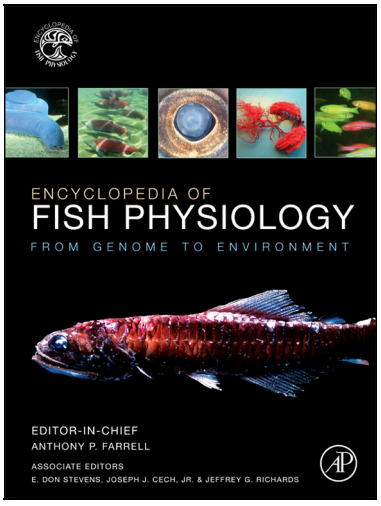

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General Energy Metabolism

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Glossary

Adenosine triphosphate (ATP) An almost universal carrier of chemical bond potential energy; fish use ATP made from catabolism of foodstuff or body reserve molecules to fuel energy-dependent processes.

Direct calorimetry The measurement of waste heat produced by metabolic processes to assess the rate of these processes.

Entropy A thermodynamic property measuring the amount of disorder in the system. Greater disorder is energetically favorable; thus, entropy favors unfolding of proteins.

Indirect calorimetry The measurement of O_2 or foodstuff molecules consumed or CO_2 produced to assess the rate of metabolic processes.

Mass exponent, β (also called allometric exponent) The power function exponent for the relationship of metabolic rate as a function of body size.

Metabolic scaling How metabolism changes as body size changes; typically summarized by the mass exponent.

$\dot{M}_{O_2}$ Mass of oxygen consumed by an organism. The SI unit is micromoles or millimoles per unit time, but often expressed as milligrams of oxygen per unit time. It can also be divided by the mass of the fish (e.g., mg-O_2 h^{-1} kg^{-1}) in which case, it is called 'mass-specific oxygen consumption'.

Quantile A value that divides a data set into parts. Where the division is made depends upon the parameter q. If $q = 0.5$, half the data are below the quantile and half above, which gives the median. If q is given as a percent instead of a proportion, the quantile can be called a percentile.

Standard metabolic rate (SMR) The minimum metabolic rate of survival. Typically, SMR is measured on resting, unstressed adult animals in the post-absorptive state under normothermic conditions. For fish, normothermic is defined as a temperature well within the species tolerance limits for which the animals have had ample time to acclimate.

Metabolic Rates of Fish

Measuring Metabolic Rate

Organismal energy use is thermodynamically inefficient, that is, about two-thirds of the potential energy of the reactants is lost as a less-useful form of energy known as heat during the execution of essential life processes. In addition, the catabolic reactions of the body that convert the energy in food to the adenosine triphosphate (ATP) required to fuel these process are themselves inefficient and generate heat. Thus, although the most useful indicator of total metabolic activity would be to measure the rate of total ATP turnover in an organism, in the absence of a convenient way to measure ATP turnover, accounting for the heat produced by these aggregate reactions is the best way to assess total metabolic activity, a process called 'direct calorimetry'. Unfortunately, the high heat capacity of water and the relatively low metabolic activity of fish result in a low 'signal:noise' ratio for direct calorimetry in aquatic studies. Therefore, we find limited measurements of metabolic rate by direct calorimetry available for the fishes; most studies instead rely upon a process called 'indirect calorimetry'.

Indirect calorimetry takes advantage of the fact that substances are consumed or produced during the catabolic conversion of foodstuffs to useful ATP energy. Eqn (2) depicts the complete aerobic respiration of an example foodstuff molecule, glucose:

$$C_6H_{12}O_6 + 36ADP + 36P_i + 36H^+ + 6O_2 \Rightarrow 6CO_2 + 36ATP + 42H_2O + Heat \quad (1)$$

Both oxygen (O_2) and carbon dioxide (CO_2) are gases at the range of temperatures and pressures encountered by fish; their stoichiometric consumption (O_2) or release (CO_2) can be monitored to gauge the rate of this reaction. Some studies also follow the decline in storage

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metabolites in starved fish as a way to monitor rates of respiration reactions. However, because starvation is a nonstandard physiological state, these types of experiments will not be discussed further here (**see also Gut Anatomy and Morphology**: Gut Anatomy). Because each foodstuff (protein, fat, or carbohydrate) produces different amounts of energy per amount of O_2 consumed or CO_2 emanated, accurate use of indirect calorimetry for bioenergetics requires a strict assessment of the substrate being respired. This is not a great challenge in laboratory situations, but may be impossible for most field studies because the exact composition of the diet is often unknown. Because measuring $[CO_2]$ in water is more challenging than measuring $[O_2]$, studies of fish metabolic rate over the past 30 years have relied upon measuring O_2 consumption almost exclusively, often without any attempt to relate the measurement back to energy usage. As such, oxygen consumption ($\dot{M}_{O_2}$) has actually become a measurement in its own right because without accounting for the substrate being oxidized or accounting for any anaerobic metabolism that occurs, it may be quite different from the actual metabolic rate. Recent careful comparisons of metabolic rate in endotherms using both direct and indirect calorimetry on the same animals suggest that the use of indirect calorimetry incurs routine errors of the order of 20%, with isolated cases as high as 35%.

Standard Metabolic Rate and its Measurement

$$C = (F + U) + (R + W + SDA) + (B + G) \qquad (2)$$

In eqn (2), a standard bioenergetics equation, (**see also Energetics**: Energetics: An Introduction) '*R*' represents the minimum rate of energy expenditure needed to keep a fish alive. It involves three broad classes of processes, the first of which is biosynthesis of macromolecules. Even when a fish is not growing, there is a constant, energy-requiring renewal of the macromolecules that make up the fish with molecules from the diet. The second class of processes concerns the chemical work of moving ions and molecules against concentration gradients or moving polar compounds across nonpolar membranes. These energy-requiring processes are essential for osmoregulation, the maintenance of internal cellular integrity, cellular communication, intra-organismal communication (e.g., action potentials or hormone release), and the transport of food molecules. The third class of processes concerns the internal mechanical work required to preserve organismal integrity (e.g., work done by the heart to accelerate blood).

R is often called basal metabolic rate (BMR) in endotherms and standard metabolic rate (SMR) in ectotherms. *R* allows for no activity, digestion, or reproduction, and care needs to be taken to exclude other sources of energy expenditure when measuring it. Measurement methodologies for BMR in endotherms are well established. In humans, for example, BMR is measured in subjects who are awake, supine, fasted for 12 h, motionless, after a 20–30-min period of rest, and isolated from external stimuli in a thermoneutral dark environment. Even so, BMR is not without criticism because metabolic rate can decrease further during sleep. Nevertheless, the stringent measurement conditions facilitate comparisons between studies and can also be applied to many nonhuman mammals.

By contrast, the conditions in which SMR is measured in aquatic ectotherms are more loosely defined. SMR is typically observed in postabsorptive (but not starving), resting organisms after acclimation to the experimental temperature and apparatus, isolated from outside stimuli (including sensory stimuli from potential predators and possibly conspecifics), during the part of the circadian activity cycle when $\dot{M}_{O_2}$ is lowest. Strictly speaking, SMR should be measured only in animals that are in a steady state (no growth or reproduction). However, the minimum metabolic rate of juvenile fish is often called SMR and the growth of many fish species is indeterminate, so the no-growth criterion is often not met. SMR of adult fish should be measured outside of the reproductive season, although in practice, the reproductive status of fish used in SMR determinations is not always known. Because there is usually some activity during measurements of metabolic rate, or because activity has not been monitored, many authors refrain from using the term SMR and instead use resting, fasting, or most commonly resting routine metabolic rate. However, this may be unduly restrictive and the term SMR should be allowed when activity level is measured and known to be at the minimum possible for the species.

Handling stress has undoubtedly inflated many published estimates of SMR in fishes. Human contact and air exposure need to be minimized before measuring SMR. Visual contact with humans or laboratory noises can elevate metabolic rate considerably even in the absence of movement. An acclimation period is required before $\dot{M}_{O_2}$ measurements can be used to estimate SMR. Handling stress and the novelty of the experimental setup may elevate $\dot{M}_{O_2}$ in fish for hours or even days. Sufficient acclimation can be verified by measuring $\dot{M}_{O_2}$ during the acclimation period to confirm that the fish has reached a state where repeatable measurements of minimum metabolic rate can be made. Striking a balance between ensuring that an animal is only postabsorptive and not starving with adequate acclimation may prove to be difficult. In social species, isolating fish to measure SMR may produce stress and increase $\dot{M}_{O_2}$. It may be necessary to determine SMR for groups of fish, after examination of the relationship between $\dot{M}_{O_2}$ and group size.

Spontaneous activity is extremely difficult to control or even measure in fish, yet activity can increase metabolic rate by an order of magnitude (**see also Ventilation and Animal Respiration**: The Effect of Exercise on Respiration). One approach has been to circumvent the problem by measuring $\dot{M}_{O_2}$ during forced activity at different speeds, and then by extrapolating the relationship between $\dot{M}_{O_2}$ and swimming speed back to zero swimming speed. However, fish can engage in spontaneous locomotor activity, have additional maneuvering costs at high speed, or be subject to stress, in addition to the sustained swimming at low speeds, thereby elevating $\dot{M}_{O_2}$ and the predicted $\dot{M}_{O_2}$ at zero speed. Because of this, $\dot{M}_{O_2}$measured at low swimming speed is generally less repeatable than at higher speeds, so if this method is employed, relatively high, but still aerobic, swimming speeds should be used. Another concern is that the routine metabolism of some organ systems (e.g., the gut) can be turned down with swimming activity, which, if left unaccounted, could lead to an underestimate of SMR.

An alternative method is to measure the level of spontaneous activity in a static respirometer, relate $\dot{M}_{O_2}$ to activity level (i.e., through regression analysis), and use the intercept (i.e., at zero activity) of this relationship as SMR. Yet another method is simply to estimate SMR only on measurements obtained when activity is zero. Some authors have employed anesthesia to eliminate activity from SMR measurements, but this method is not recommended because the anesthetic may interfere with other functions, including those responsible for SMR. However, for very active, dangerous or obligate ram-ventilating species, this may be the only tractable method available, in which case, the minimum dose to eliminate swimming activity should be used.

There is more uncertainty in estimating SMR when activity is not accounted for in static respirometry. Unless the period of minimum spontaneous activity is known for the species, $\dot{M}_{O_2}$ must be measured over at least 24 h; longer experiments increase confidence in estimated SMR by verifying that similar low levels of $\dot{M}_{O_2}$ are observed for the circadian minimum on multiple days. Long records of $\dot{M}_{O_2}$ can be obtained using two different respirometry techniques: open-flow respirometry and intermittent-flow respirometry. However, there is no standardized method to estimate SMR from these records. It may be nonadvisable to use the lowest value of $\dot{M}_{O_2}$ because it could be the result of measurement error. Often, an arbitrary number of the lowest values of $\dot{M}_{O_2}$ observed after the fish is deemed acclimated are averaged to obtain SMR, sometimes after removing 'outliers' that are arbitrarily also identified.

Another approach is to fit a mixture of normal distributions to the frequency distribution of $\dot{M}_{O_2}$ values. The mode with the lowest $\dot{M}_{O_2}$ can be interpreted as SMR; it constitutes the lowest value of $\dot{M}_{O_2}$ that is most frequently observed (**Figure 1**). This approach has two main advantages: (1) estimation of SMR is based on a large number of observations, and (2) it is unnecessary to manually select which measurements are used to estimate SMR. It turns out that this method does not work when there are many measurements with moderate activity or stress. It becomes impossible to discriminate the normal distribution corresponding to SMR from the distribution(s) corresponding to the moderately active or stressed fish and estimated SMR resembles routine metabolic rate (i.e., the metabolic rate of a moderately active fish, **Figure 2**).

Removing the 'noisy' part of the signal may improve the estimation of SMR by this approach, but the second advantage (see above) is negated (**Table 1**)

Yet another approach is to define SMR as a quantile: a proportion q (e.g., 0.05–0.25) of the measurements are assumed to be below SMR and the remaining $1-q$ of the values above SMR. Models suggest that values of 0.10–0.15 result in small errors in estimating SMR across a broad range of activity and stress levels and could be used even in situations when fitting normal distributions (**Table 1**). However, the quantile approach has not yet been adopted widely to estimate SMR.

The postabsorptive state is also loosely defined in SMR studies on fish. Only rarely is gut-passage time measured for the same species under the same conditions; most studies rely on postabsorptive times from the literature that may not account for temperature or dietary differences (e.g., gut passage being significantly longer in herbivorous fishes (**see also Food Acquisition and Digestion**: Digestive Efficiency) or scaling effects (e.g., using published values based on fish of a different size; **see also Energetics**: Physiological Functions that Scale to Body Mass in Fish).

Body Size and SMR

Of the factors that contribute to differences in SMR, body size has to be considered the most pervasive. An animal's metabolic rate changes with body size in a nonproportional manner such that tissues of larger animals consume less energy per unit tissue than those of smaller animals (**see also Energetics**: Physiological Functions that Scale to Body Mass in Fish). The original mammal-centric literature on the subject dating to the nineteenth century considered this as a consequence of heat dissipation requirements by endothermic organisms and concluded that SMR scaled as the surface area to volume ratio or as body mass (M_b) to the two-thirds power:

$$\mathrm{MR} = C{M_b}^{0.67}$$

often expressed in the logarithmic form:

$$\log\ \mathrm{MR} = \log\ C + 0.67\ \log\ M_b$$

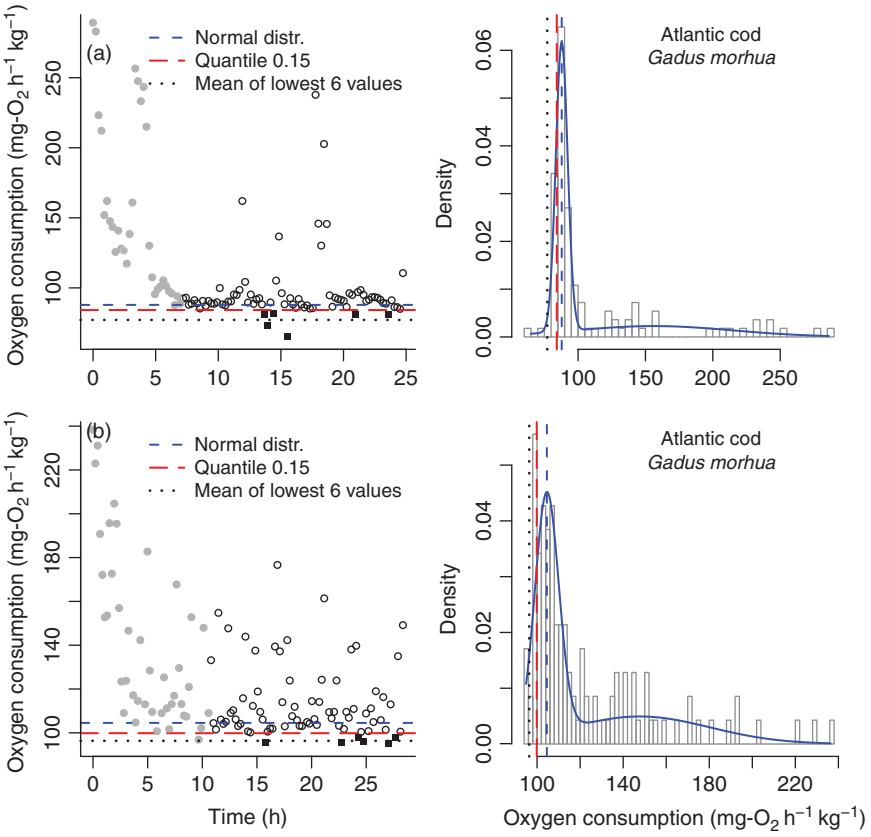

Figure 1 Oxygen consumption ($\dot{M}_{O_2}$) as a function of time in the respirometer (left), and frequency distribution of $\dot{M}_{O_2}$ values (right). (a) Juvenile Atlantic cod (*Gadus morhua*) with steady low levels of $\dot{M}_{O_2}$ and (b) juvenile cod with more variable $\dot{M}_{O_2}$ after acclimation. Gray circles and open circles represent values recorded during or after the acclimation period, respectively. The black squares are the six lowest values recorded after the acclimation period. Three estimates of SMR are shown: the average of the six lowest observations of $\dot{M}_{O_2}$, the lowest mode from fitting a mixture of normal distributions to the frequency distribution of $\dot{M}_{O_2}$ values shown in the right panels, and the quantile 0.15. The last two estimates were obtained using all available data, including the acclimation period.

where, MR is the metabolic rate, M_b the body mass, C the proportionality coefficient or MR at unit body mass, and 0.67 is β, the slope or power exponent describing the rate of change of metabolic rate with increasing body size. Subsequent empirical measurements throughout the twentieth century often arrived at a general interspecific 0.75 scaling coefficient for the metabolic rate of both ecto- and endotherms (i.e., $M_b^{0.75}$), but without a generally accepted physiological mechanism to account for it. Throughout the latter part of the twentieth century, measurements on fish generally returned a wide range of scaling coefficients. Knut Schmidt-Nielsen, in his seminal 1984 book on scaling, decried the huge variation in intraspecific β values for fish (0.37–1.1) and settled on a value of about 0.8 as the best. As the twentieth century was drawing to a close, several major new theories were advanced to either explain or discount the 0.75 scaling coefficient. These new theories created renewed interest in the field and stimulated many scientists to examine old data sets or undertake new measurements. To date, evidence has accumulated that metabolic scaling in fish may be higher than 0.75 (**see also Energetics**: Physiological Functions that Scale to Body Mass in Fish), especially if larval fishes are included. Scaling of metabolic rate under active conditions (**see also Swimming and Other Activities**: Energetics of Fish Swimming) appears to approach 1 for fish. Overall, SMR change with size is not proportional in juvenile through adult life stages in fish, and the exact scaling coefficients may be species specific. Certainly, the best way to correct for body-size differences, within a species, is to either find a carefully collected data set or construct one's own scaling factor, using specimens across a wide body-size range. Another way is to use an analysis of covariance (ANCOVA) to analyze data, using body mass as the covariate.

Sources of Variation in SMR

After correcting for body size, there still remains substantial inter-specific and intra-specific variability in SMR. Perusal of the literature can return values that vary by more than 70-fold for fish of similar lifestyles corrected

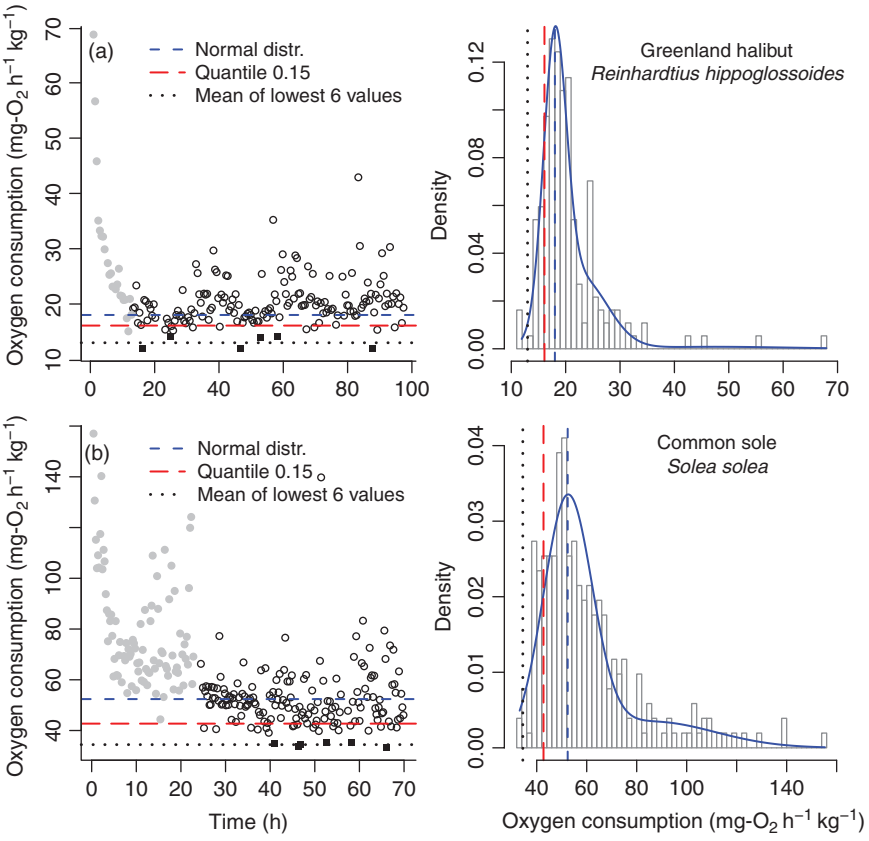

Figure 2 Oxygen consumption ($\dot{M}_{O_2}$) as a function of time in the respirometer (left), and frequency distribution of $\dot{M}_{O_2}$ values (right). (a) Mature female Greenland halibut (*Reinhardtius hippoglossoides*) and (b) juvenile common sole (*Solea solea*), both with high variability in $\dot{M}_{O_2}$ after the acclimation period. Symbols and lines are as in **Figure 1**.

Table 1 SMR estimated for the four fish shown in **Figures 2** and **3** using five different methods on four individuals from three species: averaging the six lowest values observed after the acclimation period, fitting a mixture of normal distributions to the frequency distribution of $\dot{M}_{O_2}$ measurements and selecting the leftmost mode, and taking the quantile 0.15, the latter two both with and without the acclimation period

			Normal distributions		*Quantile$_{0.15}$*	
Species	*Figure*	*Average of Six lowest values*	*All data*	*Excluding acclim. period*	*All data*	*Excluding acclim. period*
Atlantic cod	2A	77.1	87.6	87.2	84.2	83.1
Atlantic cod	2B	96.4	104.6	103.4	99.8	98.9
Greenland halibut	3A	13.0	18.1	18	16.1	16.1
Common sole	3B	34.4	52.7	47.9	42.7	41.1

for size and temperature. Much of this variation is due to experimental differences between laboratories, starting with how SMR was calculated. Many studies of standard or resting routine metabolic rate in fishes did not adequately control for human presence or elapsed time from human contact or transfer to the respirometer (see above). Thus, many of these values are inflated beyond standard or even resting routine levels. Another factor contributing to the large variance in standard metabolism estimates between species and studies are differences in water chemistry. Because of the constraints of Fick's first law of diffusion (eqn (3))

$$\mathcal{J}_{net} = DA\Delta P_{gas/\Delta d} \quad (3)$$

(where J_{net} is the net diffusional flux between two compartments, A the area over which the flux is occurring, ΔP the difference in partial pressure of the gas between the two compartments, Δd the distance separating the two compartments and D a constant), and the need for fish to extract O_2 and release CO_2, respiratory surfaces such as

the skin or gills need to be large surface-area structures with a short diffusion distance to the external environment. These characteristics present an opportunity for the salt and water of the environment to also equilibrate across electrochemical gradients in water-breathing fish (**see also Role of the Gills**: The Osmorespiratory Compromise). Fish need to counteract these movements through active, energy-requiring processes. This fundamental conflict between the need to exchange respiratory gases and the relative cost of osmoregulation in water-breathing fish has been termed the 'osmorespiratory compromise'. Active osmoregulatory and acid–base processes will contribute to the animal's SMR, accounting for some of the variance between studies performed under different water chemistries. There is a fairly extensive literature attempting to define the cost of osmoregulation, that is theoretically at a minimum in environments isoosmotic to body tissues, in fishes. Anadromous fishes and euryhaline fishes from primarily freshwater groups tend to conform to this theoretical prediction; however, euryhaline fish from primarily marine groups seem to generally have their lowest SMR's in full-strength seawater.

Even intraspecific, SMR measurements made within the same laboratory may vary as much as 10-fold between individuals of the same size at the same temperature. What accounts for this degree of variation in oxygen consumption? Studies of fish eggs have shown substantial genetic variation in SMR even before hatching. Studies of clonal fishes have also shown substantial environmentally induced variance in metabolic rate even in laboratory-reared fish, suggesting both genetic and environmental involvement. Respiratory rates of mitochondria corrected for temperature do not vary at the same magnitude as do whole animal metabolic rates among individuals of the same species or even across different species. Thus, differences in organismal metabolic rates corrected for body size and temperature differences come from different densities of mitochondria or differential patterns of either mitochondrial or whole tissue activation.

Some of the interspecific differences in SMR may arise from differential species-level activity patterns. The more aerobically active a species is, the more mitochondria they need to fuel those periods of activity. If the mitochondria can only be throttled down to a certain level, higher rates of standard metabolism are inevitable. It has been known for some time that active fish generally have a larger gill surface area and a higher muscle–mitochondrial density than less active fish. Thus, a more active species will have a relatively greater cost of osmo- and acid–base regulation and will have to maintain more metabolically expensive tissue like gills with high protein-turnover and ion-transport rates. A general linkage of SMR with activity metabolism has been found in many taxa and formed the basis of the activity metabolism hypothesis that was proposed to explain the evolution of endothermy in birds and mammals. Extrapolating this argument to the intraspecific level in fish, differential temporal selection regimes for either efficiency in standard metabolism or aerobic activity could account for the large variance in SMR that is apparent in extant populations. However, some studies have found that exercise training in fish can simultaneously increase muscle–mitochondrial density and decrease whole-animal SMR.

There is also some evidence that population-level differences account for some of the observed intraspecific variance in SMR. Regional differences in natural selection for SMR or traits linked to SMR could produce populations that differ significantly in SMR. There is some evidence that energetically expensive morphological traits that facilitate high rates of aerobic metabolism such as a high density of mitochondria (**see also Temperature**: Mitochondria and Temperature), large hearts (**see also Design and Physiology of the Heart**: Cardiac Anatomy in Fishes), and higher capillary densities (**see also Design and Physiology of Capillaries and Secondary Circulation**: Capillaries, Capillarity, and Angiogenesis) may be outcomes of natural selection for fast recovery from exercise and not for exercise performance *per se*, because the highest rates of performance in fish are fueled anaerobically, and fish with higher resting metabolic rates recover faster from exhaustive exercise. This could result in a bimodal composition of some fish populations, one mode composed of cruising specialists (e.g., with higher SMR, higher aerobic performance, quick recovery from strenuous swimming, but low maximum-burst speeds) and a second mode composed of sprinting specialists (e.g., with higher maximum-burst speeds, but lower SMR, lower aerobic performance, and slow recovery from strenuous swimming), both maintained through disruptive (or diversifying) selection in the wild.

Temporal and geographic factors may also influence SMR, independent of the controlling factors of temperature and sexual maturation (**see also Swimming and Other Activities**: Cellular Energy Utilization: Environmental Influences on Metabolism). Certainly, the evidence is better for the former. Several studies have shown seasonal cycles of SMR in sexually immature fish kept at constant temperature. Krogh suggested, as early as 1915, that polar ectotherms might compensate for the effects of temperature on reaction rate by elevating temperature corrected SMR ('idling the engine faster'). This was eventually termed the metabolic cold adaptation (MCA) hypothesis and found enough early support that it was generally accepted. However, recent studies have failed to find any difference in SMR of polar fish compared with SMR extrapolated to the same temperatures for temperate and tropical species. Therefore, early measurements of $\dot{M}_{O_2}$ in polar species may have overestimated SMR, because polar fish measured in cold

water require longer acclimation periods (just to recover from handling stress) and longer periods to become post-absorptive (**see also Food Acquisition and Digestion**: Cost of Digestion and Assimilation).

Conclusions

Despite a century of concerted effort, we have to conclude that the measurement of fish SMR is in its infancy. The lack of standardized methods and uncertainty regarding stress level or human contact in a given study leaves much of the published literature uninterpretable. The situation is far better for swimming metabolism (**see also Swimming and Other Activities**: Energetics of Fish Swimming), although repeat measurements on the same fish are rarely made to verify results. Similarly, published scaling coefficients for SMR in fish span an incredible range, with many near 0.85 for adults and juveniles and 1 for larvae. Considering the current uncertainty in fish SMR values and how they change with body size, parameterization of bioenergetic models for real-world applications can contain many uncertainties. SMR corrected for temperature and body size is quite variable both between species and within species. Much current research is focusing on the relationship between these intrinsic differences in SMR and factors that are directly related to fitness such as dominance hierarchies or aerobic scope.

See also: **Design and Physiology of Capillaries and Secondary Circulation**: Capillaries, Capillarity, and Angiogenesis. **Design and Physiology of the Heart**: Cardiac Anatomy in Fishes. **Energetics**: Physiological Functions that Scale to Body Mass in Fish. **Food Acquisition and Digestion**: Cost of Digestion and Assimilation; Digestive Efficiency. **Gut Anatomy and Morphology**: Gut Anatomy. **Role of the Gills**: The Osmorespiratory Compromise. **Swimming and Other Activities**: Cellular Energy Utilization: Environmental Influences on Metabolism; Energetics of Fish Swimming. **Temperature**: Mitochondria and Temperature. **Ventilation and Animal Respiration**: The Effect of Exercise on Respiration.

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