Summary. A reanalysis of data presented by Henneman (1983) to examine the relationship between intrinsic rate of increase and basal metabolism suggests there is no statistically significant correlation between the two variables when both are treated with comparable statistical techniques. In addition, I suggest there is no theoretical reason to expect a high correlation between basal metabolic rate and a population's maximum rate of increase.

Reanalysis of Hennemann's data

Hennemann (1983) obtained BMR values from the literature and calculated \( r \) using an equation given by Cole (1954). He then computed the expected values for \( r \) and BMR from allometric power functions: however, he did so differently for the two parameters. For \( r \) he used a least squares regression of log-transformed values to define empirically an allometric equation for the 44 species for which he has data. However, to generate expected BMR measures for these same 44 species he again used least squares regression but on a different data set - one which is taken as the mammalian standard but which, in fact, is composed of only 12 species, nine of which are domestic or traditional laboratory stock (Kleiber 1961). The equation for mass-specific basal metabolism from Kleiber's (1961) data (BMR = 1.8W_{gals}^{-0.18}) is substantially different from that which can be derived from Hennemann's compilation (BMR = 3.4W_{gals}^{-0.25}). In that Hennemann (1983) assumed his data to be representative of mammals, while Kleiber's (1961) sample clearly is not, Hennemann should have used the allometric function computed from his own data. Instead he compared deviations in BMR with respect to Kleiber's equation with deviations in \( r \) with respect to the empirically derived allometric function for \( r \). Using the non-parametric Spearman rank correlation coefficient (\( r_s \)) to examine the correlation between the two sets of deviations, Hennemann (1983) found a small positive correlation between them (\( r_s = 0.2545 \)) which was significantly different from zero (\( P < 0.05 \), one-tailed test). However, when I compared deviations in BMR with respect to the allometric function derived from Hennemann's (1983) sample with those for \( r \), there was no significant correlation (\( r_s = 0.2300 \); \( P > 0.05 \); one-tailed test; Zar 1974). Thus, using parallel methods of analysis for both parameters reduces the correlation to statistical non-significance.

A second and more direct method for estimating the relationship between two variables while holding a third constant is that of partial correlation (Zar 1974). The partial correlation of BMR and \( r \) holding body mass constant (log scale for all three) is 0.1547, which is not significantly different from zero (Zar 1974). Thus from Hennemann's own data there is no reason to conclude that the intrinsic rate of increase is correlated with the basal metabolic rate.

Is McNab's (1980) hypothesis invalid or is Hennemann's (1983) test of it simply inappropriate? Hennemann's (1983) test attempts to find a correlation between BMR and \( r \). As indicated above, when a congruent measure of BMR is used, the correlation is not statistically significant. Is Hennemann's measure of \( r \) appropriate? The equation

\[
1 = e^{-a} + be^{-as} + be^{-(a+1)}
\]

which Hennemann (1983) used to evaluate \( r \) from ages at first (a) and last (w) reproduction, and annual litter size (b) was originally derived by Cole (1954) to investigate the effects of these reproductive parameters on population growth while holding other variables (most importantly, survivorship) constant. Any interactions between metabolism and survivorship are ignored in Hennemann's analysis. In addition, Cole's equation was not intended as a method
for the estimation of \( r \) from field-gathered life history data but rather as a theoretical tool for the exploration of the populational consequences of certain life history phenomena. Hennemann (1983) observed that the estimation of \( r \) from Cole's equation assumes that the population experiences no mortality (infant, juvenile, or adult) until after reproduction has ceased, and that litter size and inter-litter intervals are constant. Without supporting data, Hennemann (1983) assumed these assumptions are realistic for mammals greater than 100 g. However these assumptions certainly do not describe the conditions under which the life history data Hennemann (1983) used were collected. In general, the environmental constraints present in natural populations will produce 1) litter sizes smaller than those which are physiologically possible and which are more variable (due to in utero, infant, and juvenile mortality); 2) an average age at last reproduction younger than that maximally possible (again due to mortality); 3) an age at first reproduction often later than possible under ideal conditions (due to social or environmental inhibition, e.g., cold, photoperiod); and 4) inter-litter intervals often longer (and certainly more variable) than those ideally possible (again due to environmental effects on hormone levels) (Sadleir 1969; Dyrmundsson 1981; Gilmore and Cook 1981). The overall effect of using the environmentally influenced parameters will, in almost all cases, be to underestimate \( r \) as calculated by equation (1), but by a different amount depending upon the parameter affected and the species observed. In addition, as Hennemann (1983) indicated, larger mammals are more likely to fit the assumptions of Cole's (1954) equation than are smaller ones. This is due to the shape of mortality curves as well as the lower variability of litter sizes in large mammals. Thus the error in estimation of \( r \) may be greater in smaller species. Hennemann's (1983) comparative BMR data are also biased with respect to body mass in that the exponent in Kleiber's equation, which he used, differs from that derived from his own data. Together, these biases might have produced an artificial association between BMR and \( r \) in his analysis, and thus, Hennemann's (1983) test of McNab's (1980) hypothesis may have been inappropriate.

**Examination of McNab's hypothesis**

McNab (1980) hypothesized a relationship between reproductive metabolic rate and the Malthusian parameter (\( r \)), which is defined by the equation

\[
1 = \sum e^{-\alpha l(x)}m(x)
\]

in which \( l(x) \) and \( m(x) \) are age specific survivorship and fecundity schedules. McNab (1980) proposed that mammals which metabolize nutrients faster can turn those nutrients into offspring faster, thus increasing fecundity. Increasing fecundity would increase \( r \) and "all species are as \( r \)-selected as is possible" (McNab 1980:119). While higher energetic turnover may contribute to increased fecundity, \( m(x) \), it may also affect survivorship, \( l(x) \). As Sacher (1976, 1979) has indicated, longevity is inversely related to metabolic rate. Higher metabolism may lead to decreased survivorship at all age classes, perhaps negating the advantages of increased fecundity. In addition, the vast literature on \( r \) vs \( K \) selection suggests that higher \( r \) values are primarily advantageous in short-term, unpredictable environments in which competition, as well as population density is low (see Stearns 1976, 1977 for review).

Even if all energetic resources were channeled into increasing \( r \), this would not mean that basal metabolic rate must be correlated with \( r \). This would be true only if basal metabolic rate were predictably and uniformly correlated with reproductive metabolic rate. Such a correlation assumes that selection operates in a uniform fashion on both basal and reproductive metabolic rates. Since the two metabolic rates do not perform the same function, the assumption of equal selection pressures does not seem reasonable.

If basal conditions are experienced frequently enough under natural conditions to be of ecological relevance, then adaptive basal metabolisms would be those which maintain life at the lowest possible energetic cost. The primary function of metabolic rates during reproduction is to transfer energy into offspring as quickly and efficiently as possible. Although marsupials may, on average, have lower BMRs than eutherian mammals, work on the North American opossum, *Didelphis virginiana*, suggests that the rate of energetic turnover during reproduction in this marsupial is not different from that found in eutherians (Fleming et al. 1981). Thus, basal metabolic rate may determine the lowest rate of energetic turnover during reproduction, but there is no reason to expect it to be strongly correlated with average reproductive metabolism. If BMRs are not consistently related to reproductive metabolic rates then there is no reason to expect a correlation between BMR and \( r \).

The assumptions of McNab's (1980) hypothesis do not seem reasonable for a majority of mammals. BMR is not expected to be highly correlated with reproductive metabolic rate across mammals. High metabolic turnover may decrease survivorship as it increases fecundity, and the species-specific intrinsic rate of increase is only one of many factors which influence an individual's actual reproductive success or fitness in the environment in which it is found. Species have had many millenia in which to coordinate the extraordinarily complex interactions between survival and reproduction. We should not be surprised if the interplay between the major components of an animal's fitness (e.g., reproduction, survival, competitive interactions, and energetic allocations) are not a simple function of \( r \), BMR, and body mass.

**References**


