kg\(^{-1}\)·day\(^{-1}\), whereas recommended energy intake is 110-120 kcal·kg\(^{-1}\)·day\(^{-1}\). This difference is due to the energy cost of growth (accretion) - 30-40 kcal·kg\(^{-1}\)·day\(^{-1}\). Positive energy balance is also necessary for “catch up” growth after a period of malnutrition in children or during rehabilitation of adults or children from severe illness. Similarly, during healing processes, rehabilitation and related muscle gain and other anabolic processes, the energy balance should be positive. Besides positive energy balance muscle protein synthesis also requires physical activity.

In conclusion positive nitrogen balance is necessary for:

• growth;
• wound healing;
• rehabilitation after severe disease;
• exercise-related muscle gain.

However, long term positive energy balance due to excess intake and/or too little exercise leads to gain of fat tissue, overweight and obesity.

**6. Summary**

All living organisms expend energy for their living activities. Animals use energy substrates initially generated in plants in the form of carbohydrates, lipids and proteins. These substrates are oxidised to water, carbon dioxide and nitrogen, the quantity of oxygen consumed and carbon dioxide produced being equivalent to energy expenditure. Indirect calorimetry, which is based on measurement of oxygen consumption or/and carbon dioxide production is the most exact practicable method for energy expenditure measurement.
Total energy expenditure consists of resting energy expenditure (REE), diet-induced energy expenditure (DEE) and activity-induced energy expenditure (AEE). Loss of energy stores is the result of negative energy balance, the character of tissue substrate loss depending on the conditions during which negative energy balance occurs. Pure starvation, at least in its early stages, leads mainly to loss of fat, whereas injury induces a proportionally greater loss of lean mass. Positive energy balance is necessary for growth, wound healing and muscle gain, but if substantial and prolonged, can lead to overweight and obesity.

7. References