Tissue temperatures and whole-animal oxygen consumption after exercise

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Brooks, George A., Karl J. Hittelman, John A. Faulkner, and Robert E. Beyer. Tissue temperatures and whole-animal oxygen consumption after exercise. Am. J. Physiol. 221(2): 427–431. 1971.—Forced treadmill running caused rat muscle and rectal temperatures to increase 8.1 and 5.1°C, respectively. After exercise, muscle temperature fell exponentially but did not reach control values in an hour. Rectal temperature fell rapidly for the first 20 min after exercise, after which only a slow rate of return to resting levels was apparent. An exercise-induced adjustment in the hypothalamic set point was suggested. O₂ consumption was high immediately after exercise, declined rapidly for the first 20 min of recovery, and then plateaued at a level significantly above resting. The hypothesis that a sizable portion of postexercise 0₂ consumption is due to increased tissue temperatures is substantiated. The fact that severe exercise results in a large, prolonged elevation in tissue temperature necessitates, as a consequence of the Q₁₀ effect, that 0₂ consumption be significantly elevated. Since a part of the postexercise 0₂ consumption is not associated with recovery from anaerobic metabolism, the classical definition of 0₂ debt requires revision.

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The measured quantity of oxygen consumed above resting levels after severe exercise (the O₂ debt) is invariably at least 3 times larger than the theoretical debt estimated from the sum of all potential O₂-requiring sources (5, 11, 22, 23). Previous considerations of the biochemical basis underlying O₂ debt (12, 14, 15) have assumed that the coupling between the mitochondrial electron transfer chain and its associated ATP-synthesizing system was complete (i.e., that for each atom of oxygen consumed, about 3 molecules of ATP are synthesized), and that cellular respiration did not proceed at an appreciable rate in the absence of ADP. However, adequate attention has not been paid to effects of elevated tissue temperatures on respiration. Recent studies (1, 2) have indicated that O₂ debts were inexplicably high when tissue temperatures were increased as the result of exercise. This observation prompted us to determine operating tissue temperatures on respiration.

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Abbreviations: ADP, ATP: adenosine di- and triphosphate, respectively; ADP:O ratio: the ratio of the number of molecules of ADP phosphorylated to the number of atoms of oxygen consumed; ATPase: ATP phosphohydrolase, E.C. 3.6.1.3; BMR: basal metabolic rate; CP: creatine phosphate.
ducted in a thermostatically controlled, heated and air-conditioned room in which the ambient temperatures ranged from 24 to 26 °C. No special care was taken to control movement of air across exercising animals because of the air-jet stimulator.

For temperature measurements after exercise, animals were rapidly placed in a Plexiglas restraining apparatus and two copper-constantan thermocouples were inserted, one by needle into the thigh muscles of the right hindleg and the other 5 cm into the rectum. Several hundred holes had been drilled in the walls of the restraining cage and several large cutouts were made where possible for the dissipation of heat. Temperatures were measured on a Minneapolis-Honeywell recorder. Due to difficulties in restraining non-exhausted rats, a temperature reading at the end of the 1st min of recovery was not possible in these animals. Resting temperatures of all animals were recorded using restraint, but only after a 48-hr respite from forced exercise. All temperature measurements were conducted in the same thermostatically controlled environment as during exercise runs.

For the measurement of rates of O2 consumption in intact animals, a water-jacketed glass chamber (Quality Glass Blowing Co., Ann Arbor, Mich.) was used. Water temperature was maintained at 25.0 ± 0.1 °C by means of a heated and refrigerated bath (Forma Scientific, model 2095). In order to remove metabolic CO2, the chamber was preloaded with CO2 absorbent (Mallinckrodt Chemicals, St. Louis, Mo.), from which the animals were separated by a wire-mesh grid. Prior to the insertion of an animal, the walls of the chamber were swabbed with warm H2O and 30 ml of warm H2O in a plastic cup were placed in the chamber beneath the grid to insure saturation of the environment with H2O vapor. The system was then flushed with pure O2, which had been bubbled through a diffuser to saturate it with H2O, and sealed to await receipt of the animal. During insertion of the animal, the system was again flushed with H2O-saturated O2. A volume meter (model 160, Med-Electronics, St. Louis, Mo.), connected to the metabolic chamber to form a closed system, was used to measure O2 consumption by the animal. After a short period for pressure equilibration within the system following insertion of the animal, O2 consumption in ATPS was read directly from the volume meter. Gas volumes were subsequently corrected to STPD by means of the appropriate factors (9).

O2 consumption was observed in rats immediately after exhaustive exercise and during rest. To help minimize physical activity during measurements, a white cardboard shroud was placed around the cylindrical wall of the metabolic chamber, but the ends were left open so that animals could be observed. Initially, attempts were made to perform oxygen consumption measurements while animals were restrained, as during the determinations of tissue temperatures. Although the average resting values obtained using restraint were similar to some published values (16), the minute-to-minute values were very erratic. As expected, it was observed that attempts by the animals to free themselves from restraint resulted in increased O2 consumption during that period. If the animals were left in the chamber unrestrained, however, significantly lower, and less variable, readings were obtained. Therefore, it was decided not to use restraint during O2 uptake determinations and to perform measurements only during the ebb of the animal's diurnal activity cycle.

Postexercise O2 consumption was measured on 10 animals randomly selected from among those used in the temperature experiments. Animals were not postabsorptive since they were used immediately after removal from communal cages. The rats were again run to exhaustion and immediately sealed in the metabolic chamber. After a 2-min lag period, O2 consumption was recorded continuously at 1-min intervals over a 1-hr recovery period. During all metabolic measurements, rats were observed and activity was noted. In nonexercised animals, the lowest O2 consumption rate recorded for a consecutive 10-min period was taken to represent the resting rate.

Problems in measurements of O2 consumption due to movement were also encountered during recovery from exhaustive exercise. Immediately after exhaustion and insertion into the chamber, rats made no attempt to move about in the chamber. Generally, they remained quiescent for the first 15-20 min of recovery. After that time, however, they were usually sufficiently recovered to investigate their environment. To correct the metabolic measurements for this uncontrolled activity of animals, it was decided to report the data in 5-min intervals. Any sudden, unexpected, large 1-min readings associated with movement during recovery were rejected, and the previous minute's value substituted. The seven experiments reported are ones in which the animals were relatively inactive during recovery runs.

RESULTS

According to the well-known Q10 effect, one would expect to observe a direct correlation between tissue temperature and O2 consumption rate. As a reflection of this, a close temporal correspondence should exist between the decay curves for tissue temperature and O2 consumption rate. This is indeed the case.

Temperature changes after exhaustive exercise. Figure 1 shows the decline of muscle and rectal temperatures (mean ± se) after both exhaustive and moderate bouts of exercise. Resting temperatures (mean ± se) are indicated by cross-hatched areas. Severe physical exercise resulted in an 8.1 C increase in muscle temperature and 5.1 C increase in rectal temperature. Thus, the normal temperature gradient that existed between core and periphery at rest was reversed in the immediate postexercise period. On the cessation of extreme exertions (i.e., at exhaustion), muscle temperatures initially fell exponentially, but did not reach control values even after an hour (P < 0.02). Rectal temperature did not fall as rapidly as did muscle temperature and, in fact, in several instances did not reach a peak until the first minutes after exercise ceased. The rectal temperature curve was sigmoidal, with the most rapid phase of decrease occurring between the 4th and the 20th min of recovery. After about 20 min of recovery, a plateau was reached at about 40 C. Rectal temperature thereafter declined very slowly during recovery (slope = -0.025 C/min) and was still about 1.6 C above resting at the end of an hour (P < 0.01). Resting rectal temperatures (38.3 ± 0.1 C) corresponded to published values for rats of similar sex and age (7).
TEMPERATURE AND O$_2$ CONSUMPTION POSTEXERCISE

FIG. 1. Thigh muscle and rectal temperatures of rats as a function of time after exhaustive and moderate bouts of physical exercise. Each point represents mean ± se of seven experiments. Resting rectal and muscle temperatures (mean ± se) are indicated by cross-hatched areas. Points at 0 time after moderate exercise are extrapolations and confidence limits are not given.

**Temperature changes after moderate exercise.** At the end of moderate exercise (Fig. 1), muscle and rectal temperatures were significantly elevated compared to their resting levels. Both muscle and rectal temperatures at $t = 0$ were estimated by extrapolation from the adjacent empirically derived points, and hence confidence limits are not given. In the rectal temperature curve, this point coincided with the linear extrapolation of the plateau attained during recovery. Muscle temperature after moderate exercise fell exponentially, but unlike the exhaustive situation, resting levels were reached within an hour ($P = 0.4$). It is interesting that rectal temperature invariably reached a maximum several minutes after cessation of moderate exercise. This peak was transient, and within 5-10 min a gradual, steady rate of return to the resting temperature was established. As after severe exercise, the plateau in rectal temperature reached after moderate exercise was around 40°C. After an hour of recovery from moderate exercise, rectal temperature was still approximately 1°C above the resting level.

O$_2$ consumption in vivo. The decline in O$_2$ consumption rate after exercise in rats (Fig. 2) is similar to that reported previously (16). O$_2$ consumption was high immediately after exercise, and declined exponentially for approximately the first 20 min of recovery. Thereafter, O$_2$ uptake leveled off, and only a gradual rate of decline was apparent. During this plateau phase, the slope of the decline in the O$_2$ consumption rate with respect to time was only $-0.05$ ml O$_2$ kg$^{-1}$ min$^{-1}$. After an hour of recovery, the rate was still significantly elevated above resting ($P < 0.01$). Our measured values for resting O$_2$ consumption rates ($17.8 ± 0.5$ ml O$_2$ kg$^{-1}$ min$^{-1}$) better reflected the true resting metabolic rate than those of McArdle (16) ($23.4$ ml O$_2$ kg$^{-1}$ min$^{-1}$), who did not correct for physical activity, and approach published values of $11.9-1.26$ ml O$_2$ kg$^{-1}$ min$^{-1}$ established as the absolute minimum O$_2$ consumption in rats of similar age and sex while anesthetized or sleeping (7).

**DISCUSSION**

Large excursions in both internal and muscle temperatures in rats as a result of extreme exertion corroborate reports of similar findings in humans (18-20). Additionally, D. R. Lamb (personal communication) has observed that the gut temperatures of guinea pigs forced to exercise exceed those reached in rats by several degrees centigrade. As originally described by Nielsen (17) and discussed by Snellen (21) in a recent review, internal temperatures in humans during exercise are proportional to the overall metabolic rate. Our results obtained on rats confirm this.

Muscle and rectal temperatures at exhaustion of 44.1 ± 0.1°C and 43.4 ± 0.1°C, respectively (Fig. 1), indicate that the heat load generated by the musculature resulted in a large increase in both muscle and internal temperatures. Presumably, this heat load was sufficient to drive the internal temperature up despite the activity of heat loss mechanisms. Whereas at rest, core temperature always exceeds that of skeletal muscle (19, 20), this gradient is reversed following extreme exertion. Thus, the core may function to absorb the peripheral heat load helping to maintain muscle temperature below the points where a reduction in phosphorylative efficiency (4, 5) and massive irreversible thermal damage may occur.

Temperature changes in core and muscle after moderate bouts of exercise can be interpreted as supportive of the hypothesis that the core functions somewhat as a heat sink for the musculature. Such a mechanism may operate not only during exercise, but immediately afterward also. The peak in rectal temperature coinciding with the rapid decline
in muscle temperature after moderate exercise (Fig. 1) is interpreted as the result of heat being carried from the musculature to the core by vascular conduction. Similarly, the rapid decline in muscle temperature parallel with a slower decline in rectal temperature during the first 4 or 5 min of recovery from exhaustion (Fig. 1) can be explained as a "heat-dumping" phenomenon.

Following both exhaustive and moderate exercise, rectal temperature fell and then reached a plateau. In view of the great rate at which animals were able to dissipate heat in the early minutes of recovery, it is interesting that rectal temperature did not return to normal sooner. Nielsen (17) has hypothesized that during physical exertion there is an elevation in the set point of the hypothalamic temperature control center. Irrespective of ambient temperature or the efficacy of the environment for heat conduction, even moderate exercise has been observed to result in an elevation of internal temperature (21) and also, supposedly, the hypothalamic set point.

The great rates at which muscle and rectal temperatures fell immediately after the cessation of exercise also indicate that the restraining apparatus did not limit the dissipation of heat. Moreover, this conclusion is supported by the observation (data not reported) that restraint for periods as long as the postexercise recovery period (1 hr) did not result in increased temperatures.

Our experiments showed (Fig 1) that even after an hour of recovery from only moderate exercise, rectal temperature was still significantly elevated above normal, and only a gradual rate of return toward the resting value was apparent. We interpret this prolonged elevation in $O_2$ consumption after exercise to be due, in part, to the hypothalamic readjustment hypothesized by others (17, 21). The determinations by Benedict and Cathcart (3) of elevated BMR in humans 24 hr or more after the cessation of physical exercise may also be explained, in part, in this way.

Additionally, it is possible that the elevated postexercise internal temperature is, in part, a reflection of increased nonconservative respiration by a population of mitochondria which sustained a degree of thermal damage during exercise. Such a mechanism, suggested by our studies on isolated skeletal muscle and liver mitochondria (4, 5), cannot yet be excluded as contributory to elevated postexercise $O_2$ consumption.

The postexercise pattern of return to a normal rate of $O_2$ consumption in rats is similar, in general, to that of humans (23). However, because a rat's high resting metabolic rate prevents it from increasing its $O_2$ consumption more than 4 or 5 times during exercise (6), whereas in humans the exercise $O_2$ consumption rate may exceed 20 times the resting rate, there are some differences between the postexercise recovery curves of the two species. As opposed to the fairly rapid return toward normal of the $O_2$ consumption rate of humans after exercise (23), the $O_2$ consumption rate of rats recovering from exercise returned to normal only slowly (Fig. 2), with a relatively prolonged elevation in the plateau phase. Nevertheless, because of the similarity of the postexercise $O_2$ consumption curves among different species, the results of this investigation make possible a new and more meaningful description of the general phenomenon of elevated postexercise $O_2$ consumption referred to as the $O_2$ debt.

Figure 3 shows a composite of Figs. 1 and 2; the top set of coordinates depicts the rate of $O_2$ consumption after exhaustive exercise, and below this are shown the decay curves of muscle and internal temperatures. The decline in the rate of $O_2$ consumption after exercise is comprised of two phases. Phase 1 is typified by a rapid decrease in the rate of $O_2$ consumption. In the rat, this phase lasts 15 to 20 min (Fig. 2), and $O_2$ consumed during this period probably represents that used to reesterify creatine and ADP, perhaps...
at a reduced phosphorylative efficiency (4, 5), as well as that which is consumed due to the direct acceleration of
respiration by the elevated temperature. Cardiorespiratory
O₂ consumption due to the direct stimulation of cellular
respiration by increased muscle and core temperatures.
Moreover, mitochondrial studies (4, 5) clearly show that at
high physiological temperatures this increase in O₂ con-
sumption occurs at the expense of energy-trapping efficiency.

The data presented here strongly support our contention
(4, 5) that there exists a previously undescribed major
component contributing to the total O₂ consumption ob-
served after exercise which is independent of the so-called
“lactacid” and “alactacid” debts. This component is not
directly related to the repayment of an energy debt. It is,
however, largely nonconservative in nature, and is a conse-
quence of the well-known effect of temperature on the rates
at which biochemical reactions proceed. On this basis it
would appear that it is not valid to equate the extra O₂
consumed after exercise with the anaerobic metabolism
which occurs during exertion. Since much of the O₂ con-
sumed after exercise may be unrelated to recovery from
anaerobic metabolism, it is suggested that the applicability
of the term “O₂ debt” be carefully reexamined. Other
investigators (11, 22) concur with us in this conclusion. We
recommend the adoption of a descriptive term such as
“postexercise O₂ consumption” be considered.

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REFERENCES

1. Barclay, J. K. The Metabolism of Contracting Dog Skeletal Muscle in

2. Bannard, R. J., and M. I. Foss. Oxygen debt: effect of beta-
adrenergic blockade on the lactacid and lactacid components.


4. Brooks, G. A. Temperature, Skeletal Muscle and Liver Mitochondrial
Respiratory Functions, and Oxygen Debt (Doctoral Dissertation). Ann
Arbor: Univ. of Michigan, 1970.

Temperature, skeletal muscle mitochondrial functions, and oxygen debt.


7. Denckla, W. D. Minimal oxygen consumption in the female rat,
some new definitions and measurements. J. Appl. Physiol. 29:

8. Dipompero, P. E., and R. Margaria. Relationship between O₂
consumption, high energy phosphates, and the kinetics of the O₂


77: 352–353, 1921.


lactic acid and the supply and utilization of oxygen. Pt. IV—VI.


14. Margaria, R. Aerobic and anaerobic energy sources in muscular
exercise. In: Exercise at Altitude, edited by R. Margaria. Amster-

15. Margaria, R., H. T. Edwards, and D. B. Dill. Possible mechan-
isms of contracting and paying oxygen debt and the role of lactic

16. McArdle, W. C. Metabolic stress of endurance swimming in the

17. Nielsen, B. Die regulierung der korperentemperatur bi muskel-

18. Robinson, S. Physiological adjustments to heat. In: Physiology of

during submaximal exercise in man. J. Appl. Physiol. 25:

atues and sweating during thermal transients caused by exercise.


deficit, steady level O₂ uptake and O₂ uptake in recovery. Med.

Brooks. Ventilatory response during recovery from muscular work