

Ontogeny of energetic relationships and potential effects of tissue turnover: a comparative modeling study on lake trout

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Abstract: Tissue turnover is endogenous energy flow and may play a regulatory role in the metabolic system of an organism. We developed a general growth model addressing potential effect of tissue turnover on energy acquisition and partitioning. We applied the model to estimate energy assimilation of lake trout (*Salvelinus namaycush*) in Lake Michigan and compared the model with a commonly used complex model. Both models are expansions of the Pütter – von Bertalanffy growth model. The new model suggested a consistent decreasing trend in energy net conversion efficiency (NCE) for somatic growth versus body energy. The complex model suggested that NCE is relatively stable in early ages and decreases slowly in comparison with the pattern suggested by the new model. The new model estimated higher specific assimilation rate and NCE for gonadal growth than for somatic growth of mature fish. The complex model did not distinguish gonadal growth from somatic growth. For a lake trout growing from the start of age-1 to the end of age-10, our new model suggested a total energy assimilation 25% higher than the complex model. The above comparisons support the inference that tissue turnover is an important bioenergetic component. Inclusion of tissue turnover in bioenergetic modeling analyses may be critical for studying the linkages among individual growth, reproduction, and population dynamics.

Résumé : Le renouvellement tissulaire constitue un flux d'énergie endogène et peut jouer un rôle régulateur dans le métabolisme d'un organisme. Nous avons élaboré un modèle général de croissance traitant de l'effet potentiel du renouvellement tissulaire sur l'acquisition et la répartition de l'énergie. Nous avons appliqué le modèle au calcul de l'assimilation de l'énergie chez le touladi (*Salvelinus namaycush*) du lac Michigan, et nous l'avons comparé à un modèle complexe couramment employé. Ces deux modèles sont des adaptations du modèle de croissance de Pütter – von Bertalanffy. Le nouveau modèle met en évidence une tendance constante à la baisse du rendement net de conversion de l'énergie (RNC) quand on considère la croissance somatique par rapport à l'énergie corporelle. Le modèle complexe fait ressortir le fait que le RNC est relativement stable dans les premières phases puis baisse lentement si on le compare au régime indiqué par le nouveau modèle. Ce dernier donne une estimation plus élevée du taux d'assimilation spécifique et du RNC pour la croissance gonadique que pour la croissance somatique des poissons à maturité. Le modèle complexe ne faisait pas de différence entre la croissance gonadique et la croissance somatique. Pour un touladi dont on suit la croissance du début de l'âge-1 jusqu'à la fin de l'âge-10, notre nouveau modèle suggère que l'assimilation totale de l'énergie est de 25 % supérieure aux résultats du modèle complexe. Les comparaisons ci-dessus viennent confirmer que le renouvellement tissulaire est une composante bioénergétique importante. L'intégration du renouvellement tissulaire dans les analyses de modélisation pourrait jouer un rôle crucial dans l'étude des liens entre la croissance des individus, la reproduction et la dynamique des populations.

[Traduit par la Rédaction]

Introduction

There have been two types of models for studying the energetic basis of fish growth (Adams and Breck 1990; Ney 1990; Gerking 1994). One uses data on the daily cycle of food mass in fish stomachs and evacuation rates (Bajkov

1935; Eggers 1977; Elliott and Persson 1978). The other is based on fish energy balance (Clarke et al. 1946; Winberg 1956). The second approach includes a variety of energetic models, and the modelers attempt to study mechanisms that regulate organismal growth (Ivlev 1945; Paloheimo and Dickie 1965; Gerking 1994). The empirical basis for those models had been summarized by the end of the 1970s (Brett and Groves 1979), while a biological understanding still required more advanced studies (Ricker 1979).

One extreme of the second approach is to use simple growth models and explore their energetic implications (von Bertalanffy 1957; Pauly 1986), while another extreme is to

Received March 25, 1997. Accepted July 24, 1998.
J13929

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model complex energetic components with as much detail as needed for a particular application (Kitchell et al. 1977; Stewart et al. 1983; From and Rasmussen 1984; Stewart and Binkowski 1986; Brandt et al. 1992). Between those extremes, there are many studies that either aggregate bioenergetic components (Mann 1965; Majkowski and Hearn 1984) or combine a simple growth model with radiotracer techniques (Forseth et al. 1992; Rowan and Rasmussen 1996). Comparing various energetic models can improve our understanding of organismal growth and food web dynamics (Boisclair and Leggett 1989). Since Ursin's (1979) work, however, few studies have attempted to bridge the two extremes and compare models by explicitly analyzing both model results and model structure.

Over the past 20 years, there have been advances in experimental studies about the relationships between protein turnover and energetics (Houlihan 1991; Hawkins 1991). The implications of those experimental studies have not been integrated into either simple or complex energetic models. Protein turnover is intracellular protein degradation to amino acids and protein renewal (Hawkins 1991). A related phenomena is seasonal dynamics of body energy density, which in turn, is a key component of reproduction processes and seasonal mortality patterns (Flath and Diana 1985; Diana and Salz 1990). In early life stages, protein turnover related to energetic constraints also influences fish mortality and recruitment processes (Blaxter and Hempel 1963; Miller et al. 1988). Although the exact linkage between protein turnover and energy turnover is not clear yet, we anticipate that total tissue turnover is a key component for extending commonly used energetic models (e.g., Kitchell et al. 1977; Stewart et al. 1983) to study fish life histories and population dynamics.

In this paper, our objective is to develop an energetics-based growth model that includes effects of tissue turnover on the specific rate of energy assimilation and energy net conversion efficiency (NCE). Such a development allows for addressing linkages and differences between somatic and gonadal growth. To maintain a focus on available empirical observations and address integrative feedback mechanisms, we develop our model by combining the simple models of Pütter – von Bertalanffy (Pütter 1920; von Bertalanffy 1957) and Pauly (1986) with a more general structure. To evaluate the advantages of addressing the potential effects of tissue turnover, we apply our model to lake trout (*Salvelinus namaycush*) in Lake Michigan and compare our model with a commonly used complex model (Stewart et al. 1983).

Complexity of fish energetics and the importance of tissue turnover

Complex fish energetic models are reinterpretations and expansions of the simple Pütter – von Bertalanffy model (Ursin 1979; From and Rasmussen 1984):

$$\begin{aligned} \text{Growth} &= \text{assimilation} - \text{respiration} \\ &= \text{consumption} - \text{egestion} - \text{excretion} - \text{respiration} \end{aligned}$$

Those expansions assume that relationships among various energetic components are simply additive. When more and more details have been included, the additive expansions re-

veal their current limitations for expressing integrative regulatory mechanisms. Recent debates about model structure and applications have focused on submodels of respiration, including basal respiration, activity-related respiration, and specific dynamic action (SDA) (Boisclair and Leggett 1989). There are direct correlations among consumption, growth, and activity-related respiration (Kerr 1982, Boisclair 1992; Björnsson 1993; Madon and Culver 1993). Specific dynamic action is primarily related to growth, while processing food and eliminating excreta only account for a very small part of SDA (Ashworth 1969; Tandler and Beamish 1979; Jobling 1983; Brown and Cameron 1991a, 1991b). Statistically, SDA is also correlated with food intake (Beamish 1974), food composition (Beamish and Trippel 1990), body mass (Beamish and Trippel 1990), and even activity (Krohn et al. 1997).

The foregoing multiple correlations support an inference that total respiration is quantitatively not divisible (Jobling 1981, 1983). Experiments for estimating SDA used a condition in which activity-related respiration was a constant and would not be affected by feeding (Beamish 1974; Beamish and Trippel 1990). An estimated relation between activity-related respiration and consumption, however, had to assume that SDA was a constant proportion of consumption and was not related to feeding-induced activity (Kerr 1982). Experiments for estimating energetic costs of swimming excluded effects of both feeding-induced activities and SDA (Brett 1964; Rao 1968; Tytler 1969; Beamish 1970; Muir and Niimi 1972; Stewart et al. 1983); otherwise, the relationships of respiration to swimming speed would be different among periods of pre-feeding, feeding, and post-feeding (Durbin et al. 1981). Observations of the correlation between swimming and consumption did not consider the existence of SDA but had to use the results of the above-mentioned nonfeeding experiments to calculate swimming energetic costs (Boisclair 1992; Björnsson 1993). Even the concepts of maintenance respiration and the cost of growth have been often interpreted with ambiguities. Maintenance respiration was related to rebuilding of dead tissues or metabolic substance (Gerking 1962), and the cost of growth has been calculated as total respiration minus maintenance respiration. Recent studies indicated that the specific rate of tissue turnover increases with swimming activity and the specific rate of growth (Houlihan and Laurent 1987; Houlihan et al. 1988). Thus, if maintenance is an energetic component of a growth process, it must be larger than the basal respiration. Commonly used fish energetic models have adapted to our current incomplete understanding of the biological complexity. The estimation of activity-related respiration is independent of consumption, and the value of SDA is simply calculated as a proportion of absorbed exogenous energy (Kitchell et al. 1977; Stewart et al. 1983).

Tissue turnover is an important energetic component, but the above energy balance does not give it an explicit treatment. Von Bertalanffy (1957) explained growth as the difference between “building up” and “breaking down.” His meaning was straightforward and did not explicitly address energy cost for building up and energy loss due to breaking down. The above reinterpretations and expansions of the Pütter – von Bertalanffy model actually followed Winberg's (1956) energy balance but apparently ignored details of tis-

sue turnover that were covered by von Bertalanffy's (1957) original concept. In Gerking's (1962) study, when a bluegill sunfish (*Lepomis macrochirus*) grew from 17.5 g to 34.0 g, total protein synthesis was 7.87 g, protein turnover (replacement) was 5.06 g, and net protein growth was only 2.81 g. Based on small animal experiments, a recent general estimate is that 1 g of protein retention requires 2–4 g of protein synthesis (Waterlow 1995).

When food is sufficient and an organism is rapidly growing, endogenous excretion of nitrogen may be negligible (Hawkins 1985; Jayaram and Beamish 1992). If protein turnover is at least one to three times protein retention, breakdown materials must be recycled. Then, the net effect of tissue turnover is only an increase of respiration. Unfortunately, there have been no experiments that attempted to separate this increase from, or partition it into, SDA and activity-related respiration. There are seasonal dynamics of body energy density, and there is energy or protein loss due to reproduction. Thus, endogenous excretion is not negligible in natural conditions and materials from tissue turnover are not completely recycled, particularly for large-scale measurements such as yearly growth and energy assimilation. We cannot, however, add tissue turnover as a separate term into an energy balance unless future studies can explicitly quantify two components of the net effects of tissue turnover. One is material from tissue turnover that has not been recycled, and another is a part of respiration, which may or may not be covered by current formulations of SDA or activity-related respiration.

An alternative approach to address the above complexity is to properly aggregate detailed energetic components (Ney 1990). The basis for such an aggregation must be a reconsideration of energy or mass balance, because tissue turnover is endogenous and partially recycled flux inside the system boundary. Even when organisms increase their body mass with sufficient food, at least protein turnover is still occurring, and the specific rate of protein turnover is positively related to the specific rate of protein growth (Houlihan et al. 1988).

The positive relation between growth and tissue turnover indicates that endogenous energy flow may play a regulatory role as an organism adapts to an given environment or responds to environmental changes. In a given environmental setting, efficient organisms have a low ratio of protein turnover to protein synthesis and a high specific rate of growth (Hawkins et al. 1986; McCarthy et al. 1994). That is because of either relatively low basal metabolism or relatively slow increases in the specific rate of tissue turnover as specific growth rate increases. As body mass increases, the ratio of protein turnover to protein synthesis increases and the specific rate of growth decreases (Goldspink and Kelly 1984, Tables 1 and 2; Houlihan et al. 1986, Tables 2 and 3; Houlihan et al. 1988, Fig. 4). When body mass is close to its asymptotic value and the specific growth rate equals or is close to zero, tissue turnover will account for most of tissue synthesis.

Energetic basis of the simple model by Pütter – von Bertalanffy and Pauly

Before we address the above regulatory mechanism using

an aggregated simple model, we need to provide further clarification of fundamental concepts. The basic equation of Pauly's (1986) model for estimating consumption is as follows:

$$(1) \quad GCE = 1 - (W/W_a)^\theta$$

where GCE is gross conversion efficiency (growth/consumption), W is body mass, W_a is the asymptotic body mass, and θ is a nondimensional constant. When an increase in body mass (ΔW) has been measured, we may estimate consumption (g or J) in the growth period ($\Delta C/\Delta t$) using

$$(2) \quad \Delta C/\Delta t = \Delta W/GCE = \Delta W/(1 - (W/W_a)^\theta)$$

Here, we do not need to address the method for estimating θ value, because Pauly's model (eq. 1) is identical to the Pütter – von Bertalanffy model (Silvert and Pauly 1987):

$$(3) \quad \Delta W/\Delta t = pW^M - qW^N$$

where pW^M refers to consumption rate ($\Delta C/\Delta t$), qW^N refers to the sum of nongrowth components in the consumption rate, p and M are regression parameters, $q = (p/W_a^\theta)$, and $N = M + \theta$. In the following analysis, we use the specific rate of growth (dW/Wdt), so eq. 3 is rewritten as

$$(4) \quad dW/Wdt = pW^m - qW^n$$

where $m = M - 1$ and $n = N - 1$. Based on eq. 4, Pauly's model (eq. 1) can be written in a more general form:

$$(5) \quad GCE = 1 - xW^y$$

where $x = q/p$, and $y = (n - m)$. Usually, $n > m$ and $y > 0$, so GCE decreases with increasing body mass. Reiss (1989) reviewed the theoretical basis and empirical evidence for eq. 5, but there are still ambiguities in eq. 5 that deserve clarification.

Von Bertalanffy (1957) considered growth to be the difference between "anabolism" (pW^m) and "catabolism" (qW^n). Subsequent studies have interpreted growth in various ways. One is the difference between consumption and all of nongrowth components in consumption (Reiss 1989), which gives Ivlev's (1945) growth efficiency $k1$ or GCE. The second interpretation is the difference between assimilation and respiration (Ursin 1979), which may relate to Ivlev's growth efficiency $k2$ or net conversion efficiency (NCE = growth/assimilation, where assimilation = consumption – egestion – excretion). The third interpretation for growth is the difference between tissue synthesis and tissue turnover (Houlihan et al. 1992), so we have Ivlev's growth efficiency $k3$ (growth/production, where production = assimilation + tissue turnover – respiration and growth = production – tissue turnover).

Ivlev (1945) introduced the important definition of production as tissue generation regardless of subsequent fate (i.e., production is non-negative), but he did not make his $k3$ concept clear (Winberg 1956; see also comments by Ricker in the 1996 English translation of Ivlev's original paper). After Ricker's (1979) extensive review, few studies have attempted to clarify energetic interpretations of growth models. In analyzing individual growth and related energy demand, the important concept of non-negative production has never been used. Consequently, von Bertalanffy's self-

regulation ($k3$ relationship) and Winberg's energy balance ($k2$ relationship) have been incorrectly used as interchangeable models. They have been expressed by the same equation (eq. 4), and their differences and linkages have been confounded. Without applying the concept of non-negative production, Winberg's energy balance may not be complete. Without a complete energy balance, it also may be difficult to understand von Bertalanffy's organismal self-regulation.

The first of the above interpretations for growth (eq. 4) made an aggregation of egestion, excretion, and respiration. Such an aggregation is useful for evaluating trophic interactions but may confound self-regulation of an organism. A fish may increase body length as it loses its body mass. A fish also may produce gonad mass using its somatic energy. Both assimilation and endogenous energy utilization are energy flows in the metabolic system of an organism, while some parts of consumption, such as egestion and excretion, are not in the metabolic system. The second of the above interpretations for growth may not encompass the complete meaning of Ivlev's $k2$ efficiency. It regards the residual between assimilation and growth as total respiration, but that residual also includes material for tissue replacement (Pandia 1967). The third of the above interpretations ($k3 = \text{growth/production}$) does not provide a basis for estimating exogenous energy demand. Production may rely on either or both exogenous and endogenous materials.

To study mechanisms for regulating organismal growth as well as reproduction, it is important to clearly set up the system boundary, so eq. 5 (and eq. 1) may not be a correct reinterpretation of the Pütter – von Bertalanffy growth model. To make a correction, a necessary step is to extend Winberg's (1956) energy balance as follows:

$$(6) \quad dW/Wdt = A - R - \beta(\tau) = \alpha(dC/Wdt) - R - \beta(\tau)$$

where A is specific assimilation rate ($\text{g}\cdot\text{g}^{-1}\cdot\text{t}^{-1}$ or $\text{J}\cdot\text{J}^{-1}\cdot\text{t}^{-1}$, where t is unit time), R is specific respiration rate ($\text{g}\cdot\text{g}^{-1}\cdot\text{t}^{-1}$ or $\text{J}\cdot\text{J}^{-1}\cdot\text{t}^{-1}$), τ is specific rate of tissue turnover ($\text{g}\cdot\text{g}^{-1}\cdot\text{t}^{-1}$ or $\text{J}\cdot\text{J}^{-1}\cdot\text{t}^{-1}$), α is non-dimensional assimilation efficiency (assimilation/consumption), and β is a replacement coefficient or the proportion of tissue turnover that is not recycled (endogenous excretion). A similar analysis can be seen in Jobling (1985). Unfortunately, he did not have information about the positive relationship between tissue turnover and growth. He followed von Bertalanffy in regarding tissue breakdown as a constant proportion of body mass.

If τ had been evaluated in the context of a detailed energy balance study, we would be able to build on the complex model (Kitchell et al. 1977; Stewart et al. 1983) to analyze relationships of τ with other energy components and to evaluate its effects on energy balance. In the absence of such studies, we may aggregate R and $\beta(\tau)$ and modify eqs. 5 and 1 as follows:

$$(7) \quad \text{NCE} = 1 - aW^b$$

Although we did not change the form of equations (eqs. 5 and 7), we clarified the biological meaning of mathematical terms. Such clarifications allow us to consider a growth process including both somatic and gonadal growth. It may appear reasonable to aggregate somatic and gonadal growth as total "production," but such an aggregation needs to be done

with caution. Tissue replacement ($\beta(\tau)$) is also a part of production but would be aggregated with respiration. Gonadal growth is at least partially turnover from somatic tissue to gonadal tissue but would be aggregated with somatic growth. After sexual maturity, female fish can increase gonadal percentage of total body mass from 1–3% to 10–70% in a few months, showing an exponential growth of gonadal mass (e.g., Ware and Tanasichuk 1989; Hop et al. 1995). The differences between gonadal growth and somatic growth, and their relative metabolic costs, should not be confounded.

A proper aggregation may be to consider reproduction as a part of tissue turnover. Such an aggregation would allow us to estimate energy demand based on somatic growth patterns (Pauly 1986). The parameter a in eq. 7 is related to the maximum NCE. If we use body energy rather than body mass, the maximum NCE is about 0.96 (Calow 1977; Brafield and Llewellyn 1982), so the parameter a has a theoretical value of about 0.04. At the theoretical asymptotic size, somatic NCE is zero, although gonadal growth and tissue turnover are not zero. Setting NCE equal to zero at the asymptotic body energy, we can estimate the parameter b for eq. 7 and calculate an NCE curve versus body energy (see detailed assumptions in following application section). Using that NCE curve, we must be aware that reproduction energy has been aggregated with tissue turnover for mature fish and has not been quantified explicitly.

With either of the foregoing schemes of component aggregation, we cannot use eq. 7 to address the linkages and differences between somatic and gonadal growth. The second scheme of aggregation, however, has much less conceptual ambiguity than the first one. Eventually, gonadal tissue will be lost or reabsorbed, so it is a special case of tissue turnover. From such a consideration, we may take the Pütter – von Bertalanffy model (eqs. 4 and 7) as the critical first step to developing a general growth model.

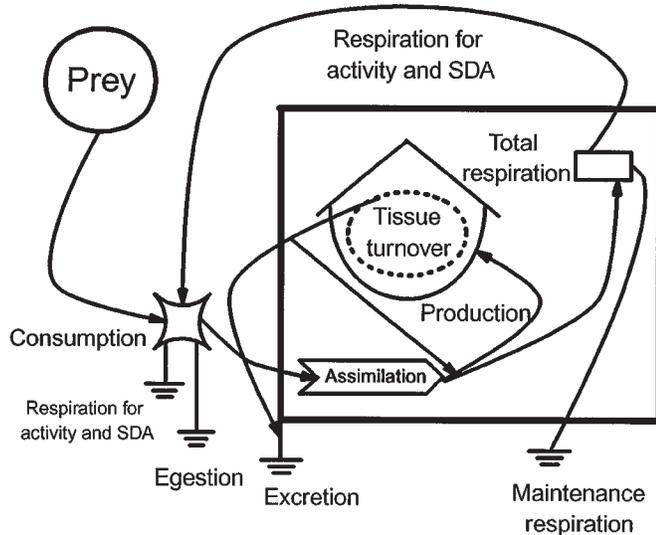
Towards a general model for growth and reproduction

A growth process includes both energy demand–supply relationships (Winberg 1956) and the self-regulation of an organism (von Bertalanffy 1957). What has been missing in energetics-based growth models is the relationship between endogenous and exogenous flows or the regulatory role of endogenous energy flow (Fig. 1). In subsequent paragraphs, we combine the Pütter – von Bertalanffy model into a general structure to address the above relationships.

At every growth step, there is a given G , a given τ , and a given NCE. These variables are all allometric functions of total body energy. At a given growth step, if an organism could grow from the existing body energy (W) to its asymptotic body energy (W_a), it would require a certain quantity of assimilation ($(W_a - W)/\text{NCE}$) and would have a certain quantity of tissue turnover, $t(\tau)W$, where t is the time required for W to produce $(W_a - W)$ with its given G . Both $(W_a - W)/\text{NCE}$ and $t(\tau)W$ are potential energy flows in the metabolic system under that conditional growth. The former would be exogenous, and the latter would be endogenous.

In a growth process, negative feedbacks of the increasing total body energy are the ontogenetic changes in the foregoing energetic relationships. When total body energy in-

Fig. 1. Energy flows in the metabolic system of an organism, indicating production as a non-negative concept, where assimilation = consumption – egestion – excretion; production = assimilation + tissue turnover – respiration; and growth = production – tissue turnover.



creases, the ratio of τ/G increases, NCE decreases (eq. 7), and the metabolic demand $((W_a - W)/NCE)$ for producing $(W_a - W)$ increases. In total respiration, the proportion of maintenance respiration including energy cost for rebuilding broken-down tissues will increase as body energy increases, and the proportion of useful activity for energy acquisition will decrease (Fig. 1). Thus, the organism must decrease its specific rate of assimilation as its total body energy increases.

The above observations and concepts can be expressed as follows:

$$(8) \quad G = (NCE)A$$

$$(9) \quad A = A_m(W_a - W)/[(W_a - W)/NCE + t(\tau)W]$$

where G is specific growth rate, NCE follows eq. 7, A is specific assimilation rate, and A_m is the maximum specific assimilation rate for an organism in a given environment. W_a is the body energy at infinite time when NCE is zero. When NCE equals 1 and τ equals zero, A is A_m . Both W_a and A_m are theoretical. Their empirical foundations are the above-mentioned negative allometric functions of total body energy, including NCE, specific growth rate, and specific assimilation rate. In comparison with the Monod model (Monod 1942, 1950), eq. 9 uses energetic components in a metabolic system, rather than environmental factors such as resource abundance. In comparison with the logistic model (Verhulst 1838; Pearl and Reed 1920), eq. 9 expresses potential exogenous and endogenous metabolic demand for producing $(W_a - W)$, but does not regard $(W_a - W)$ as “unutilized environmental capability.”

Because t actually equals $(W_a - W)/GW$, eq. 9 can be rewritten as

$$(10) \quad A = A_m / ((1/NCE) + \tau/G)$$

Rearranging eq. 10 with eq. 8, we have

$$(11) \quad A = A_m(NCE) - \tau$$

From eq. 11, we can provide further explanation of our model. There are positive empirical relationships among NCE, τ , G , and A (Houlihan and Laurent 1987; Houlihan et al. 1988, 1992). When obtaining exogenous energy is the purpose of an organism and its endogenous energy release is a necessary input or an unavoidable byproduct of that action, the above positive relationships are understandable. There must be a final constraint, however, on total energy flow in the metabolic system. That constraint is the potential tissue rebuilding ability ($A_m(NCE)$) of an organism because absorption of exogenous energy and mobilization of endogenous energy are similar processes. In other words, a fish could maximize its tissue mobilization according to its potential rebuilding ability, but part of its effort is always directed to acquisition of exogenous energy (eq. 11; Fig. 1). Notice that, like W_a , A_m is a theoretical potential of a fish in a given environment, rather than an observed maximum specific flux in the metabolic system. As total body energy increases, the ratio of τ/G increases (i.e., $k3$ decreases), so NCE must decrease (eq. 7). Then, for a given A_m , there will be correlated decreases in specific assimilation rate (A ; eq. 10), specific growth rate (G ; eq. 8), and the specific rate of tissue turnover (τ ; eq. 11).

After sexual maturity, there will be gonadal growth:

$$(12) \quad G_g = A_g(NCE_g)$$

where NCE_g is energy net conversion efficiency for gonadal growth, G_g is gonadal specific growth rate, and A_g is gonadal specific assimilation rate. In the relatively constant condition inside the body, gonads grow exponentially (Eschmeyer 1955; Ware and Tanasichuk 1989; Hop et al. 1995), so G_g , A_g , and NCE_g are approximately constant, at least for a fish during a given reproductive cycle. In a reproductive season, the ratio of gonadal to total body energy increases, so A_g and NCE_g must be larger than A and NCE, respectively.

Growth potential should be the same for both somatic and gonadal tissue because there is no fundamental difference between them. They have different specific growth rates because endogenous energy demand for gonadal growth draws on somatic tissue. Under the conditions where gonadal tissue is rapidly growing, the reabsorption of gonadal tissue is negligible. In contrast, the specific rate of somatic turnover will increase after sexual maturity because of the added turnover demands related to gonadal growth.

To implement the above relationships for mature fishes, we need to replace $A_m(NCE)$ with $A_m(NCE_g)$ in eq. 11:

$$(11a) \quad A = A_m(NCE_g) - \tau$$

and express gonadal specific assimilation rate as a special case of eq. 11a:

$$(13) \quad A_g = A_m(NCE_g)$$

Notice that A is specific assimilation rate for the whole body, and NCE in eqs. 7 and 8 treats energy for reproduction as a part of the “cost” component. Independently, eqs. 12 and 13 treat gonadal tissue as a subsystem. That subsystem can simply use somatic energy whenever necessary, so $A_g W_g$

can be a part of AW or a part of τW , where W_g is gonadal energy (J). If we use NCE rather than NCE_g in eq. 13 and do not distinguish eqs. 11 and 11a, simulated gonadal growth will be too slow for old age-classes.

In addition to the direct energy demand for gonadal growth (eqs. 12 and 13), the effect of gonadal growth on somatic tissue is also expressed in eq. 11a. In this study, we can estimate energy assimilation based on observed growth using eq. 7. Then, with a consistent decreasing trend of specific assimilation rate versus total body energy, the difference between eqs. 11 and 11a implies a relative increase in the specific rate of tissue turnover after sexual maturity, rather than increases in specific rates of assimilation or growth. We suggest that the value of NCE_g results from adaptation of a fish to a given environment. A high NCE_g can lead to high reproductive effort (eqs. 12 and 13) but that usually involves high mortality risk (Gunderson 1997). Our model suggests that natural mortality related to energetic constraints can be quantified based on the specific rate of tissue turnover. Those additional contexts, however, will not be presented in this paper.

Some fish do not have exogenous assimilation during reproduction. Their reproduction fully relies on turnover of somatic tissue, so $A_g = \tau$ when A equals zero (eqs. 11a and 13). Somatic tissue is larger than gonadal tissue, so somatic tissue turnover will provide energy for respiration. The equivalence of A_g and τ reflects our inference that absorption of energy and mobilization of body energy are similar processes. The average specific flux in somatic tissue is the same as in gonadal tissue, but energy partitioning within those two compartments differs. The former includes energy transformation to gonadal tissue, necessary rebuilding of somatic tissue or metabolic substances, and somatic respiration. The latter only includes gonadal growth and respiration.

If resource abundance is very low and somatic storage is very poor, a fish may fail to reproduce even after sexual maturity (Trippel and Harvey 1989; Henderson et al. 1996), or a considerable proportion of an ovary may fail to ripen. Those phenomena result from temporal, spatial, and individual variations in A_m and NCE_g . After reproductive season or when a fish stops gonadal growth, gonadal tissue can be considered a form of somatic storage; thus, its reabsorption may follow eq. 11. Those details are also beyond the scope of this paper (but see a slightly different treatment by Van Winkle et al. 1997).

Energy acquisition by lake trout in Lake Michigan: comparisons between two models

In this section, we apply eqs. 7–13 to estimate energy assimilation of a lake trout in Lake Michigan and compare our results with those from a more complex model (Stewart et al. 1983). Both models are expansions of the Pütter – von Bertalanffy model. To improve our biological understanding, it is interesting to analyze reasons why the two models can or cannot give similar results.

Estimates from the model of Stewart et al. (1983)

The Stewart et al. (1983) model is one of the most comprehensive energetic models for fishes (Hewett and Johnson 1992; Gerking 1994; Hanson et al. 1997). The framework of the complex model has been continuously used for evaluating salmonine stocking rates and prey fish production in the Laurentian Great Lakes (Stewart et al. 1981; Stewart and Binkowski 1986; Brandt et al. 1991; Stewart and Ibarra 1991; Lantry and Stewart 1993; Negus 1995; Rand et al. 1995; Rudstam 1996; Rand and Stewart 1998). The basic model structure also has been applied to estimate consumption by various salmonines in the Pacific Northwest, and model estimates have compared favorably with independent field estimates (Beauchamp et al. 1989; Brodeur et al. 1992). Such comparative analyses, however, require catching and sacrificing large numbers of individuals for a single daily consumption estimate. That requirement so far has precluded testing the lake trout model of Stewart et al. (1983) against independent field estimates of consumption.

To facilitate comparisons between models, we summarized the complex model in Table 1, and a general discussion about the model structure has been given in a previous section. We retained the same site-specific variables such as temperature regime, prey composition, and prey energy density that were applied in Stewart et al. (1983, p. 689 and Table 2). We updated the model based on new information on lake trout body size at ages (Keller et al. 1990).

The model was run on a daily time scale, and we summarized modeling results on a yearly basis (Table 2). Stewart et al. (1983) provided an explicit treatment of energy flow and conversion. For their practical application, however, they only reported mass growth and consumption. They also expressed GCE as mass conversion efficiency. In this paper, to compare the modeling results with that from our new model, we used the specific rates of energy assimilation and energy growth ($J \cdot J^{-1} \cdot t^{-1}$; Table 2). The assimilation efficiency and NCE were expressed as energy efficiencies. Stewart et al. (1983) treated gonadal growth and somatic growth as the same process. Such a treatment does not allow gonadal percentage to increase within a given reproductive season. At spawning time, however, 6.8% of total body energy was reduced as reproductive loss (Stewart et al. 1983). To compare results from the 1983 model with those estimated by eq. 7, the calculated energy NCE excludes gametes from observed growth (Table 2).

Estimating energy assimilation using eq. 7

Using only two parameters, eq. 7 allows us to estimate the pattern of specific assimilation rate ($J \cdot J^{-1} \cdot \text{year}^{-1}$) based on a pattern of specific growth rate. From parameters for the von Bertalanffy growth curve and the length–mass relationship presented in Keller et al. (1990) and mass–energy relationships in Table 1, we calculated W_a as $129.15 \times 10^6 J$. Following Calow (1977), we set the parameter a in eq. 7 equal to 0.04, assuming that an extremely small fish with only 1 J of body energy has the theoretical maximum NCE value of 0.96. In fish early life history, there may be an initial stage where NCE increases with increasing body size (He 1996). Here, the theoretical maximum value is used for understanding the decreasing trend of NCE versus body size, which is typical of fishes beyond that initial stage.

Table 1. Lake trout bioenergetics model of Stewart et al. (1983).

(A) The specific rate of consumption (C , $\text{g}\cdot\text{g}^{-1}\cdot\text{day}^{-1}$) is a function of body mass (W , g), temperature (T), and food availability (P):

$$C = 0.059W^{-0.307}e^{0.123TP}$$

(B) The proportion of egestion (F) to consumption is a function of temperature and food availability, but it needs to be adjusted for food composition:

$$F/C = [(0.212T^{-0.222} e^{0.631P} - K_{\text{inv}})(1 - K)/(1 - K_{\text{inv}})] + K$$

$$K = (P_{\text{inv}}K_{\text{inv}}) + (1 - P_{\text{inv}})K_{\text{fish}}$$

where $K_{\text{inv}} = 0.10$ is indigestible proportion of an invertebrate diet, $K_{\text{fish}} = 0.033$ is indigestible proportion of a fish diet, and P_{inv} is the invertebrate proportion of food.

(C) The proportion of excretion (E) to ($C - F$) is also a function of temperature and food availability:

$$E/(C - F) = 0.0314T^{0.580}e^{-0.299P}$$

(D) The specific dynamic action (SDA) is a constant proportion of ($C - F$):

$$\text{SDA} = 0.17(C - F)$$

(E) The specific rate of respiration (excluding SDA) in terms of prey biomass (R , $\text{g}\cdot\text{g}^{-1}\cdot\text{day}^{-1}$) is a function of lake trout body mass, temperature, and swimming speed (U , $\text{cm}\cdot\text{s}^{-1}$); the swimming speed in turn is a function of body mass and temperature:

$$R = 0.00463(Q_{\text{O}_2}/Q_{\text{prey}})W^{-0.295}e^{0.059T}e^{0.0232U}$$

$$U = 11.7W^{0.05}e^{0.0405T}$$

$$Q_{\text{O}_2} = 13560$$

$$Q_{\text{prey}} = f(\text{time})$$

where, Q_{O_2} is energy value of oxygen ($\text{J}\cdot\text{g O}_2^{-1}$) and Q_{prey} is energy density of prey ($\text{J}\cdot\text{g}^{-1}$), which may change through time.

(F) The specific rate of growth (G) is adjusted by prey and predator energy density (for practical operation, see Stewart et al. 1983, eqs. 16–19):

$$G = [(C - F - E) - (\text{SDA} + R)]Q_{\text{prey}}/Q_{\text{pred}}$$

$$Q_{\text{pred}} = 5700 + 3.08W, \text{ when } W \leq 1472 \text{ g}$$

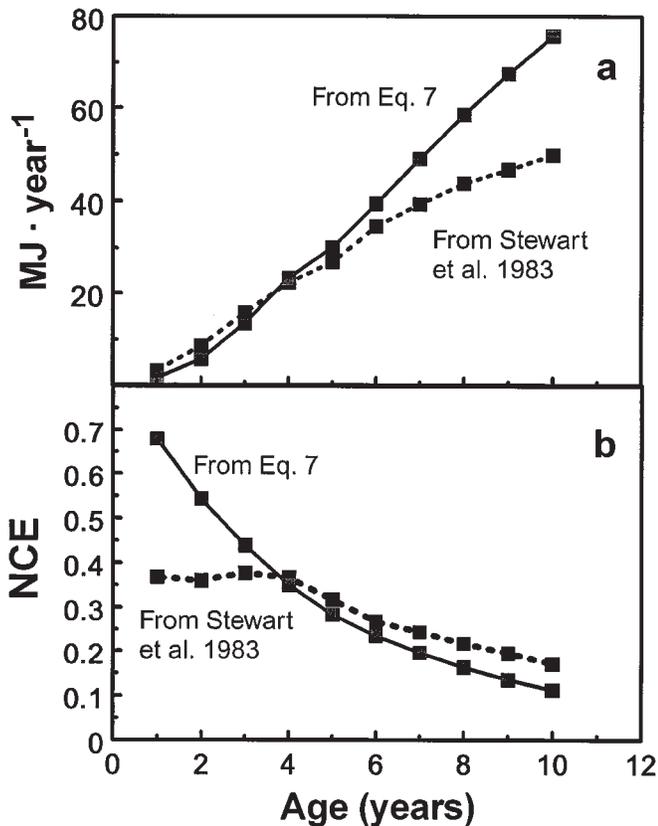
$$Q_{\text{pred}} = 9090 + 0.778W, \text{ when } W > 1472 \text{ g}$$

Table 2. Growth of average individual lake trout in Lake Michigan and modeled energetics using the model of Stewart et al. (1983).

Age-class	Initial body mass (g)	Gametes lost (g)	Initial body energy (J, $\times 10^6$)	Specific rate of assimilation ($\text{J}\cdot\text{J}^{-1}\cdot\text{year}^{-1}$)	α	Specific rate of growth ($\text{J}\cdot\text{J}^{-1}\cdot\text{year}^{-1}$)	NCE	P
1	30.7		0.18	18.35	0.76	6.75	0.368	0.6543
2	216.7		1.38	6.30	0.78	2.26	0.360	0.5604
3	597.8		4.51	3.51	0.79	1.32	0.378	0.5700
4	1138.7		10.48	2.13	0.79	0.78	0.367	0.5459
5	1781.4		18.66	1.44	0.80	0.46	0.318	0.4905
6	2470.2	192.6	27.20	1.28	0.79	0.34	0.268	0.5216
7	3160.9	240.4	36.51	1.08	0.79	0.26	0.244	0.5092
8	3822.7	285.4	46.12	0.95	0.80	0.21	0.217	0.4997
9	4436.3	327.0	55.64	0.84	0.80	0.16	0.195	0.4903
10	4991.7	364.0	64.76	0.77	0.80	0.13	0.171	0.4815

Note: Body mass is the initial body mass of each age-class on July 1. For mature fish, average gametes lost for both sexes is 6.8% of total body mass and is treated as a step function on October 30. Age-class 0 is not included because virtually all lake trout in Lake Michigan are of hatchery origin and stocked at age 1. α , Assimilation efficiency (assimilation/consumption); NCE, energy net conversion efficiency (growth/assimilation), where to compare with estimates from eq. 7, growth does not include gametes lost; P , ratio of estimated specific rate of consumption to the specific rate of consumption when food is unlimited.

Fig. 2. (a) Comparisons between two model estimates of energy assimilation for a lake trout growing from the start of age-1 to the end of age-10. The estimates are for both somatic and gonadal growth when a fish is mature (age-6 and older). (b) Comparisons between two model estimates of energy net conversion efficiency (NCE = growth/assimilation; growth did not include gametes lost).



Calow's (1977) theoretical calculation has rarely been applied in practical studies. We found that when we use body energy (J) rather than body mass, eq. 7 with Calow's maximum NCE value provided a reasonable estimation of NCE pattern versus body energy. With a equal to 0.04 and setting NCE equal to zero at the asymptotic body energy (129.15×10^6 J), we solved for the parameter $b = 0.1723$ in eq. 7. Then, a 1-year-old lake trout, with 30.7 g body mass and 0.18×10^6 J body energy, has a NCE value of 0.679. Lake trout energy density was higher than its prey energy density, and the invertebrate diet of age-1+ lake trout leads to a lower assimilation efficiency than that of older age-classes feeding on fish (Stewart et al. 1983). Thus, mass NCE at age-1 will be less than 50%, and mass GCE at age-1 will be around 35%.

From body energy at ages, we calculated the specific rate of growth ($J \cdot J^{-1} \cdot t^{-1}$) for each age-class (Table 2). Using eq. 7 with the above-estimated parameter values, we calculated specific assimilation rate (assimilation = growth/NCE). For immature lake trout growing from the initial size of age 1 to the final size of age-5, the estimated total assimilation (MJ) from eq. 7 was 3.1% less than that from the complex model (Fig. 2a). For mature lake trout growing from the initial size of age-6 to the final size of age-10, the estimated total as-

simulation from eq. 7 was 35.4% higher than that from the complex model (Fig. 2a). For the whole process of 10 years of growth, the estimated total assimilation from eq. 7 was 25.3% higher than that from the complex model.

When empirical growth pattern and energy density were given, the results from eq. 7 depended on the value of parameter a . A 10% increase or decrease in the value of parameter a did not change the general results. The estimated total energy assimilation from eq. 7 was about 21.7–28.7% higher than that from the complex model. From the complex model, assimilation efficiency (α) of food energy had a range of 0.76–0.80 (Table 2); those values can be used to calculate consumption (consumption = assimilation/ α) for each age-class. For our new model, assuming an assimilation efficiency of 0.80 (Winberg 1956) yielded total consumption estimates that were also about 25% higher than the complex model.

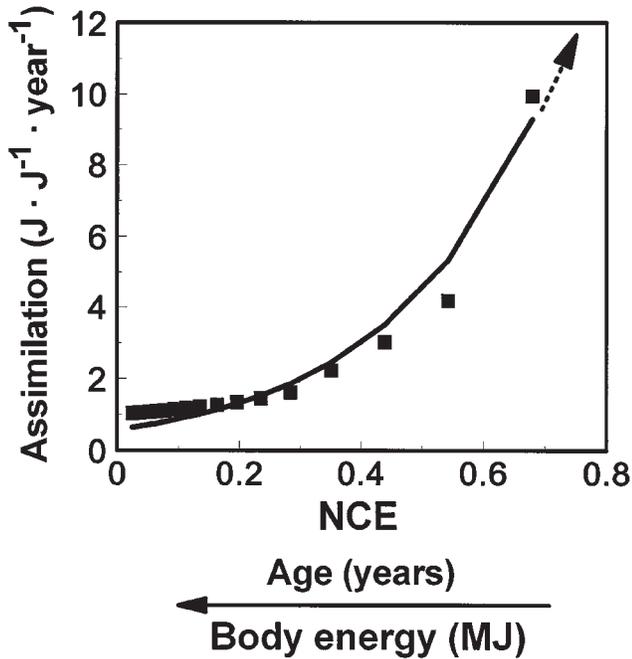
Do reproduction costs explain differences between model estimates?

Using eqs. 7–13, we may evaluate the effect of gonadal growth on total energy assimilation. First, we need to estimate the maximum specific rate of assimilation (A_m). Notice that in the complex model (Table 1), the maximum specific rate of consumption was defined as the specific consumption rate of any given-sized individual when food is unlimited. For our new model, the maximum specific assimilation rate (A_m) is defined such that, in a given environment, specific assimilation rate decreases as body energy increases. From eqs. 9–11, when NCE equals 1 and tissue turnover equals 0, A equals A_m . Considering that both NCE and specific assimilation rate decrease as body energy increases, we can plot the estimated specific assimilation rate versus NCE and extrapolate that relationship to estimate the theoretical maximum value A_m (Fig. 3). The estimated A_m was 34.39 ($J \cdot J^{-1} \cdot \text{year}^{-1}$).

Then, on a daily scale, we used eqs. 7 and 8 to simulate somatic growth and eqs. 12 and 13 to simulate gonadal growth (Fig. 4). Eschmeyer (1955) reported that average gonadal percentage of total body mass was 12.3% for lake trout females. The pattern of somatic growth is also known (Table 2), so the above simulation allowed us to estimate NCE_g for gonadal growth. Our simulation for an age-6 lake trout (first age of more than 50% maturity) suggested that reaching a gonadal energy of 12.3% total body energy requires an NCE_g of 0.270. A 10% decrease or increase in the estimated NCE_g value yielded gonadal percentages of 9.2 and 16.8%, respectively. A 10% decrease or increase in A_m produced gonadal percentages of 10.6 and 14.3%, respectively. With NCE_g equal to 0.270 and A_m equal to 34.39, an age-10 female lake trout will have a gonadal percentage of 13.8%. All of those estimated values fall within the range of empirical observations (Eschmeyer 1955). In contrast to females, males have a small gonadal percentage and a short gonadal development period (Eschmeyer 1955). We assumed that lake trout males have NCE_g values similar as those for females.

Based on eq. 7 and related parameter values, the estimated NCE values for mature lake trout (age-6 to age-10) were 0.235–0.112 (Fig. 2). Based on the daily scale simulation for both somatic growth (eqs. 7 and 8) and gonadal growth

Fig. 3. Estimating the maximum specific assimilation rate based on the trends of NCE and specific assimilation rate as age or body energy change. $A = 0.5821e^{4.0788NCE}$ ($R^2 = 0.98$), so A_m equals $34.39 \text{ J}\cdot\text{J}^{-1}\cdot\text{year}^{-1}$ when NCE equals 1.0. Regression was done using a nonlinear procedure (SAS Institute Inc. 1992).



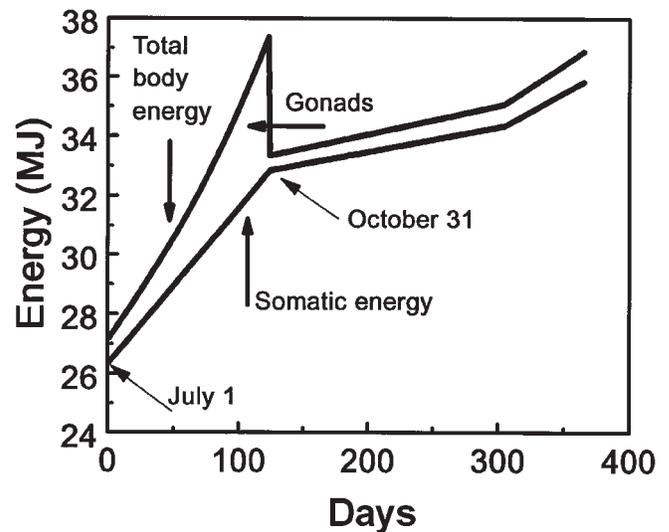
(eqs. 12 and 13), the exponential growth of gonadal tissue required a nearly constant NCE_g (0.270). Many empirical comparisons between gonadal and somatic growth efficiencies provided similar results (Brody 1945; Wootton and Evans 1976; Calow 1983; Rogers 1988). When gonadal growth is more efficient than adult somatic growth, we cannot use the direct cost of gonadal growth to explain the differences in estimated total energy assimilation between eq. 7 and the model of Stewart et al. (1983), although the latter model regarded gonadal and somatic growth as equivalent processes.

The primary difference between the two models and implications for future studies

There have been studies suggesting that the commonly used fish energetic models may underestimate consumption (Boisclair and Leggett 1989), particularly for mature fishes (Rowan and Rasmussen 1996). Their focus and energetic implications were different from our modeling analysis. For example, Rowan and Rasmussen (1996) suggested that activity can increase respiration of mature lake trout to as high as 4.19–6.97 times basal respiration. Using those estimates of activity costs and the swimming speed model of Stewart et al. (1983), mature female lake trout in lakes would be swimming 62–84 cm/s 24 h per day. In comparison, 3+ kg coho salmon (*Oncorhynchus kisutch*) and 5+ kg steelhead trout (*O. mykiss*) in the Pacific Ocean typically swim less than 45 cm/s (Ruggerone et al. 1990; Ogura and Ishida 1992; Rand et al. 1993).

The term of activity has been widely used without a clear definition. It is often calculated as (assimilation – growth – basal respiration)/basal respiration (Rowan and Rasmussen

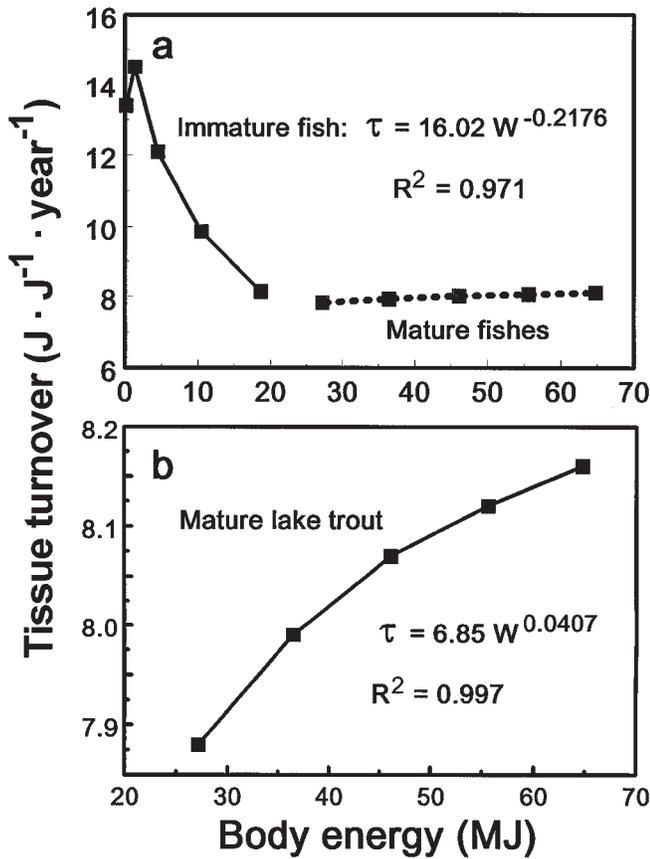
Fig. 4. Simulated daily somatic and gonadal growth of an age-6 lake trout based on our new model. The simulation was for 1 year. Initial and final body energy were calculated from empirical data (Stewart et al. 1983; Keller et al. 1990). Somatic growth follows eq. 8, and NCE for somatic growth follows eq. 7 ($a = 0.04$, $b = 0.1723$). The specific assimilation rate (A , $\text{J}\cdot\text{J}^{-1}\cdot\text{year}^{-1}$) was calculated from empirical annual specific growth rate (Table 2) and NCE, but the value of A was transformed to daily scale values ($\text{J}\cdot\text{J}^{-1}\cdot\text{year}^{-1}$) according to the following seasonal distribution: 70% in July to October; 15% in November to April; and 15% in May to June (Fry 1952; Lawrie 1963; Eck and Wells 1986). For this simulation, we did not consider fine details of daily temperature effect. Gonadal growth follows eqs. 12 and 13. The maximum annual specific rate of assimilation was estimated as $34.39 \text{ J}\cdot\text{J}^{-1}\cdot\text{year}^{-1}$ (Fig. 3), but that value was also transformed to daily scale values according to the above seasonal distribution. We adjusted the value of NCE_g for gonadal growth until our model predicted average female gonad percentage of 3% on July 1 and 12.3% on October 31. The gonadal growth pattern follows Eschmeyer's (1955) field observations, and the model simulation treated reproduction as a step function on October 31. The estimated NCE_g for gonadal growth was 0.270.



1996), but the result has been referred to as swimming or mechanical activity. Special care must be taken as follows:

- (1) Basal respiration is temperature dependent. Using the complex model to calculate activity must specify the temperature that has been used for calculating basal respiration.
- (2) When assimilation or consumption is estimated based on the specific rate of growth, the conceptual basis for the calculation also must be specified. In Stewart et al. (1983) and Rowan and Rasmussen (1996), gonadal growth was actually treated as energy or mass transformation from somatic tissue to gonadal tissue with an implied efficiency of nearly 100% (i.e., loss of somatic tissue to gonadal tissue without metabolic cost). Such an treatment may lead to an overestimate of energy demand in Rowan and Rasmussen's approach (He and Stewart 1997) and leave ambiguities in the complex model framework.

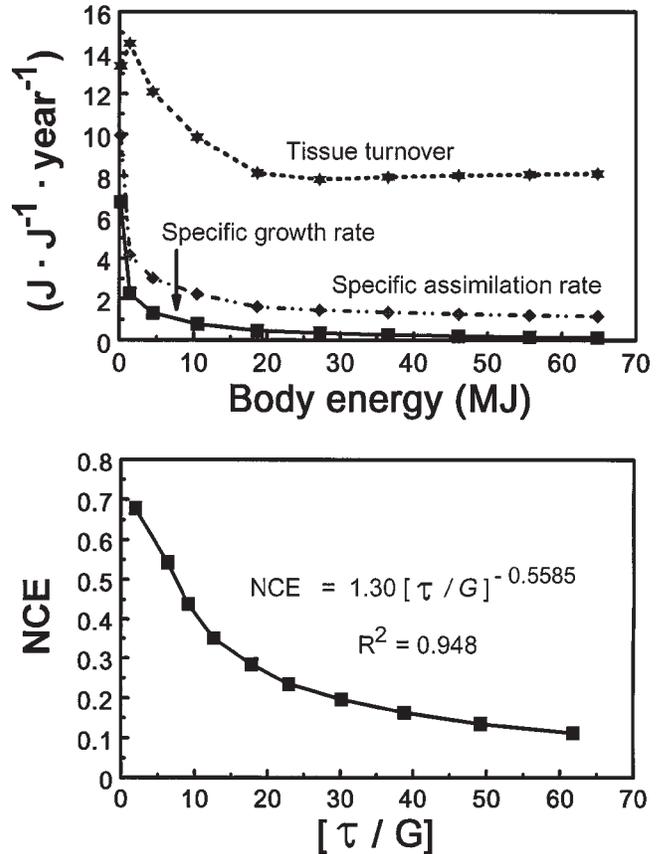
Fig. 5. (a) Modeled specific rate of tissue turnover as function of body energy, with emphasis on immature lake trout (age-1 to age-5; eq. 11). (b) Modeled specific rate of tissue turnover for mature lake trout (age-6 to age-10; eq. 11a).



(3) If tissue turnover increases with somatic or gonadal growth, related energetic cost should not be vaguely covered using mechanical activity.

A fish can have a higher specific assimilation rate for gonadal tissue than for somatic tissue, and our new model provided an explicit treatment. In addition to the direct energy demand for gonadal growth (eqs. 12 and 13), the rapid growth of gonadal tissue leads to increases in specific rate of somatic tissue turnover (eqs. 11 and 11a). For immature age-classes (age-classes 1–5), the estimated specific rate of tissue turnover followed a simple negative power function, except that age-class 1 somehow appeared to have different behavior (Fig. 5a). In an experimental study on rainbow trout, Houlihan et al. (1986) found a similar relationship, but their observations were based on body mass and protein turnover rather than total body energy and energy turnover. For sexually mature fishes, no experimental observations are available for comparison. Our model simulation suggested that, from age-6 to age-10, the specific rate of tissue turnover was relatively stable (Fig. 5a). On closer examination, however, the specific rate of tissue turnover increased slowly with total body energy, following a positive power function (Fig. 5b). Based on eqs. 7–11a, the modeled relationships among specific rates of growth, assimilation, and tissue turnover are positive (Fig. 6a). As the modeled ratio of tissue turnover to growth increases, NCE decreases

Fig. 6. (a) As body energy increases, specific rates of growth (G), assimilation (A), and tissue turnover (τ) decrease. The relationships among G , A , and τ are positive. (b) As modeled ratio of tissue turnover (τ) to growth (G) increases, NCE decreases.

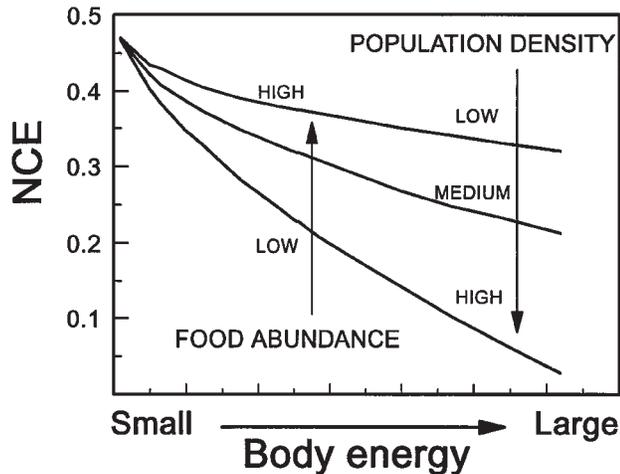


(Fig. 6b). Those relationships are consistent with empirical observations cited repeatedly in previous sections and were the basis for our model structure. Notice that the structural developments of eqs. 7 and 11 are independent of one another.

We cannot add the above-estimated tissue turnover as a new energy component in the energy balance of the complex model. As we indicated in previous sections, there are complex relationships among tissue turnover and other energetic components. The energy loss or cost related to tissue turnover may be partially covered by other energy components as formulated in the complex model. Our modeling results suggested that the important interrelationships of tissue turnover with other energetic components deserve explicit consideration in future studies.

The primary difference between the two models is the NCE pattern versus body energy (Fig. 2b). The complex model suggested NCE values with a relatively small range of variation (0.171–0.377). It also suggested that food availability (P value, Table 2) decreases as body size increases. Equation 7 does not involve any assumption about food availability related to age or body size. As age increases from 1 to 10 and body energy increases from 0.18 to 64.76 MJ, eq. 7 implied that NCE decreases from 0.679 to 0.112. From eq. 6, we understand that eq. 7 covers potential energy

Fig. 7. Qualitative relationships among food abundance, population density, body energy, and energy net conversion efficiency (NCE).



loss or cost of tissue turnover. In contrast, the complex model included many detailed components but lacked an explicit treatment of tissue turnover. The new model implied that rapid growth must accompany higher NCE, but the model does not explicitly consider seasonal variation in the pattern of NCE versus body energy. That may lead to a slight overestimation of energy demand. Advantages of the complex model include treatments of daily temperature regime and related seasonality of growth and energy density.

Perspectives

Normally, it is relatively easy to fit a growth curve, but it is very difficult to quantify energy demand–supply relationships and to confirm or explain a regulatory mechanism (Paloheimo and Dickie 1965; Ricker 1979). Field estimates of fish consumption also require modeling processes (Bajkov 1935; Eggers 1977; Elliott and Persson 1978), so few energetic models have been really confirmed (Boisclair and Leggett 1989). To improve that situation, there have been continuing efforts to independently analyze detailed energetic components in experiments or field studies. In conjunction with those studies, our effort addresses overall structures of models as foundations for designing various empirical investigations.

Ware (1982) suggested that food consumption was a function of swimming speed. Kerr (1982) suggested that activity was a function of consumption. Those two investigators used different cause–effect relationships, although both of them were discussing similar phenomena in field or laboratory populations. The commonly used complex model used a different approach. Its flexibility in practical applications has relied on the P value (Rice and Cochran 1984; Boisclair and Leggett 1989), which is normally explained as food availability or ecological constraint on feeding level. When we force a model to fit an empirical growth curve, the P value shows either an increasing or decreasing trend versus age or body energy (Table 2; see also Stewart et al. 1983; Rudstam et al. 1994). Such regular trends have not been clearly explained.

The relationship between consumption and swimming activity may never be explainable in terms of cause and effect. The relationships of SDA to consumption, food composition, body mass, temperature, and activity are even more complex (Beamish and Trippel 1990; Krohn et al. 1997). Beamish and Trippel (1990) cautioned that modelers should be aware of those real-world complexities and not oversimplify by using simple proportions or constants. We hope that interactions between modeling analyses and empirical investigations ultimately will lead to fundamental understanding.

Various energetic components are related to each other. To understand an integrative regulatory mechanism, we need to identify a key component and its relationships to others. Traditionally, the most important component has been chosen as consumption. With zero consumption, organisms have endogenous excretion and lose their body energy. With overconsumption, organisms will increase egestion and reduce assimilation efficiency (Elliott 1976). Feeding is an essential factor that influences swimming state of a fish; swimming activities, in turn, will require more consumption. Feeding also leads to SDA. From Winberg (1956), Ivlev (1961), and Paloheimo and Dickie (1965) to ongoing concerns about complex models (Kerr 1982; Boisclair and Leggett 1989; Beamish and Trippel 1990; Boisclair 1993; Krohn et al. 1997), consumption has been regarded as the most important driving factor in an energy balance system. Paloheimo and Dickie (1965) suggested that energy conversion efficiency decreases with increasing consumption rate ($g \cdot r^{-1}$). After their work, no other integrative mechanism has been suggested. Ware's (1982) work may represent a different approach. His model uses swimming speed as the central factor for optimizing energy acquisition and metabolic cost, but his energy-balance model does not address the allometry of consumption versus increasing body size.

Our new model provides an alternative perspective by emphasizing ontogeny of energetic relationships inside the metabolic system of an organism. We regard tissue turnover as the central factor. The specific rate of tissue turnover is a function of body size and various physiological states such as resting, swimming, growth, and reproduction. Basal metabolism is related to minimum rebuilding of tissues or metabolic substances. The cost of swimming, energy acquisition, and growth includes the increases in maintenance demand due to increases in tissue turnover.

On one hand, the specific rate of tissue turnover is positively related to NCE and various activities mentioned above. On the other hand, an increase in the ratio of tissue turnover to growth is responsible for the ontogenetic decreases in NCE and specific rates of assimilation and growth. Thus, endogenous energy flow plays a regular role for living activities. Certainly, there will be dynamic linkages between environmental changes and those ontogenetic processes. With low food abundance and high population density, endogenous demand will be high for acquiring exogenous energy, and NCE will decrease rapidly with increasing body size (Fig. 7). We leave more detailed discussions to subsequent developments and applications.

The Pütter – von Bertalanffy model has been widely used in both theoretical and empirical studies (Ricker 1975, Beverton and Holt 1993). It is good for fitting and comparing growth data, but its energetic basis has never been made

clear (Ricker 1979). It was originally based on mass-balance considerations (von Bertalanffy 1957) and has been the basis for various complex energetic models (e.g., Kitchell et al. 1977; Stewart et al. 1983). The Pütter – von Bertalanffy growth model, however, has rarely been used for quantifying energy demand–supply relationships. Following Brett (1970), Ursin (1979), Pauly (1986), and Reiss (1989), we show that the Pütter – von Bertalanffy model expresses a decreasing trend in NCE versus body energy, but the model structure does not express a mechanism for regulating ontogenetic changes in energy acquisition.

Our new model embodies an integration of fundamental concepts from the Pütter – von Bertalanffy, the Monod and the logistic models. The model structure includes three allometric functions of total body energy: growth, assimilation, and tissue turnover. The last one is the endogenous energy flow and may play a regulatory role when an organism responds to various environmental factors. Once those allometric functions have been independently quantified in a given experimental setting, we can use their relationships to exactly evaluate our model structure (eqs. 7–13). The model structure is analogous to the Monod model, so we may have a basis for studying the linkages between environmental controls and organismal responses. In analogy to the logistic model (see Ricker 1979), our model uses the W_a and A_m . The former is the body energy where NCE equals zero. The latter is the maximum specific assimilation rate when NCE equals 1 and specific rate of tissue turnover equals zero. Both W_a and A_m are theoretical, but both of them are implied by empirical patterns of NCE, specific assimilation rate, and specific growth rate versus body energy. The linkage between W_a and A_m is NCE, which is expressed by the Pütter – von Bertalanffy growth function (eqs. 4 and 7). Those relationships allow us to quantify parameter values when growth patterns are the only available data. Thus, our model is operational for practical applications.

Previous studies towards energetics-based population dynamic models include Ursin (1967), Van Winkle et al. (1997), and Jensen (1998). Using our new model we can address general regulatory mechanisms and provide an energetic framework for combining individual growth and reproduction. Such a linkage is essential for applications to population and food web dynamics. To emphasize integrative feedback structures (eqs. 9–11; Fig. 1), we reduced the complex expansion of the Pütter – von Bertalanffy model (Stewart et al. 1983) to its simplest form (NCE; eq. 7). Future experimental studies may provide a basis for including tissue turnover in the framework of the complex model. That, in turn, may allow us to abandon the use of P values to force a model to fit observed growth. In future developments and applications, it may be useful to expand our NCE equation to encompass much of the complex model with tissue turnover included. Those energetics details would allow us to apply our model on a finer time scale to evaluate dynamic effects of temperature, prey abundance, and other environmental factors. The specific rate of tissue turnover and its relationship to energetic constraints may provide a mechanistic basis for estimating natural mortality. Adding mortality due to predation and fishing will yield a complete energetics-based population dynamics model. Such a model will allow for evaluating future population trajectories in the

context of food web dynamics and under various management scenarios.

Acknowledgments

This study was funded by the National Oceanic and Atmospheric Administration (NOAA) award No. NA46RG0090, Project No. R/CE-8, to the Research Foundation of State University of New York for the New York Sea Grant Institute. This study was also funded, in part, by the Natural Sciences and Engineering Research Council of Canada and Environment Canada. The U.S. Government is authorized to produce and distribute reprints for government purposes notwithstanding any copyright notation that may appear hereon. The views expressed herein are those of the authors and do not necessarily reflect the views of NOAA or any of its sub-agencies.

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