

Chapter 16

Total Energy Expenditure of Exercise and Recovery

The differences between closed and open systems are very real and must be accounted for; survival comes at a continual cost. Life's expenditures have been flipantly described as:

You can not win,
You can not break even,
You can not get out of the game. (Anonymous)

“Playing life's games” requires the use of body's musculature. Movement in all forms – exercise, physical activity, work, training, competing, fleeing – acutely increases energy expenditure (perhaps chronically too in the well trained (1)). Feats of speed, strength, and power also require energy to be expended during a recovery period. Exercise and the recovery energy expenditure that results are combined in an estimate of total energy expenditure requiring three independent measures (Fig. 16.1):

Aerobic exercise energy expenditure
Anaerobic exercise energy expenditure
Aerobic recovery energy expenditure

16.1 Aerobic Exercise Energy Expenditure

Energy is expended during the exchange of chemical energy to mechanical force; work, heat, entropy, carbon dioxide, and water are put-out to the environment as oxygen is consumed. Using twenty-first century technology, the measurement of oxygen uptake for the estimation of aerobic energy expenditure is a rather simple task. In addition to individual differences in oxygen uptake economy during exercise, researchers further understand that a given amount of biological and measurement variability is always evident (the shortest sampling periods lasting seconds (e.g., breath-by-breath) have the largest variance as compared with measurements that average oxygen uptake over a 1-min period (or longer) (2)).

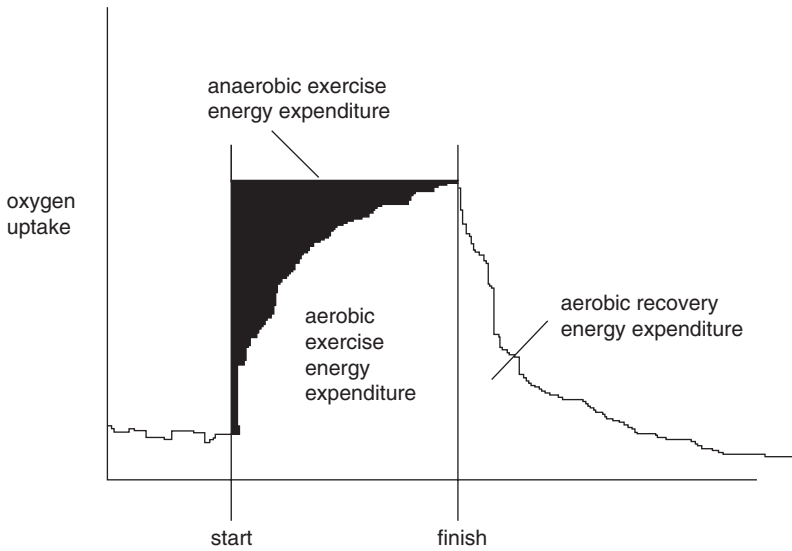


Fig. 16.1 The three components of total energy expenditure during and after a brief bout of heavy exercise

Lower intensity, steady-state, longer duration exercise is supported in full by aerobic metabolism so that a measurement of oxygen uptake adequately estimates the energy expenditure associated with oxygen-related ATP turnover (Fig. 16.2). Depending on the type or mixture of substrates consumed by working muscle during steady-state exercise, energy expenditure per liter of oxygen consumed ranges from 19.6 kJ (all fat) to 21.1 kJ (all carbohydrate) (see Table 12.1).

Muscle contraction may be fueled largely by anaerobic metabolism during heavy to severe exercise. To obtain a more complete estimate of the energy expenditure for intense exercise, anaerobic energy exchange warrants consideration.

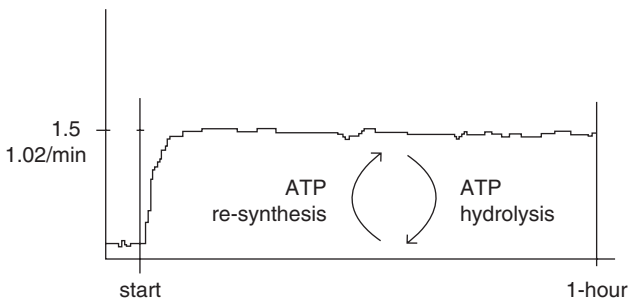


Fig. 16.2 Moderate intensity steady state exercise is depicted above lasting 1 h in length. A measurement of oxygen uptake (at 1.5 L of O₂ min⁻¹) provides an estimate of the energy expended during ATP hydrolysis (energy demand) and ATP resynthesis (energy supply) for this period of exercise

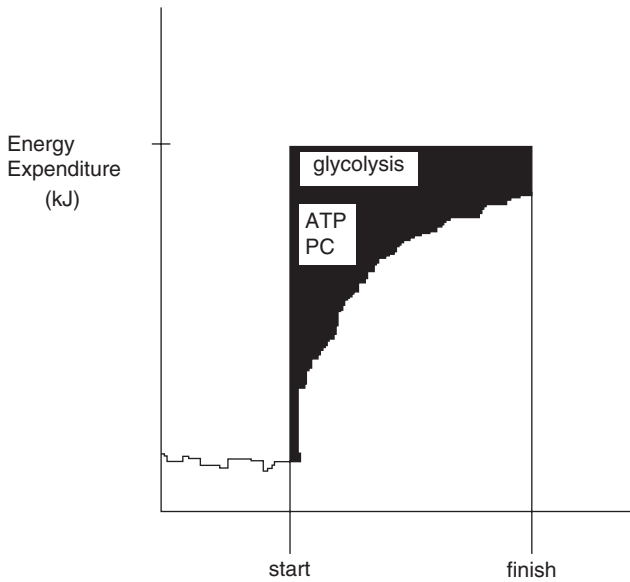


Fig. 16.3 The anaerobic energy expenditure components are shown for a brief bout of heavy to severe exercise

16.2 Anaerobic Exercise Energy Expenditure

The two predominate sources of anaerobic energy expenditure are (Fig. 16.3):

1. rapid anaerobic glycolysis (substrate-level phosphorylation)
2. stored “high energy” phosphates (ATP, PC)

Both sources of anaerobic energy exchange need to be accounted for when attempting to estimate anaerobic energy expenditure (3). Muscle biopsies appear to provide the most insightful reflection of anaerobic energy exchange. The biopsy procedure is, however, invasive and a minuscule sample of muscle only hints at the metabolic events, aerobic and anaerobic, within entire muscle beds made-up perhaps of thousands of muscle fibers composed of several fiber types (ranging from fast-twitch glycolytic fibers to slow-twitch oxidative fibers). As described earlier, the two non-invasive methods of estimating anaerobic energy expenditure require a measurement of blood lactate or the oxygen deficit; both provide an indirect accounting (or estimate) of anaerobic energy exchange.

In real time, the oxygen-deficit period contains anaerobic glycolysis along with the use of the ATP, PC stores. A measurement of lactate concentration provides only an estimate of the anaerobic glycolytic contribution to exercise energy expenditure. Contributions by the two anaerobic components can vary dramatically depending on the duration, intensity, and type of exercise. For example, short bouts of strength-type, heavy-resistance, weight training involving

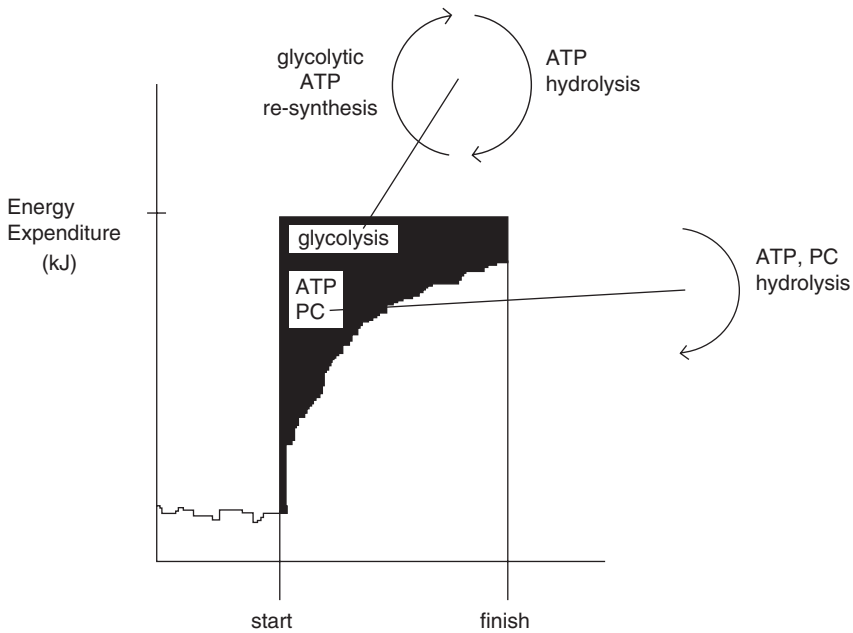


Fig. 16.4 Two components of anaerobic exercise energy expenditure as part of the oxygen deficit (in black) are shown in *real time* for a brief bout of heavy to severe exercise. Note that glycolysis is associated with complete ATP turnover. ATP and PC hydrolysis represents only a half-cycle of ATP turnover

limited repetitions may be fueled largely by the ATP, PC stores. To the contrary, longer lasting, moderate resistance, muscular endurance-type resistance training may contain a larger anaerobic glycolytic component with limited ATP, PC contributions (4).

Recall that a measure of oxygen uptake to estimate energy expenditure (kJ) represents a complete cycle of ATP turnover. Lactate measurements in turn are converted into oxygen equivalent units (then into kJ) so that they too represent a complete cycle of ATP turnover (5, 6). A measure of the oxygen deficit likewise is portrayed by oxygen equivalent units, also representing complete ATP turnover. In *real time*, however, the oxygen deficit contains ATP and PC usage to support muscle contraction but not the metabolic resynthesis needed for restoration (Fig. 16.4) (7, 8).

In real time, resynthesis of the ATP, PC stores actually takes place after exercise, during recovery (9–11). Thus, when expressed in oxygen equivalent units, a measure of the oxygen deficit cannot be included along with a recovery oxygen uptake measurement because when doing so the ATP, PC turnover component is accounted for twice. A measurement of the oxygen deficit in the portrayal of anaerobic energy expenditure is an acceptable practice only when estimating aerobic and anaerobic exercise energy expenditure (Fig. 16.5), not exercise and recovery energy expenditure.

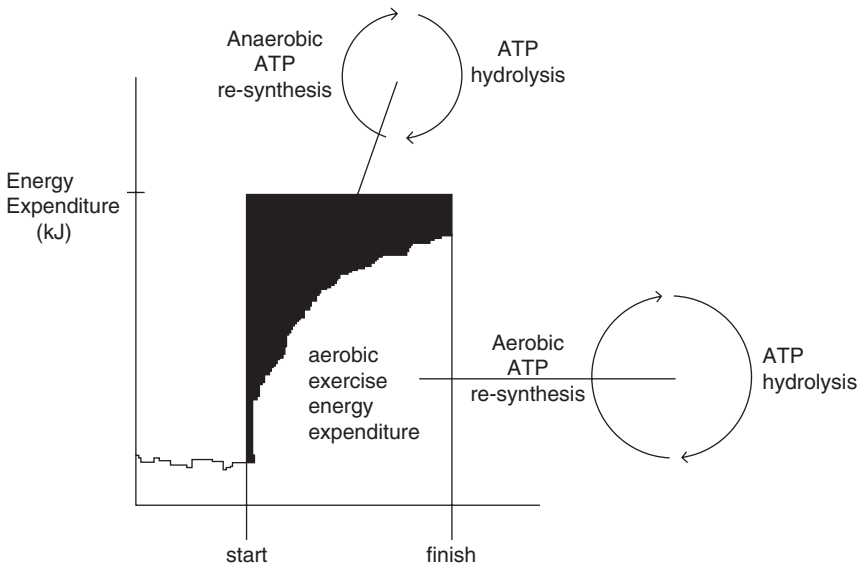


Fig. 16.5 The oxygen deficit (in black) does not partition anaerobic exercise energy expenditure into anaerobic glycolytic and ATP, PC components. When measured respectively as oxygen uptake and oxygen equivalent units, aerobic (in white) and anaerobic (in black) energy expenditures are expressed as a complete cycle of ATP turnover. Recovery energy expenditure is absent from the above diagram

16.3 Aerobic Recovery Energy Expenditure

Whole-body energy demands elevated throughout exercise gradually diminish to resting levels during recovery and the pattern of oxygen uptake follows suit (see Figs. 16.6 and 16.7). Depending on exercise intensity and duration, recovery oxygen uptake can remain elevated for minutes to hours (perhaps even days after extreme sporting events) (10). Over the years the oxygen consumed in recovery has been described using terminology that both offers and dismisses explanations of how anaerobic exercise and aerobic recovery energy expenditure are to be interpreted. Respectively, the oxygen debt and excess postexercise oxygen consumption (EPOC) are two prominent examples of such terminology. In a straight-forward manner the energy expenditure of recovery here will be termed *aerobic recovery energy expenditure*. Skeletal muscle is not contracting during an inactive recovery but energy exchange is essential to reacquire a resting homeostasis, in muscle cells and throughout the body. Some of the demands for aerobic energy exchange during recovery are provided in Table 16.1 (from (10)).

Research has shown that the recovery from exercise appears to be solely aerobic; there is little to no rapid anaerobic glycolytic component to recovery energy expenditure (9, 11). Oxygen uptake measurements are therefore extremely useful in the estimation of recovery energy expenditure, again representing a complete cycle

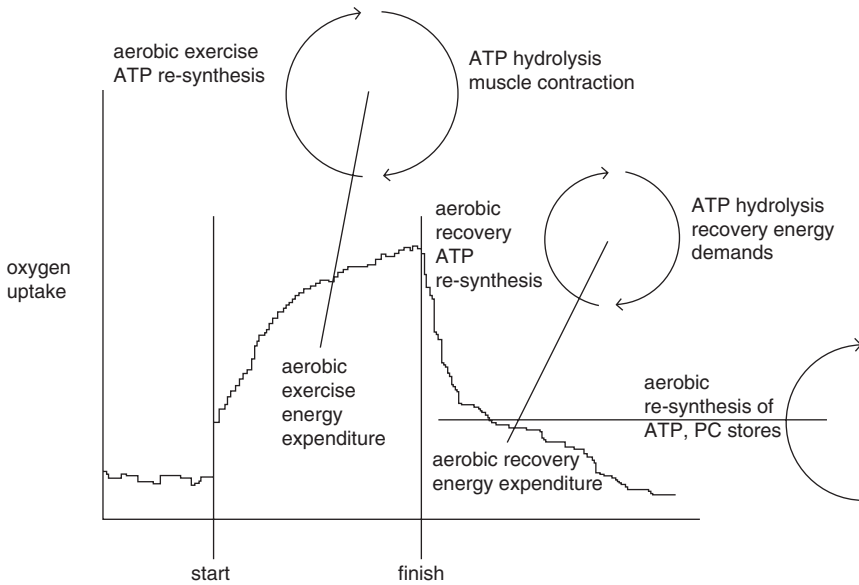


Fig. 16.6 Aerobic energy expenditure for exercise and recovery is depicted in *real time*; anaerobic exercise energy expenditure is not shown. Complete ATP turnover is accounted for by exercise related oxygen uptake. Recovery related oxygen uptake also is associated with complete ATP turnover in the reattainment of cellular and whole-body homeostasis. However, resynthesis of the ATP, PC stores represents an aerobic ATP half-cycle and, in *real time*, is not associated with ATP, PC hydrolysis during exercise

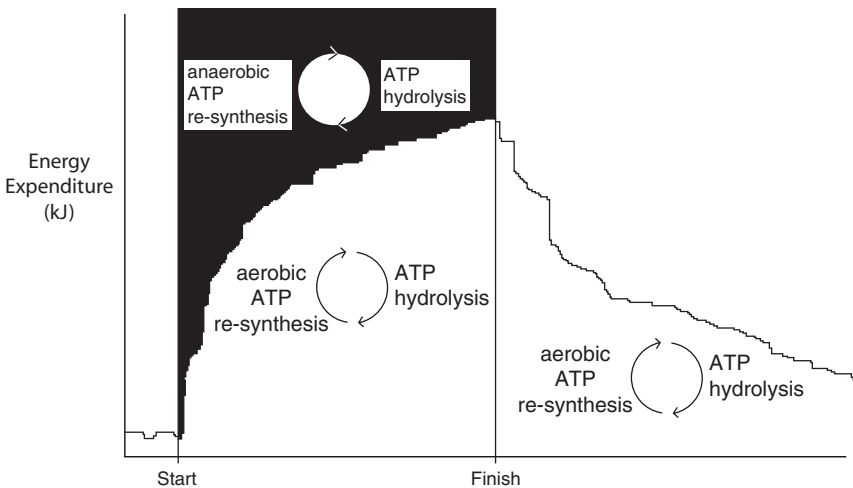


Fig. 16.7 The above diagram portrays an all-out 2-min sprint up a steep hill: start to finish along with recovery. Anaerobic glycolytic exercise energy expenditure is shown in black as measured by Δ blood lactate. Aerobic exercise and recovery energy expenditure also are displayed. This figure demonstrates each of three complete estimations of complete ATP turnover that comprise total energy expenditure

Table 16.1 ATP demands during the recovery from exercise

Resaturation of hemoglobin and myoglobin (this is an O ₂ demand not an ATP demand)
Cost of ATP, PC resynthesis
Cost of increased circulation (e.g., heart) and ventilation (e.g., diaphragm)
Cost of glycogen resynthesis from lactate
Triglyceride-free fatty acid cycling
Protein turnover
Hormonal effects on energy expenditure (e.g., epinephrine, growth hormone/factors)

of ATP hydrolysis to fuel recovery energy demands and the mitochondrial resynthesis of that ATP. But again care must be taken when considering restoration of the ATP, PC stores. In real time, resynthesis of the ATP, PC stores in recovery (but used during exercise) represents only a half-cycle of ATP turnover (Fig. 16.6). Yet recovery energy expenditure accounted for by oxygen uptake represents complete ATP turnover (Fig. 16.7).

The conundrum created by an indirect estimate of ATP, PC usage and resynthesis has straight-jacketed exercise physiologists for years. The same is true for anaerobic glycolysis as described in the next section (the oxygen debt hypothesis). As a result many exercise scientists have resorted to measuring only one or two of the three components of total energy expenditure in the avoidance of redundancy issues:

1. *Aerobic exercise energy expenditure* (lacking anaerobic exercise and aerobic recovery measurements). This is extremely useful for estimating the energy expenditure of low-to-moderate intensity steady-state exercise (Fig. 16.2) (Table 12.1). It is unacceptable when estimating energy expenditure for anaerobic exercise.
2. *Aerobic and anaerobic exercise energy expenditure* (lacking an aerobic recovery measurement). This is ideal for those who desire only to consider the cost of a bout of exercise (Fig. 16.5).
3. *Aerobic exercise and aerobic recovery energy expenditure* (lacking an anaerobic exercise measurement). Acceptable only for low to moderate intensity exercise and its recovery; these components have perhaps been the most problematic in allowing (or rather not allowing) for a reasonable estimate of anaerobic energy expenditure (Fig. 16.6). These two measurements do not properly account for anaerobic exercise that contains a significant glycolytic component (i.e., a large lactate concentration after exercise is complete).

16.4 Dismissing the Oxygen Debt Hypothesis

It has been hypothesized since the 1920s that aerobic recovery oxygen uptake represented anaerobic exercise energy expenditure in addition to aerobic recovery energy expenditure. This idea was known as the oxygen debt hypothesis and its lasting

influence has perhaps been influenced by the fact that it characterizes a half-truth: the “debt” of ATP, PC resynthesis created during exercise *is* repaid in recovery.

Supporters of the oxygen debt hypothesis went too far, however, in suggesting that recovery oxygen uptake not only represented use and resynthesis of the ATP, PC stores, but also the ATP utilized and resynthesized as rapid anaerobic glycolysis with lactate production. Much of the hypothesis was based on knowing how much lactate was formed and how much was later oxidized in recovery. A valid determination of either complete lactate production or complete removal would seemingly allow for a reasonable estimate of anaerobic glycolytic energy expenditure. Disproof of the oxygen debt hypothesis was demonstrated by the ubiquity of lactate removal (12). Lactate can be converted into glucose, glycogen, amino acids, and protein. It also is found in sweat, urine, and saliva. With so many avenues of disappearance, it is not possible to obtain a valid account of lactate removal. Yet as interesting as the ubiquity of lactate removal is, this information does little to quantify anaerobic glycolytic and recovery energy expenditure. Indeed, information concerning lactate removal has instead been used to inform scientists how not to obtain an estimate of anaerobic energy expenditure! So how can a concurrent estimate of anaerobic exercise and aerobic recovery energy expenditure be obtained?

Recall that the breakdown of glucose-to-pyruvate evolves less heat than does the breakdown of glucose-to-lactate; the reduction of pyruvate to form lactate is the largest source of heat production during glycolysis, from the metabolic system to the surroundings. Based on the *reversible* thermodynamics of a closed system as the oxygen debt hypothesis apparently was, a reversal in heat transfer – from the surroundings to the system – during the removal or reconversion of lactate-to-pyruvate would promote the concept that oxygen uptake can account for an accelerated pyruvate-to-lactate energy exchange. However, within the context of an open *irreversible* system, it is difficult to conceive of how the heat expended from glycolytically (anaerobic) supported muscle contraction could be represented by a measurement of mitochondrial oxygen uptake during recovery as the oxygen debt hypothesis proposed. Anaerobic metabolic glucose degradation as a part of aerobic energy exchange (at 1.5 kJ LO_2^{-1}) can only be represented by oxygen uptake when the rate of glycolysis matches that of mitochondrial respiration (i.e., when anaerobic pyruvate production matches aerobic pyruvate removal, where $1 \text{ LO}_2 \text{ min}^{-1} = 21.1 \text{ kJ min}^{-1}$). When rapid anaerobic glycolytic energy exchange proceeds at a rate that exceeds that of mitochondrial respiration, anaerobic energy expenditure is not accounted for by a measure of recovery oxygen uptake (13).

As an open system, muscle contraction fueled by rapid anaerobic glycolysis irreversibly increases heat and entropy in the surroundings; they are not later removed from the surroundings by mitochondria during the oxidation of lactate (14). [Table 16.2](#) reveals that in respiring cells and tissues that were separately “fed” pyruvate and lactate, similar amounts of heat were produced per volume of oxygen consumed. This experiment demonstrates that when lactate is reconverted back into pyruvate for complete oxidation by mitochondria, heat is not consumed (reversed) as part of the reaction. Lactate production and lactate oxidation involve independent

Table 16.2 Respiratory heat production (ΔH) for pyruvate and lactate oxidation

	ΔH (kJ mol O ₂ ⁻¹)		
	Pyruvate	Lactate	<i>p</i>
Hybrid cells	-517	-516	0.97
Cardiac muscle fibers	-506	-502	0.92

energy-exchange devices, each representing separate and additive components to the measurement of total energy expenditure for exercise and recovery (14).

Recovery energy expenditure can be described in the context of an oxygen debt for ATP, PC restoration. Aerobic recovery energy expenditure also has traditionally and incorrectly been used to estimate rapid anaerobic glycolytic ATP resynthesis as part of oxygen uptake during the recovery from exercise as, 1 L of O₂ uptake = 21.1 kJ. This conversion contains an anaerobic glycolytic energy expenditure component (1.5 kJ) that should not be accounted for twice, once as a Δ blood lactate or oxygen deficit estimate of anaerobic energy expenditure (in O₂ equivalents) and again as part of recovery oxygen uptake. Instead, the energy expenditure of recovery should be free of the anaerobic glycolytic component, being based entirely on aerobic (mitochondrial) respiration. How is this accomplished? The all-aerobic conversion demonstrated earlier as Thornton's law, where 1 L of O₂ uptake = 19.6 kJ, void of the anaerobic glycolytic component, eliminates the false rationale of rapid anaerobic glycolysis during exercise being portrayed by a measurement of recovery oxygen uptake (14–19).

16.5 Total Energy Expenditure

Three measurements are available to provide a reasonable estimation of total energy expenditure for both exercise and recovery (with limitations):

1. anaerobic (glycolytic) exercise energy expenditure,
2. aerobic exercise energy expenditure (oxidizing fats and/or carbohydrates),
3. aerobic (nonglycolytic) recovery energy expenditure.

Within the strict limitations of brief and intense exercise, when peaking in recovery (not during the exercise), lactate concentrations can provide a reasonable estimate of *anaerobic (glycolytic) exercise energy expenditure* (3–6, 19, 20).

$$\Delta \text{blood lactate} \times 3 \text{ mL O}_2 \times \text{weight (kg)} = \text{mL O}_2 \text{ equivalents}$$

then 1 L O₂ = 21.1 kJ (assuming equivalent aerobic and anaerobic efficiency)

The estimation of *aerobic exercise energy expenditure* is straight-forward, using a measurement of oxygen uptake during exercise (dependent on substrate utilization, see Table 12.1):

$$1 \text{ L of exercise O}_2 \text{ uptake} = 19.6 - 21.1 \text{ kJ}$$

Aerobic (nonglycolytic) recovery energy expenditure representing: (1) fat and lactate oxidation (10), (2) use and resynthesis of the ATP, PC stores, and (3) the energy demands of recovery:

$$1 \text{ L of recovery O}_2 \text{ uptake} = 19.6 \text{ kJ}$$

ATP turnover for each of the three components of total energy expenditure is detailed in [Fig. 16.7](#).

16.6 Weight Loss: Low vs. High Intensity Activity

It is acknowledged that, (1) “physical activity affects body composition and weight favorably, by promoting fat loss, while preserving lean mass” and, (2) “the rate of weight loss is positively related to the frequency and duration of the exercise program, thereby suggesting a dose-response relationship” (21). Exercise prescriptions that focus on dose-response relationships also include exercise intensity as a key variable. How does exercise intensity affect the rate and amount of body weight and/or fat mass loss? The answer is complex and seemingly dependent on how one examines this issue.

Part of the complexity resides in the variable being studied. For example, is it body weight or body fat losses that are of utmost concern? Other issues are at play here in that energy intake may not be taken into account and this confounds the problem when comparisons are made – people may eat more after exercising at higher exercise intensities and not lose weight. Higher intensity activity may conserve or build lean body tissue (fat-free mass) (22, 23). Lower intensity activity has the potential advantage of reducing injury in overweight exercisers, a legitimate concern.

Reciprocal arguments have been created to support both low and high intensity programs in the reduction of body fat. Exercise programs have focused on substrate utilization during activity, glucose (and glycogen) vs. fat “burning” for example. As an example, exercise programs designed for fat loss traditionally focus on fat “burning” at lower exercise intensities coupled with longer durations. From this perspective long slow distance training has been advocated as the best way to reduce the body’s fat stores. While it is true that higher exercise intensities are associated with muscle glycogen utilization, it is apparent that a significant amount of fat is also broken down and subsequently dumped into the blood stream during intense exercise, even though that fat may not be consumed during exercise. This fat (triacylglycerides) appears to be ready as a fuel for recovery (24). Scientists have shown that a period of initial exercise followed by passive recovery can subsequently affect substrate oxidation during additional exercise bouts with greater fat and less carbohydrate utilization (25, 26). In this context exercise programs designed for fat oxidation should be intermittent in nature.

Several studies have in fact revealed greater body fat losses with higher intensity exercise (27–32). If high-intensity exercise “burns calories” at a greater rate

than lower intensity exercise, should the exercise program focus more on how much energy is expended as compared to what type of fuel is utilized? Statistical comparisons of groups exercising at low and high intensities are limited because the amount of energy expended is often different between groups exercising at differing intensities; for example, 30-minutes of heavy exertion “burns more calories” than 30-minutes of low exertion activity. A better study design would involve identical energy expenditure between exercising groups to provide a clear example of the effects of intensity alone on fat loss. When overall energy expenditure is taken into consideration, being identical for low- and high-intensity exercise, higher exercise intensities are generally not associated with greater body weight or fat loss (22, 33, 34). As pointed out in the beginning of this section, it may be that body fat losses associated with higher intensity exercise are simply a result of energy expenditure in a dose–response manner, the greater the total energy expenditure for both exercise and recovery the greater the chance for body fat loss (23). In practice then, the best exercise programs for body weight or fat loss would involve intermittent heavy to severe work bouts utilizing large muscle groups followed by an active recovery. Sprinting a given distance or over a given time “burns more calories” than walking; walking or jogging during the recovery from that sprint continues to elevate “calorie burning” above resting levels, and consumes fat to do so. It all adds up (Fig. 16.8).

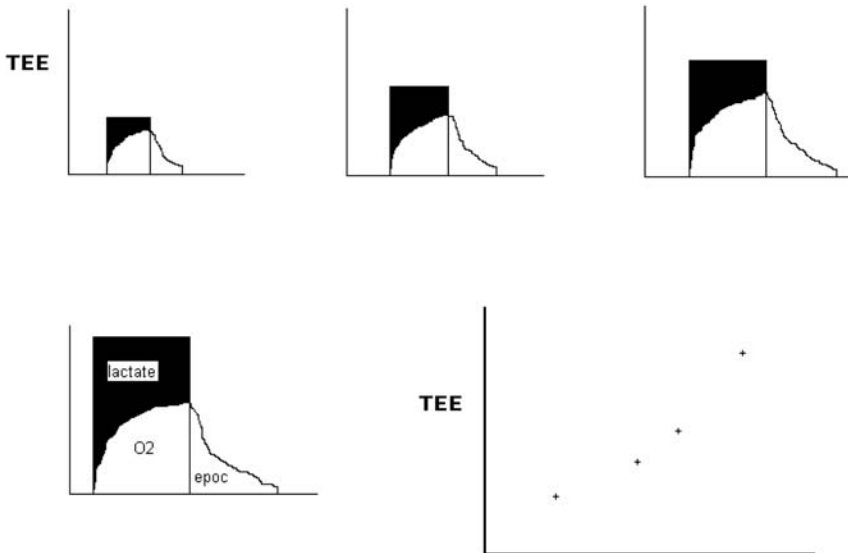


Fig. 16.8 Total energy expenditure (TEE) is shown for 4 intense workloads. Each of these is plotted in the bottom right to reveal a true curvilinear relationship as opposed to predicted and false linear relationship

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