Introduction

It is somewhat disconcerting to have been asked to give the Samuel Brody Memorial lecture for the second time. Clearly my first attempt must have been regarded as less than satisfactory and I can only conclude that the organizers thought that I should be given a second chance! This I welcome because it enables me once more to pay tribute to a remarkable man whom I had the pleasure and stimulus of meeting and to show how his prescience continues to illuminate much of what we do in the biological sciences. Morgan (1960), in her biography of Samuel Brody, wrote of his culminating research (Brody, 1945), “seldom has so comprehensive, scholarly and original a tome been added to our scientific heritage.” With her view I wholeheartedly agree; one can find in that book ideas related to overall aspects of metabolism that are still central to much current investigational research.

Brody’s genius resided in his ability to integrate vast amounts of information so as to obtain beguilingly simple relationships. His approach may well be regarded as reflecting more closely the scientific ethic of the 19th century rather than the reductionism characteristic of our present age. His search was for unifying principles and his concern was with the whole animal rather than with the biochemical and biophysical processes, which must underlie this uniformity. Perhaps this emphasis on the whole has been the reason why his research has engendered so much; exceptions to his generalities and the search for explanation of them in other conceptual frames have proved intriguing to countless investigators.

I wish in this paper to begin with some of Brody’s broad concepts of bioenergetics and growth, particularly those that Brody did not develop, although I am sure he was aware of them, and dealt with their interrelations. I will continue by exploring some of these broad concepts in more biochemical terms.

Basal Metabolism and Body Weight

In 1932, Brody and Proctor published their study of basal metabolism of mature animals of different species, as did also Max Kleiber. As shown in figure 1, Brody and Proctor (1932) found by regressing the logarithm of basal heat production on the logarithm of weight that metabolism was proportional to body weight raised to the power .734. Kleiber (1932) estimated the logarithmic slope to be 3/4. Later (in 1961), Kleiber stated that the slope estimated statistically from his original set of data was .739. Brody and Proctor (1932) suggested rounding down to .7 while Kleiber (1932) had clearly rounded up. Each had used sets of data that gave indistinguishable values. In both sets the errors were such that either conclusion was reasonable and contentions about priority for this discovery are best resolved by calling this the Brody-Kleiber relationship.

There is little doubt that metabolism varies with a power of body weight greater than 1/2, which would apply if heat production was proportional to surface area, and less than 1.0, which would apply if metabolism was proportional to weight. Analyses of the data up to 1971 by both Poczopko (1971) and Blaxter (1972) showed this unequivocally for the Mammalia and Hemmingsen (1960) had shown that the same power of weight also scales metabolism in poikilotherms and in unicellular organisms. There are, however, differences even within the Mammalia as a class. As shown in table 1 and figure 2, there are real differences between subclasses and orders within the Mammalia with respect to the coefficient to be attached to weight raised to the power .75. Metatherian mammals have a lower metabolism than Eutherian ones and within the Eutheria the Chiroptera have a particularly low metabolic rate. When data for birds are considered as well (Lasiewski and Dawson, 1967), there appears to be a relationship between the deep body temperature of a group and their metabolism per unit of what Brody called “metabolically effective body weight,” W<sup>75</sup>. This is shown in table 2.
and it suggests that because heat emission is proportional to the temperature gradient from deep body to air, the original ideas that basal metabolism was in some way related to heat emission may contain an element of truth, but not in the way envisaged by the so-called surface area law.

**TABLE 1. FASTING METABOLISM OF 54 MAMMALIAN SPECIES CLASSIFIED ACCORDING TO ORDER OR INFRA-CLASS**

<table>
<thead>
<tr>
<th>Order or class</th>
<th>Slope &amp; error</th>
<th>kJ/kg W^{0.734}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polyprotodonts</td>
<td>.753 ± .046</td>
<td>221</td>
</tr>
<tr>
<td>Diprotodonts</td>
<td>.756 ± .018</td>
<td>195</td>
</tr>
<tr>
<td>Rodents</td>
<td>.751 ± .010</td>
<td>276</td>
</tr>
<tr>
<td>Primates</td>
<td>.757 ± .028</td>
<td>291</td>
</tr>
<tr>
<td>Artiodactyls</td>
<td>.811 ± .076</td>
<td>277</td>
</tr>
<tr>
<td>Perissodactyls</td>
<td>.793 ND</td>
<td>290</td>
</tr>
<tr>
<td>Bats</td>
<td>.790 ± .136</td>
<td>76</td>
</tr>
<tr>
<td>Marsupials</td>
<td>.754</td>
<td>214</td>
</tr>
<tr>
<td>Eutherians</td>
<td>.761</td>
<td>281</td>
</tr>
</tbody>
</table>

The slopes were determined by logarithmic regression analysis: none of the values was significantly different from .75, the Brody-Kleiber value. Only one value was included for each species in the analysis.

**Explanations of the Brody-Kleiber Relationship**

Understandably, there have been numerous explanations put forward about why, between adults of different species, metabolism varies with the power 3/4 rather than 3/5. A recent one relates to the possibility that the primary data are complicated by failure to take into account circadian variation in metabolism (Prothero, 1984). Most of the explanations have been reviewed (Pedley, 1977; Calder, 1981; Economos, 1982; Peters, 1983). It suffices to state that some explanations propose that metabolic rate is composed of two or more components, varying with different exponents of weight. A variant of this is that one component is weight-related, thus dependent on the gravitational field, and the other mass-dependent (Economos, 1979); a supposition negated by the observation that the metabolism of men in Space Lab was unaffected by zero gravity. Another suggestion has been that the difference between the observed power of .734 and that relating surface area to mass can be regarded as an effect of time that can be expressed as a linear dimension ( Günther and Martinoya, 1968; Günther, 1975). This constitutes perhaps what many scientists would call a "fudge factor," though it is distinguished by the term "operational time expo-
The most plausible explanation stems from the analysis of animal shape by Rashevsky (1960) and by McMahon (1973). Animals of different body weights are not homologous furry balls but have to be constructed, not only to resist compressive stress, but also buckling stress. Analysis of the linear dimension at which failure of a linear structure occurs shows that it is proportional to the \( \frac{3}{2} \) power of its diameter.

\[
\text{mass at failure (length cubed)} \propto \text{diameter squared} \quad \therefore \text{diameter} \propto \text{mass}^{\frac{3}{2}}.
\]

This means that mass at failure (length cubed) is proportional to diameter squared and diameter is thus mass raised to the power \( \frac{3}{2} \). Muscle energetics shows that power output depends solely on cross-sectional area. On the basis of this reasoning, power output will vary with diameter squared, namely with mass raised to the power \( \frac{3}{4} \).

Whether such arguments are reasonable or not, plausible or not, or whether they are simply intellectual exercises, the fact remains that Brody regarded the relationship between metabolism and weight as empirical and metabolic body size as a convenience in scaling attributes of animals of different body weight, particularly with respect to their maintenance needs of energy. If fasting metabolism is described by the Brody-Kleiber relationship,

\[
H = BW^{3/4},
\]

This means that metabolism (kJ/d) is proportional to BW to the power \( 3/4 \).

### Table 2. Metabolic Rate and Body Temperature in Mammals and Birds

<table>
<thead>
<tr>
<th>Group</th>
<th>Metabolism, kJ/kg W(^{3/4})</th>
<th>Temperature, °C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paserines</td>
<td>116</td>
<td>42</td>
</tr>
<tr>
<td>Eutherans</td>
<td>67</td>
<td>39</td>
</tr>
<tr>
<td>Marsupials</td>
<td>51</td>
<td>35</td>
</tr>
<tr>
<td>Bats</td>
<td>18</td>
<td>31</td>
</tr>
</tbody>
</table>

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Figure 2. The relation between basal metabolism and body weight in mammals, distinguishing sub-orders and classes (Blaxter, 1972).
then maintenance requirements expressed in terms of the metabolizable energy of food are

\[ M = \frac{B}{k_m} W^{3/4} \text{ or } M = a W^{3/4}, \quad (2) \]

where \( k_m \) is the efficiency with which metabolizable energy is used to replace the body constituents broken down during a fast.

**The Partition of Energy During Growth**

Brody, in his studies of energy requirements and efficiency, devised multiple regression methods in which maintenance was taken to be proportional to metabolic body size. These were some of the first systematic statistical studies to be undertaken of the relation between feed intake and production, and the form of the relationship he used was

\[ \text{feed/day} = b_o \times \text{metabolic size} + \sum b_i \text{(daily product, i.e., growth, milk, eggs)}. \quad (3) \]

For growth alone the relationship can be expressed more explicitly in the form of a differential equation:

\[ \alpha \frac{dW}{dt} + f(W) - \frac{dF}{dt} = 0. \quad (4) \]

Here \( W \) is body weight, \( F \) is cumulative feed intake expressed as metabolizable energy and \( t \) is time. The coefficient of the differential of weight, \( \alpha \), is the heat of combustion of unit gain, \( C \), divided by \( k_f \), the efficiency of utilization of feed energy above maintenance (Blaxter et al., 1982). This equation can be solved by substituting different functions to describe maintenance \( f(W) \) or rate of feed intake, \( dF/dt \), and many of these have been explored algebraically (see Blaxter, 1968; Blaxter et al., 1982). These explorations led to the following general conclusions, which are interesting.

1. Equilibrium weight, \( A \), is the ratio of rate of feed intake per unit time to the rate of metabolism per unit weight, the latter being the function relating maintenance metabolism to weight \( f(W) \) in the differential equation. Formally this is \((dF/dt)/(dM/dW)\).

2. For constant rate of feed intake and irrespective of the function used for maintenance, the solutions to the differential equation are of the following form:

\[ W = A (1 - \exp [-k (t - t*)]). \quad (5) \]

When feed intake is changing exponentially the solution approaches this form with time.

3. The rate of approach to equilibrium \([k \text{ in equation (5)}] \) is the rate of metabolism per unit weight divided by the coefficient of gain, i.e., \((dM/dW)/\alpha\).

The first finding allows estimation of mature weight from a knowledge of equilibrium feed intake and metabolic rate, for at equilibrium, rate of feed intake, in terms of metabolizable energy, is the same as rate of metabolism. Mature weight is thus dependent on nutrition, a matter we have all known for many years. The second finding is of interest because the resulting equations are of the same form as that which Brody used to describe growth after puberty in animals. The third conclusion is most interesting of all because it implies that the time constants of Brody’s growth equations for what he called the self-inhibiting phase of growth are simple functions of maintenance energy expenditure per unit weight.

In 1965, Taylor, following Günther and Guerra’s analysis (1955), showed statistically by analyzing the mass of growth equations given by Brody that the time constant \( \tau \) in them \((k = 1/\tau)\) were related to mature weight, \( A \), by the equations:

\[ \log \tau = .27 \log A + 2.0 \quad (6) \]

or

\[ \tau = 100 A^{.27} \text{ days}, \]

where \( A \) is mature body weight. Taylor also showed that the value of the constant \( (2.0) \) varied with the body temperature of the species—a conclusion that agrees with the observations in table 2.

Taylor’s results were empirical ones. The analysis of the relation of feed intake to weight change using the differential equation shows that his findings are consistent with metabolic considerations. I showed (Blaxter, 1968) that when the maintenance requirement was taken to vary with metabolic weight and rate of feed intake was constant, according to the equation

\[ \frac{C}{k_f} \frac{dW}{dt} + \frac{B}{k_m} W^n - Z = 0, \quad (7) \]

the rate at which body weight approached equilibrium weight was

\[ k = \frac{k_f B n W^{n-1}}{k_m C}. \quad (8) \]

If the power of weight to which metabolism was proportional was .73 (Brody’s original esti-
mate), then the relation between the time constant \( \tau \) and body weight was

\[
\tau = \frac{1}{k} = 111 \text{ W}^{0.27} \text{ days}.
\]

Shown in figure 3, taken from Blaxter (1968) is the agreement between Taylor’s statistically derived estimate of \( \tau \), that derived from overall metabolic considerations, and some of Brody’s original estimates of this constant taken from his book. The agreement between all three is remarkably close.

Brody, while he regarded the relationship he had found between basal metabolism and weight as empirical, nevertheless regarded his formulation of relationships between weight and time, his growth equations, as having the status of laws. The arguments given above show that the two are closely interrelated and that the constants of his growth equations, \( A = \) mature weight and \( k = \) the exponential rate of maturation, both involve the concept of metabolic size that arises from the basal metabolism equation.

Basic to this interrelationship is the contention that maintenance requirement of animals in the postpuberal phase of growth is proportional to body weight raised to the power \( \frac{3}{4} \) or \( .73 \). The experiments of Taylor and Young (1966, 1967, 1968) involved feeding cattle fixed amounts of feed for long periods of time and observing, among other things, the equilibrium weight attained. It was found that the amount of feed required to maintain equilibrium weight was directly proportional to the equilibrium weight maintained; that is maintenance requirements were directly proportional to weight. The line describing this has been called by Parks (1982) “the Taylor diagonal.” If, in equation (4), maintenance is stated to be directly proportional to body weight \( (M = aW) \), then the rate constant of growth is \( a/\alpha \), a constant irrespective of body weight. All animals would approach equilibrium weight at the same rate and the relationship in figure 3 would not apply as a generality to all species. Elsewhere, Armstrong and I (Armstrong and Blaxter, 1983) have criticized the experiment, pointing out that the body compositions of the animals given the smaller amounts of feed must have been grossly abnormal.

Parks (1970, 1982) has used a different approach to relate growth in time to feed intake. Commencing with the Spillman equation relating weight gain to feed intake, he generalized to show that in Brody’s growth equation the value of \( k \) could be regarded as the product of a constant \( B \) and the maximal rate of feed intake, \( C \).

\[
W(t) = A(1 - \exp \left[ - (BC)(t-t^*) \right]).
\]

Further development, in which the relation between feed intake and weight was regarded as an exponential relationship reaching towards a limit, resulted in a Gompertz function of time in which the measurements are mature weight, mature feed intake, initial feed intake and the constant \( B \) as above. This relationship describes the whole course of growth, which includes both Brody’s self-accelerating and self-inhibiting phases, and is an interesting approach. It does not have the simplicity of that given above, nor does it relate easily to the basal metabolism equation.

**Experimental Verification**

The theoretical relationship outlined above was developed some time ago and I have made two tests of it (Clapperton and Blaxter, 1965; Blaxter et al., 1982) and a further test is in progress. In the first test, calorimetric methods were used to estimate body energy retention when constant feed was given for periods of 10 mo and in the second, body composition was
determined by serial slaughter of animals fed ad libitum for periods of over 4 yr. In the latter instance, voluntary intake of feed showed a seasonal variation, but total consumption in 6-mo periods was invariant with body weight in the range of weight of individual sheep from 30 kg to in excess of 130 kg. Equilibrium weight was related to the mean six monthly feed intake and calorimetric measurements of metabolism, which showed that fasting metabolism varied with the .74 (rams) or .76 (wethers) power of weight. The composition of the gain in weight was found to be invariant with body weight, confirming the observations made by Searle et al. (1972).

These experimental verifications of the algebra, their concordance with Brody’s original findings on growth in different species and Taylor’s (1965) demonstration of the interrelation of mature body size and its rate of attainment all suggest that the differential equation relating body gain of energy to feed intake is meaningful and useful in explaining and integrating Brody’s two major findings—the relationship of metabolism to weight and the relation of weight to time, that is growth. That the differential equation is in fact a more elegant expression of the relationship that Brody had used to analyze animal efficiency in feed conversion illustrates once more how Brody had anticipated much. It is perhaps curious that he had not taken the analysis as far as he might have done.

**Growth at the Cellular Level**

The study of growth and bioenergetics, however, is not simply about broad species generalizations. Growth is much more than an increase in total body mass and demands analysis at a much deeper level of sophistication. Questions arise about how these broad, descriptive relationships relate to events at the organ, cellular and subcellular level, what are the controlling mechanisms and, highly relevant to animal husbandry, how they can be manipulated. The examples taken all relate to growth of muscle tissue because this is the tissue of major importance in animal production. The research described mostly stems from that which has been undertaken by my colleagues at the Rowett Institute in Scotland.

On an interspecies basis, muscle increases with a power of body weight greater than 1.0, paralleling that of the skeleton (Anderson et al., 1979). Its mass ranges from 25 to over 40% of total body mass. In mature animals, 90% of its protein is associated with the muscle fiber either as myofibrillar, or sarcoplasmic protein. The muscle cells are poor in nuclei, indeed connective tissue nuclei make up over 60% of the nuclei in muscle (Corring et al., 1982). The major proteins involved are collagen (type III), actin and myosin. In passing it should perhaps be remarked that two proteins, collagen and actomyosin account for well over 50% of the protein of the total body. Collagen alone accounts for 33%—a sobering thought in view of its abysmal nutritive value as food. It is indeed disturbing to think that animal scientists spend so much time and treasure in producing animal protein when a third of it has been recognized since the times of the Paris soup kitchens to be of very poor nutritive worth.

**Energy Expenditure and Protein Metabolism**

One can commence by asking questions about the origin of the energy expended in metabolism and how this relates to protein growth and particularly to muscle. Using the conventional framework of thermodynamics one can distinguish heat and work. The work done by the body consists of three main components, pressure-volume work such as that of the heart, lung and gut in movement, work done in synthesizing new macromolecules and work done in maintaining electro-chemical gradients across membranes of cells and organelles. This work eventually appears as heat; pressure-volume work is dissipated in fluid friction, new chemical bonds are broken in turnover and ion gradients dissipated through diffusion. These three components of work can be estimated—from pressure-volume integrals, from rates of turnover and the free energies of the bonds formed and from ion fluxes. Their sum is only a small proportion of the total energy expended by the resting animal—about 3% for mechanical work, slightly more for turnover of protein, lipid and carbohydrate and up to about 15% for the maintenance of gradients. The major component of energy expenditure arises from the low efficiencies with which ATP is formed from ADP during the oxidation of food or body tissue and the considerable amount of ATP or its equivalent that is needed to effect synthesis of new chemical bonds, notably peptide bonds.

It has been known for some time that there is a direct relationship between the rate of synthesis of protein and the metabolic rate of animals of different species (Waterlow, 1968).
adult animals the amount of protein synthesized per day is about 15 g/kg of body weight raised to the power .75 (Reeds and Harris, 1981) and the relationship between metabolic rate and rate of synthesis is given by the following relationship:

$$H(kJ/d) = 20 \times \text{rate of protein synthesis (g/d)}.$$  

This general relationship between energy metabolism and protein synthesis is further illustrated in figure 4 (Reeds and Harris, 1981). Rates of protein synthesis are higher in young animals when metabolic rate is high and are lower in man than in other animals, a conclusion to be expected because the rate of protein accretion is lower in man than in most mammals.

The above estimates of rate of protein synthesis refer to the whole body. Studies in several laboratories, summarized by Millward et al. (1981) and Lobley (1978), show that rates differ significantly from tissue to tissue. The results obtained by Lobley are shown in table 3. They show that the intestine had the highest rate of synthesis of protein, higher than muscle despite the considerable mass of the musculature. The rates per g of protein (the fractional synthesis rates) expressed relative to muscle were muscle 1, skin 4, liver 15 and intestine 30. These relative contributions to protein synthesis differ from the relative contributions of tissues and organs to energy expenditure as first studied by Barcroft (1908) and later by Field et al. (1939) and Martin and Fuhrman (1955). These studies showed that muscle accounted for the largest proportion (about 30%) of the oxygen consumption of the whole animal and that the intestine and skin each contributed less than 10% of the total. Both intestinal and skin epithelial cells regenerate, that is, turnover involves loss of the whole cell, but this hardly accounts for the difference between protein synthesis rates and energy expenditure. While protein synthesis may account for a fairly constant proportion of total metabolism, it does not appear to do so at tissue level.

Despite this difficulty, which may be due to the considerable technical problems involved in measurement of tissue metabolism under conditions that simulate those operating in the intact animal, there is no doubt that growth is concerned with the net accretion of protein, that is, the balance between synthesis and degradation of protein, and that the former is energetically expensive.

### Collagen Synthesis

The complexity of collagen synthesis illustrates how difficult it is to estimate theoretically the energy cost of protein synthesis (see Robins, 1981). The pre-mRNA is about eight times larger than the final mRNA due to the presence of introns in the genomic DNA, entailing about 50 splicing steps. The translation of the mRNA results in the formation of pre-procollagen chains. Signal peptides are then removed to give the procollagen chains, which then are subject to hydroxylation of some of their prolines and lysines. Glycosylation of some of the hydroxylysines occurs concurrently when disulphide bridges are formed to result in a helical structure that is then secreted by the cell. The
N- and C-terminal peptides of the central helical region are then excised, oxidative deamination of particular lysine or hydroxyproline residues takes place, and new cross links are formed. Further "maturation" of the cross linking then occurs to give mature, insoluble collagen fibers. It is obvious that there are many steps in the biosynthesis of collagen that are energy-demanding. Not only do the peptide bonds of signal and telopeptides have to be made, but energy is expended in secretion of the procollagen by the microtubular system and at various post-translational stages when the procollagen is modified. The cost of synthesis is clearly more than that computed on the assumption that four ATP or their equivalent are required to make each peptide bond of mature collagen. The synthesis of the myofibrillar proteins equally involves post-translational modification in the methylation of histidine residues.

Growth of Muscle

Studies of the changes in net protein accretion (from N retention studies) show that these are not solely due to a change in the rate of synthesis or in the rate of degradation. In many instances enhanced growth is associated with an increase in both, as illustrated in figure 5 (Reeds and Fuller, 1983). In others, net accretion is largely achieved by a decrease in degradation, which appears to be the case in growth promotion by anabolic steroids (Vernon and Buttery, 1976; Millward et al., 1981), though even here there is a small increase in rate of synthesis. Again thyroxine augments both synthesis and breakdown of protein.

Studies of isolated muscle show that stretching induces an increase in synthesis (Palmer et al., 1981). This is not due to changes in membrane electrical activity but to an activation of the plasma membrane phospholipase A2. There is now considerable evidence that arachidonic acid released from phosphatidylethanolamine gives rise to a chain of events leading to synthesis of the prostaglandins and thromboxanes. It has been found that prostaglandin F$_{2\alpha}$ (PGF$_{2\alpha}$) increases protein synthesis (Smith et al., 1983) while prostaglandin E$_{2}$ increases protein degradation (Rodemann et al., 1982; Rodemann and Goldberg, 1982). Prostaglandin F$_{2\alpha}$ is also closely involved in the stimulation of protein synthesis by insulin (Reeds and Palmer, 1984). As Reeds and Palmer (1984) have remarked, "a simultaneous change in PGF$_{2\alpha}$ and PGE$_{2}$ concentrations could explain the dual response of protein synthesis and degradation," which characterizes the majority of studies of altered muscle growth, and that "alterations in the activity of phospholipase A2 may therefore be the common link between a number of different changes in the extracellular environment and the ability to synthesize and degrade proteins."

In the above studies, protein synthesis has been measured in the muscle as a whole, including proteins of the myofibril, the sarcoplasm and those associated with the plasma membrane. In liver cells the turnover rates of individual proteins vary considerably. In muscle, studies of the individual proteins suggest that their rates of synthesis are all very similar. Thus Lobley and Lovie (1979) isolated actin and myosin from rabbits given radioactive tyrosine. The turnover of these, of other fibrillar proteins and of the collagen types were all closely similar to that of the muscle as a whole. These results imply that, unlike liver, muscle proteins must all have the same synthesis rates and, by implication, must also have the same degradation rate. The concept that a muscle consists of a relatively inert collagen matrix surrounding a metabolically active series of fibers can hardly be correct. Questions thus arise about how muscle does in fact grow. Many studies have shown that the number of muscle fibers in most mammals is fixed at birth, a recent example being that of Timson (1982). Replacement of fibers through the so-called satellite cells could be responsible for the phenomenon of continuous and variable rates of synthesis and degradation.

![Figure 5](image_url). The relation between protein synthesis (●) and protein breakdown (○) in relation to the level of total energy intake. Both the ordinate and the abscissa are scaled so that maintenance values = 1.0 (Reeds and Fuller, 1983).
Little is known, however, about how protein turnover is accommodated at the level of the muscle cell. Lysosomal systems are not of any importance in muscle and cannot be induced, while apoptosis has not to my knowledge been observed in muscle.

I have dealt, in this paper, with aspects of growth and bioenergetics that have included, at one extreme, isolated events at the cellular level and their complex interactions and, at the other extreme, growth of the mass of the bodies of all mammalian species in relation to generalities about their metabolism. In each instance an integration is possible. Protein synthesis and degradation can be explained by an integrating hypothesis relating to membrane function; energy expenditure in protein synthesis can be generalized with respect to its contribution to total animal metabolism. Consideration of rates of synthesis of the separate proteins that make up the muscle cell and its associated connective tissue lead to a concept of the muscle cell as a dynamic entity, possibly exhibiting life-time kinetics under nutritional and endocrine control, rather than as a collection of different enzymes and structural components all turning over at different rates. The broadest generalization of all shows that growth rate is a simple function of the minimal metabolism of the species.

What perhaps this illustrates is that growth and bioenergetic phenomena can be studied at many different levels—those that are concerned with animals as wholes, with animals as organ or cellular systems or with animals as biochemical and biophysical systems. At each and every level of conceptual approach an integration is possible. Equally, findings and hypotheses that are formulated and found applicable at one level must be related to those at some higher or lower one if they are to be of value as real explanations of biological phenomena. Any final theory of growth should enable complete and overall integration to be accomplished; this however is well beyond any of our capabilities at the present time. We can however take heart, for Samuel Brody, although he accomplished so much in simplifying complex interrelationships and in converting rather dull facts into a coherence, realized full well that such goals lay far in the future.

**Literature Cited**


