Modeling livestock systems. II. Understanding the relevant biology

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ABSTRACT: Essential components of bio-mathematical simulation models of livestock systems were examined for degree of understanding, qualitative and quantitative, of the relevant underlying biology. Key knowledge gaps in modeling of growth, lactation, and energy metabolism were shown to exist. Suggestions for discipline-oriented research, directed to close these gaps, are given.

Key words: Bio-mathematical models, livestock systems, systems analysis

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Modelos de simulación de sistemas de producción animal. II. Comprendiendo la biología relevante

RESUMEN: Se presenta un análisis de componentes claves de modelos bio-matemáticos de sistemas de producción animal. Los resultados sugieren conocimientos deficientes en descripción cuantitativa de crecimiento, lactación, y metabolismo energético. Se presentan sugerencias para la investigación científica de la biología relevante para modelos de simulación de producción animal.

Palabras clave: Modelos bio-matematicos, sistemas de producción animal, análisis de sistema

Introduction

Pittroff *et al*. (2002) argued that challenges faced by the livestock industry due to increasing demand for livestock products, environmental damage caused by livestock production, and increasingly difficult resource constraints will require the development of production system technology capable of addressing complex interactions between livestock and the natural and production environments. One of the driving forces behind the development of simulation models of livestock systems was the desire to quantitatively describe the interaction of a farm animal with its environment: given a certain genotypic performance potential, a certain management regime, and a certain environment, how will an animal perform? This kind of prediction capability would be required in order to optimize livestock systems under a variety of objective functions and constraints. However, quantifying the effects of the interaction of a farm animal with its environment requires the ability to qualitatively understand and to quantitatively describe the biological functions that account for the variation in observed phenotypic performance. Here, we will discuss the current state of qualitative and quantitative understanding of those biological processes considered to be relevant for nutrient supply driven simulation models of livestock systems. In a previous paper (Pittroff and Cartwright, 2002), this model archetype was identified as the design of broadest potential applicability.

Modeling the Biology of Traits Accounting for Phenotypic Variation

The design of the perhaps most widely applied nutrient supply driven production system model, the Texas A&M Beef Cattle, Sheep and Goat Model (Sanders and Cartwright, 1979a,b; Blackburn and Cartwright, 1987), is based on the assumption that the largest amount of variation in overall performance in livestock breeds is due to four characteristics: (1) mature size, (2) maturing rate, (3) milk production, and (4) fiber production (in the case of sheep and goats, where applicable). Many other model designs have adopted this assumption. Accordingly, the model defines

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the genotypic performance potential in these traits for each animal, and evaluates at each time step or over each integration interval if the nutrient supply available to the animal is commensurate with the performance potential. Actual growth, reproduction, and survival are a function of the degree of match of performance potential with actually available nutrient supply.

The functions employed to quantify these relationships are quite aggregated, i.e. operate on a much lower level of resolution than physiology models using stoichiometric relationships for the description of digestion or anabolic processes or DNA/cellularity approaches to modeling growth physiology. There have never been any serious attempts to combine detailed nutrient supply driven physiology models with herd level production system models, but that should not rule out such exercises in the future. We would like to caution, however, that increasing the level of resolution generally leads to increasing omission of essential information, usually (but not exclusively) caused by failure of the real world to provide input and validation data commensurate with the level of detail of the model. This runs counter to the intuition of many.

Regardless of specific design, nutrient supply driven models must accomplish two essential tasks: the quantification of nutrients available for the animal and the specification of genotypic performance potential.

Modeling Growth

In growing animals, nutrient requirements for growth constitute the largest fraction of overall requirements. A realistic quantification of this component is desirable for two reasons: (1) intake regulation is closely linked with overall nutrient requirements, and (2) one of the most critical needs of the livestock industry is the ability to predict body composition based on estimated intake of nutrients.

All nutrient supply driven models of livestock systems employ a mathematical description of growth as the 'backbone' of the quantification of nutrient requirements. Animal performance is calculated according to the degree of match between nutrient supply and requirements. Sanders (1977) proposed the entity 'structural size' (*WM*) for the theoretical growth curve. This curve computes extent and rate of growth that would be achieved under ideal nutrition and environmental conditions. Actual growth is computed relative to this theoretical function. Parameters of the function describing WM are considered breed properties, allowing in principle the definition of phenotypic and genotypic variability for potential growth. The equation for *WM* after puberty is:

$$WM = WMA - (WMA - WMP) * e^{-k(t-t_i)}$$

where *WMA* defines mature weight, *WMP* denotes weight at puberty, k is absolute growth rate and t_i stands for age beyond puberty. The author described this equation as being 'of the same type' as the function used by Brody (1945) for the description of post-pubertal growth. However, this assessment may be misleading. Brody (1945) concluded that growth is continuous and has continuous rates of change of all orders, except at the point of inflection, which he considered to occur at puberty. Consequently, rate of change is not defined at this point. Brody (1945) reasoned that growth seems to occur in two distinct phases, a self-accelerating and a self-inhibiting phase. His equation for the post-pubertal phase is:

$$W = A - B * e^{-k*t}$$

which can be transformed by starting to begin to count t from the point where this curve intersects the x-axis (t^*):

$$W = A * (1 - e^{-k(t-t^*)})$$

Note that Brody (1945) did not state an explicit domain-restriction of this curve, which leads, as pointed out by Doren *et al*. (1989) to large residual errors when fitting *W,t* data including age intervals before puberty to this function. Fitting of the Brody curve to data usually involves a heuristic estimate of the inflection point of the growth curve, such as in the study presented by Nelsen *et al*. (1982a). Brown *et al*. (1972a,b) fitted *W,t* data involving observations pre-dating the point of inflection of the growth curve. From Table 2 in Brown *et al*. (1972a) it is evident that their estimates were associated with substantial residual errors. This is in part a result of their method of curve fitting, in part a result of the extremely heterogeneous data set which involved weight records on Angus cattle from 1950 to 1967, a period during which major changes in the growth properties of that breed occurred. Both cases illustrate the problems encountered when using the Brody function.

A potential problem introduced by use of the Sanders (1977) equation is that his and Brody's equation are only equivalent if an explicit statement is made about *WMP*. This by necessity also includes a statement about age at puberty and can be made more apparent by solving for the two age origins as follows and setting *WMA* equal to *A, WM* to *W* and denoting *WMP* as *P*:

Sanders:

$$t' = \frac{1}{k} * \left(\ln(A - W) - \ln(A - P)\right) + t$$

Brody:

$$t^* = \frac{1}{k} * \ln\left(1 - \frac{W}{A}\right) + t$$

Brody (1945) seemed to have been aware of the difficulties of estimating age origin or the inflection point of the growth curve. It should be pointed out that in his examples (Brody 1945, Chapter 16) the parameter *B* is fitted graphically, and t^* is derived subsequently. This is done by plotting *(A-W)* on a logarithmic scale against age on an arithmetic scale. If a correct value for *A* is chosen, a straight line results. The intersection of this line with the *(A-W)* axis yields the parameter *B* and the age origin can be derived thereafter.

Since the Brody equation does not allow determination of the point of inflection of the growth curve, the only feasible approach using least-squares curve-fitting would be to use growth functions which possess a point of inflection and assume that the derived value would be equivalent to the point at which puberty occurs. Doren *et al*. (1989) is an excellent example of the difficulties encountered using this method. Recent experimental data (Pittroff *et al*., 1999; Pittroff and Keisler, unpublished) suggest that the assumption of occurrence of puberty at the inflection point of the growth curve may not be correct. Table 1 summarizes the effects of three distinct nutritional treatments (CONTROL: alfalfa plus minor grain supplement; LEAN: high energy, high protein to maximize growth; FAT: high energy, protein limited (<8% CP) to maximize fat deposition under lean growth restriction) on the growth program and onset of puberty (as indicated by plasma progesterone levels 0.5 and 1.0 ng, respectively) of ewe lambs in two genetic lines (41: long-term selected for weaning weight/ewe; 42: unselected control line). It is obvious that there is no discernible relationship between the commonly used indicator for onset of puberty and the inflection point of the growth curve. Incidentally, it was not possible to fit to these data a new growth model proposed by Lopez *et al.* (2000) that claims high flexibility due to a variable inflection point. Fitting of the data to this curve

$$W = \frac{W_0 * K^c + W_f * t^c}{K^c + t^c}$$

with W_0 and W_f denoting initial and final weight, respectively, and K and c denoting parameters, failed for all age-weight relationships (with 23 measurements from birth to approximately 300 days of age available) for this experiment comprising 6 groups of approximately 20 animals each.

In order to parameterize the Sanders (1977) model (and other models based on his approach, such as the Sheep and Goat Model (Blackburn and Cartwright, 1987), several assumptions are required. First, *WM* at birth is set to 1/15 of the mature size; and fixed proportions of *WM* correspond to the one, two and three-year age points. The model uses a reference breed to parameterize the variables for other breeds; consequently, all breeds 'behave' similarly. For example, animals of larger mature size mature slower. That is, if the reference breed is used, growth curves of all simulated breeds have essentially identical shapes. Since it is known that the shape of the growth curve does vary between breeds, the application of this model is limited by available information about these parameters. This becomes an especially critical problem considering that stage of maturity in the simulation models discussed here is quantified not only in terms of *WM* but also entails fixed percentages of body fat. That is, rate of maturing is conceptually equivalent to rate of fattening in these models. However, according to Webster *et al*. (1982), this is not necessarily the case. An analysis of growth records of sires of four breeds by these authors showed that there are late maturing and early fattening cattle breeds, such as the Angus, and late maturing and late fattening ones, such as the Charolaise. However, it must be added that this study calculated degree of maturity using the metabolic age function presented by Taylor (1965). This approach has potential shortcomings, as will be discussed below. Since *WM* of males is considered to be a fixed proportion of *WM* of females at all stages of development, additional discrepancies occur. These apparent inconsistencies, together with the need to parameterize the models based on a reference breed, are likely the reason for frequent deviations of model results from observational data for body composition.

Scaling Rules

The nutrition literature almost always presents measurements scaled to the unit of metabolic weight. Taylor (1965) introduced widely cited scaling rules with the objective of placing growth, feed intake and performance characteristics of different breeds on a common scale. It seems worthwhile to trace the origin of these rules. Taylor (1965) derived an expression for standardized time taken to mature based on a reference to mature weight as a linear regression of $\log 1/k$ on $\log A$ (where k, as above, has the meaning of growth rate, and A denotes mature size). The data set was Brody's (1945) table on p. 567, with 75 data points from 12 species.

Table 1. Comparison of age at which specific threshold levels of progesterone concentration in plasma were first detected, with age at the inflection point of the growth curve (three parameter logistic curve).

Line	Treatment	Age P4[1] 0.5 ng (days)	Age P4 1.0 ng (days)	Age Inflection Point (days)
41	Control	210.3	218.9	124.2
41	Lean	195	206.2	162.5
41	Fat	203.2	207.3	162
42	Control	207.4	216.4	113.2
42	Lean	209	220	154.2
42	Fat	209.8	217.6	174.4

[1]Plasma concentration of progesterone.

With several species (which were considered to be deviant from the average) excluded, Taylor (1965) finally derived a regression coefficient of 0.27. From the same data set, he derived an expected value of the time origin of the Brody curve (denoted as t^* above, and with a starting point of 3.5 days after conception) as:

$$E_{(t^*-3.5)} = 0.5 * k^{-1}$$

Taking both derivations together, a proportionality for time required to reach fixed stages of maturity was developed as:

$$E_{(t_u-3.5)} = \alpha * A^{0.27}$$

which, as observed by Webster (1988, 1989) constitutes an allometric relationship. This can be conveniently expressed as:

$$\Phi = \gamma * t * A^{0.27}$$

where Φ is metabolic age, γ denotes a constant introduced to ensure consistent dimensions and t stands for time since conception. This is the function for metabolic age, and was extended by Webster (1988, 1989) to describe presumably related entities such as 'metabolic turnover time' and protein turnover time.

This relationship is remarkable in two ways. One, it seems to confirm the law of proportionality relating metabolic measurements in an allometric expression to weight; and two, its derivation is solely based on *one* data set comprising 75 points from 12 species.

It seems appropriate to question this relationship in terms of its usefulness for scaling for inter-breed comparisons (e.g. Taylor *et al*., 1986). Parks (1982) noted that it may be appropriate for *inter-species comparisons*. Such a study was conducted by Webster (1989). When discussed in the context of the relationship between rate of fattening and rate of maturing, it would follow that the context of metabolic age would also imply the existence of identical coefficients in allometric relations between body fat and body weight. However, as summarized by Parks (1982), there are large within-breed differences in that relationship and this author concluded that there is no answer to the question: "At what age does an animal begin to fatten?" That is, either the concept of allometric relations is not valid or the widely accepted hypothesis of a close association of rate of fattening and rate of maturing is not correct. In either case, in-depth re-evaluation of quantitative relationships used in modeling of animal growth and feed intake is required. This contention is further corroborated by Peters (1988) who conducted an analysis of the statistics of allometric relationships involving taxa of different order. His conclusion was that allometry (he was regressing growth rate on body weight) becomes less effective at defining functional relationships as smaller taxa are considered because of a sharp decline observed in the R^2 values, as comparisons involve smaller taxa. Peters (1988) also stated that the decline of the standard error of the mean in allometric relationships involving progressively smaller taxa would indicate better prediction capabilities. Unfortunately, Peters (1988) denoted S_{xy} as 'standard error of the mean, calculated as the root of the mean squared error'. What he was actually referring to was the standard deviation for the predicted value of growth rate at a given value of weight (he implicitly referenced the prediction band for growth rate), which is obviously not the root of the mean squared error of the regression equation. The notation S_{xy} is also not correct as it commonly denotes the sum of products divided by degrees of freedom, i.e. a covariance. It seems that the high R^2 value obtained by Peters (1988) for a regression equation with the independent variable ranging from 10^{-9} to 10^6 units is attributable to a large value of total sums of squares rather than a good explanatory model, even when considering that he used a logarithmic model. Conversely, the smaller standard deviation of the predicted values for data sets involving smaller taxa is in all likelihood the result of a generally more homogenous data set within taxon. Thus, Peters' study (1988) illustrated potential fallacies of inter-species comparisons judged by regression diagnostics.

The practical use of the metabolic age function is discussed by Taylor and Fitzhugh (1971), Fitzhugh and Taylor (1971), Fitzhugh (1976), Smith *et al*. (1976a,b), Taylor (1980a,b), and Nelsen *et al*. (1982b) for the derivation of 'genetically standardized growth equations' and their application to the estimation of genetic parameters of size at maturity and maturing interval. Expected values for degree of maturity achieved at a given metabolic age, or an expected body weight curve can be obtained from knowledge of the mature size of the animal. Standardization of the parameters of the Brody curve is achieved as follows:

$$\frac{W}{A} = 1 - e^{-k^{*}(\Phi - \Phi^*)}$$

where $k^{'}$ denotes the growth rate multiplied by $A^{0.27}$ and stands for metabolic age, as above. Obviously, mature size must be known and use of the relation depends on applicability of the assumed proportionality which is based on a very small data set involving inter-species comparison. Further, the use of what Fitzhugh (1976) termed the 'equation free method' (he was probably referring to a 'growth equation-free' method), namely the following model:

$$Y_t = U_t * A$$

where Y_t is size at age t, U_t is degree of maturity and A is mature size, not only depends on the accuracy of the estimate of mature size, but also on the independence of W,t data from nutrition. That is, for the purpose of breed comparisons it must be assumed that environmental conditions are sufficient for all breeds to express their maximum growth potential, or conversely, if environmental conditions are limiting, that all breeds are affected in similar manner. This seems to be highly unlikely. In other words, the estimation of genetic parameters for growth curve parameters using the currently available information seems to be subject to intractable confounding with environmental effects.

Taylor (1968) found values for the exponent in the allometric relationship of maturing interval to mature size rang-

ing from 0.182 to 0.485. One has to wonder how the confidence in a value of 0.27, considered to be a mean applicable to inter-breed standardizations, is justified. Immediately, an analogy to the many divergent values given for the exponent of 'metabolic weight' comes to mind. Notter *et al*. (1984) found very large deviations from Taylor's exponent of 0.27 in their study of breed effects on feed efficiency in sheep.

Taylor made the very important point, consistently overlooked in the literature, including Webster (1989), that the proportionality was expressed as a power function of *mature size*, not current weight. However, the latter proportionality is commonly used in animal nutrition to standardize nutrient requirements and feed efficiencies. This means in essence that the calculations of requirements for growth based on a power function of actual weight cannot be correct. In other words, all current requirement prediction systems may not correctly calculate nutrient requirements for growth. Parks (1982, p. 102-103) stressed that the use of proportionalities to metabolic weight of growing animals is inappropriate. That is, the extrapolation of the relation between basal metabolic rate to a power function of weight, which may have a basis in mature animals, is not correct for all other purposes.

Although Parks (1982) is a widely cited source, no reference in the literature could be found relating to his discussion of allometric relationships between body components (Parks p. 250-251). This discussion is based in part on his finding of contradictory values for the exponent in the allometric relationship between body fat and body weight between and within species, and secondly on the mathematical properties of allometric relationships between body components. Considering growing animals, this relationship can only be correct if all exponents are equal to unity, or their sum is unity. His proof proceeds as follows:

Let W be the total weight of the organism, w_i be the weight of the i-th component, c_i be a constant factor and d_i be the exponent in the power function of total body weight:

$$W = \sum_{i=1}^{n} c_i W^{d_i}$$

then differentiating W with respect to time yields:

$$\frac{dW}{dt} = \sum_{i=1}^{n} c_i d_i W^{d_i - 1} \frac{dW}{dt}$$

with: and, this reduces to:

$$W = \sum_{i=1}^{n} d_i w_i$$

This derivation (equivalence of the first and last equation) can only be correct if all the exponents are equal to 1, or if their sum is equal to 1. Obviously, this is not correct for weight relationships between body components at various stages of growth, as data from many experiments suggest. As an example, Baker *et al*. (1991) found exponents greater than 1 for carcass weight, empty body protein, empty body fat, carcass protein and carcass fat in allometric relationships of these entities to live weight. Unfortunately, this latter study presents some difficulties for interpretation because the exponents were termed 'growth coefficients'. However, the analysis did not develop growth curves for individual components but analyzed cross-sectional data (serial slaughter design) within breed with a linear regression model with the log-transformed observations for empty body weight, carcass weight, empty body protein, empty body fat, carcass protein and carcass fat as dependent variables and log-transformed values of empty body weight, live weight and carcass weight as independent variables. The analysis of Baker *et al*. (1991) pooled all cross-sectional data. Because the coefficients (estimated as regression coefficients) did not describe the rate of change in the sense of k in a growth equation, the term 'relative growth coefficient' employed by the authors is inappropriate and confusing.

Baker *et al*. (1991) referenced Huxley (1932) as the original source for the use of allometric relations, but this author did not use the term 'growth coefficient' either. He used the term 'growth ratio', and postulated that although the growth coefficients of any two body components change in absolute terms during the development cycle, their ratio does not. Huxley (1932, p. 6) also emphasized that the base to the exponent denoting the 'growth ratio' must be calculated as some linear measurement of the size of the animal *minus* the measurement of the size of the component or organ whose allometric relationship with the whole is determined. If W in the above proof by Parks (1982) is set to W-w_i, his proof is not valid.

On the other hand, the study by Baker *et al*. (1991) produced the very interesting result that except for the relationship between empty body weight and live weight, no statistically significant differences among breeds in the exponents could be found. This result may be attributable to the relatively small subclass size and the use of cross-sectional data, however. The authors speculated that the absence of significant differences was in part explained by the nutritional regime not ensuring maximal growth. This explanation seems highly unlikely given that the animals in this study were fed a high-quality diet ad libitum. Rather, the results of this study, in spite of its small data set, should give rise to re-examination of (a) the usefulness of allometric relations, and (b) the apparent contradictions in the literature regarding breed differences in body composition independent of stage of maturity and nutrition.

Unfortunately, the literature on growth and requirements is almost intractable because of divergent methods for 'standardizing' or scaling results. Few if any studies are directly comparable. Consider Jenkins and Ferrell (1997), for example, who found differences in proportion of body components of mature cows attributable to breed and feeding regime differences in a planned study. However, the point of reference used in this study was mature weight scaled to a fixed fat percentage by use of various regression techniques. This is an example of application of the assumption that rate of maturing and rate of fattening are equivalent,

which clearly would be wrong, if the analysis of Webster *et al*. (1982) produced correct results. The study of Jenkins and Ferrell (1997) was a replicate of Taylor and Murray (1991); the latter authors also found breed differences in proportion of body components. It is interesting that Williams and Jenkins (1997) stated that the calculation of body composition at the same proportion of the respective mature body size varies between models of growth. These authors questioned the widely used value of 25% body fat achieved under 'normal' feeding conditions because they could find only one reference to experimental data. Williams and Jenkins (1997) further stated that in this experimental work, a different value could have been found, had the animals been fed differently. This statement is appropriate, and sheds doubt on the regression procedure used by Jenkins and Ferrell (1997). These sources document the degree of disagreement even within work groups found in the literature on key postulates relevant for modeling of growth and body composition.

We conclude regarding current issues in modeling growth:

- Difficulty to define point of inflection of the growth curve;
- Sparse information on breed specific parameters of the growth curve (between and within breeds);
- Unclear breed differences in rate of maturing of different body components;
- Unclear effects of level of feeding on proportions of body components at various stages of maturity;
- Unclear relationship between rate of fattening and rate of maturing;
- Unclear relationship between growth of intact males, castrated males and females;
- Possibly improper scaling and standardization methods for experimental results;
- Possibly inappropriate data analysis methods in the past (allometric relationships).

It appears that only a systematic effort, both literature review and experimental work, can produce the required advance in knowledge. Little explanatory insight may be gained from entirely empirical models of body composition such as the models published by Keele *et al*. (1992), Williams *et al*. (1992) and Williams and Jenkins (1997). On the other hand, the paper by Lopez *et al*. (2000) proposes a 'flexible' model for animal growth that is capable of describing very different growth patterns (sigmoidal and diminishing returns; variable point of inflection). The authors suggest that the mathematical form chosen can overcome many problems encountered in the past when fitting *w,t* data. This publication, however, seems to be symptomatic of incorrect priorities: a model of animal growth (and composition, for that matter) that can serve as a *prediction* model must be capable of predicting the *modification* of some genotypically specific quantitative description of *growth potential* as the response to provision of actually available nutrients. But consider that even detailed models conceptualizing growth as a function of DNA pools ultimately have to resort to astonishingly simple 'nutrition factors' (see Oltjen *et al*., 2000) that are but multiplicative, entirely empirical correction factors provided as inputs to these models. Further, the Oltjen *et al*. model, as an example, considers the effects of synthesis and degradation, but does not specify the effects of diet composition on either process. The reason is simple: There are no suitable experimental data on ruminants available. Absence of data is not equivalent to irrelevance – consider the finding of Radcliffe and Webster (1976, 1978, 1979) that rats fed a source of strongly imbalanced protein maintained intake of that diet on a level leading to accelerated weight loss. The 'imbalanced amino acid (IMB)' experimental paradigm employed in research on neuro-hormonal regulation of intake has shown convincingly the magnitude of effects of protein quality on tissue degradation and synthesis (Gietzen, 1993). Data on absolute or relative (temporal) nutrient imbalances are relevant also for ruminants – for example, yield level effects of (a) synchronization of protein and energy supply to the host animal (Robinson *et al*., 1997; Shabi *et al*., 1998), (b) protein – energy ratio in the diet (Fattet *et al*., 1984; Vipond *et al*., 1989; Sinclair *et al*., 1995; Witt *et al*., 1999), and (c) physical form of the feed (Reynolds *et al*., 1991; Lachica *et al*., 1997) have been clearly documented in ruminants. Summarizing, we suggest that real progress in quantitative understanding and prediction of growth is unlikely without (a) a definition of growth potential (see above discussion specifically on properties of breeds), and (b) appropriate prediction of intake and nutrient fluxes.

Modeling Lactation

Issues of modeling lactation essentially mirror those found in growth model concepts. The basic physiology of mammogenesis, lactogenesis and galactopoiesis seems to be well understood, perhaps with the exception of early embryonic processes, in particular differentiation. However, important knowledge gaps in nutrient uptake and partitioning remain. Thus, mechanistic modeling concepts on various levels of aggregation rooted in physiology have been developed (Bywater, 1976; Cañas *et al*., 1982; Waghorn and Baldwin, 1984; Baldwin *et al*., 1987a,b,c). A chapter in Baldwin (1995) on the biology of lactation (Baldwin and Ferrell, 1995) describes the evolution of a mechanistic model of lactation as an attempt at identifying knowledge gaps in the understanding of galactopoiesis, a rather interesting exercise as it uncovered deficient information in other areas of physiology as well (protein turnover, maintenance energy requirements, nutrient partitioning, e.g.). This explains why mechanistic models often produce unsatisfactory results, in particular in regards to prediction of yield response to changes in nutrition. Prediction of response in milk composition to changes in diet seems to be particularly refractory. Not only nutrient transactions are important, however. Davis *et al*. (1999) reviewed the available infor-

mation on yield loss effects of once daily milking, a practice quite relevant in extensive, pasture-based systems, and found evidence pointing to local, intra-mammal mechanisms largely independent of genetic differences for milk composition and of nutritional regimes. A hormonal (autocrine) regulation mechanism seems to await full elucidation (Davis *et al*., 1999). In any case, the capacity to predict lactation performance based on a model of the three elementary processes involved in lactation under a variety of feeding regimes, for a range of genotypes, ultimately requires a thorough understanding of the interaction between hormonal regulation and nutrition, specific to genotypes. This implies the necessity to expand the focus of models purely based on nutritional interactions. However, mechanistic modeling of lactation considering nutritional interactions does not seem to be a very active field, l*et al*one in integration with endocrinological applications. Under such circumstances, how could one expect so-called functional genomic approaches in lactation biology to be successful?

Akin to the situation found in growth modeling, efforts to find appropriate mathematical representations of the lactation curve dominate the literature. This interest is easily explained by the need to predict yield based on partial (test) records. Bayesian methods have helped to advance such applications (Goodall and Sprevak, 1985), as in growth modeling (Oltjen and Owens, 1987). However, fitting a lactation curve is retrospective (Broster and Thomas, 1984). In terms of functional understanding required for prediction, it might as well be called irrelevant. Nevertheless, there are curve-fitting exercises yielding functional insight. Madalena *et al*. (1979) conducted an ANOVA of parameters of the Gamma function (Wood, 1967) fitted to lactation data in Brazilian cattle. This study is interesting because it sheds some light on the relative significance of genotypic performance potential vs. nutrition in lactation. The Gamma function model of Wood (1967) has three parameters:

$$y_n = a * n^b * e^{-c*n}$$

where y_n denotes average daily yield in the n^{th} week.

Initial and maximum yield is determined by a, whereas persistency can be expressed as

$$S = c^{-(b+1)}$$

Season of calving was the most significant factor explaining variation in a. Under the conditions of tropical Brazil, this points to overriding significance of nutrition in determining maximum yield. Since initial and maximum yield differences are the most important characteristics distinguishing lactation curves of dairy cattle under tropical compared to temperate conditions (Madalena *et al*., 1979), it follows that genetic differences between breeds per se may not be as important as one could expect from a data material comprising performance records ranging from purebred Holstein to ¾ Zebu genotypes. Breed was still a moderately significant factor for a; however, lactation curves for all breeds were essentially linear and parallel, with the F1 between Zebu and Holstein ranking first. Further, breed was significant for only one parameter involved in persistency, whereas season of calving again was significant for both. This paper suggests that the basic processes underlying milk production are probably the same for all breeds, even genetically distant ones; however, fitness traits related to adaptation to climate, parasites, and feed quality in terms of being permissive for the expression of inherent lactation performance potential are probably of pervasive significance under harsh production conditions. Clearly, very little is understood in this area. It has been known for a long time that lactation length under tropical conditions tends to be considerably shorter than in the temperate zone, regardless of breed (Mahadevan, 1966). Interactions between genetic and environmental factors may be expected to play a major role. The fact that a significant phenotypic correlation between performance traits and 'adaptation traits' (we still have no proper definition of these) may exist without any underlying genetic correlation does not bode well for genomics approaches to improving lactation performance under harsh production conditions, but definitely warrants substantial research into functional understanding of the underlying physiology, in particular nutrient acquisition and nutrient partitioning. We do not currently see pertinent research programs. It is noted in passing that 'harsh production conditions' may also apply to high performance dairy cows constantly operating at or beyond their physiological limits. Those are cows whose yield levels compromise their fitness, by causing considerable rates of reproductive failure, the most important cause of involuntary culling in intensive dairy systems.

Modeling Maintenance Requirements

It is currently at least as difficult to correctly estimate nutrient requirements for maintenance as is the case with requirements for growth or lactation. However, there seems to be more information available about the nature of the contradictions in published data. Wallach *et al*. (1984) published a detailed comparison of equations used for calculating energy maintenance requirements for sheep. Unfortunately, their discussion did not address the questions of scaling of requirements to metabolic weight and change of maintenance requirements under prolonged nutrient deficiency. Their review however, did point out that available quantitative models produce highly divergent predictions for identical animal types and feeding conditions.

Koong *et al*. (1985) presented a discussion of the dynamics in energy maintenance requirements and their causes. A number of experiments involving rats, pigs and sheep clearly show that level of nutrition has a profound impact on the size of the metabolically most active organs. Maintenance requirements (as expressed by fasting heat production (FHP)) are much more closely related to the weight of these organs than to any power function of live weight. For example, Koong *et al*. (1982) found that FHP of

pigs raised to the same final weight on two different feeding schedules differed by about 50%. These observations are supported by the review by Ortigues and Doreau (1995) of the effects of current feeding level on energy requirements of splanchnic tissues and the relevance of these changes for the calculation of energy requirements. In Koong *et al*. (1982), animals fed intensively during the last half of the experimental period had higher requirements than animals fed vice versa, presumably due to the increased weight of metabolically active organs. Genetic differences seem to be important as well. Koong *et al*. (1983) found that genetically obese and lean pigs showed different increases in organ size following the change to intensive feeding in the second half of the experiment. This study, however, may indicate that selection for obesity (as measured by backfat thickness) merely reduced mature size. The observed line differences may disappear if the animals would be compared at similar stage of maturity. Campbell and Taverner (1988) presented another example for line differences in pigs in terms of body composition. This study indicated that the line which was selected for growth performance under ad libitum feeding conditions was less mature at the same weight as compared to the control line; this experiment has frequently been misinterpreted as an example of how breeding could produce an animal with almost unlimited growth potential. As Taylor made clear in 1965, breed or line differences tend to decrease greatly when comparisons are made not at the same weight or age but at the same stage of maturity.

Koong *et al*. (1982) concluded that it would be impractical to express the total metabolic requirements as the sum of individual allometric relationships because of 'prohibitive statistical analyses required'. Instead, they used the following model:

$$FHP = a * W^{b_1 + b_2 * ADG}$$

where *FHP* denotes fasting heat production, *W* is empty body weight and *ADG* denotes average daily gain. The authors pointed out that accuracy of prediction of maintenance requirements could be greatly improved by considering information about the mass of metabolically active organs, such as the liver and gastro-intestinal tract. This view is shared by Webster (1989). Webster (1989) however, pointed out that not just the weight of visceral mass may determine to a large degree FHP (and accordingly, maintenance requirements) but weight of protein tissue in general. This speculation is clearly supported by data reported by Campbell and Taverner (1988). In this study, the intact males of the line with the highest lean proportion had 28.2% higher maintenance requirements for energy but almost identical feed intakes as intact males of the line with the lowest maintenance requirements. However, in this study line and sex differences were partly confounded as the comparison included only castrates of one line. Webster (1989) pointed out that intact males always have higher FHP than castrated males or females.

Koong *et al*. (1985) emphasized that extremely little quantitative information is available about the primary contributors to energy expenditure. That is, currently only minor improvements of the prediction of maintenance energy requirements are possible by fitting more detailed empirical models. The use of a power function of live weight is certainly not correct.

The above discussion showed why it is impossible to separate nutrient requirements for maintenance from those for growth and lactation. This is further illustrated by the review of Webster (1989). He summarized literature results on estimates of thermogenesis in adult cattle originating from long-term experiments. These data illustrate a differences between cattle breeds in terms of fasting heat production. Webster (1989) speculated that these differences are attributable to the greater mass of metabolically active organs in dairy breeds compared with beef breeds. Obviously, differences in FHP are but one component contributing to efficiency of utilization of nutrients.

Do Animals Eat for Energy or for Protein?

Pittroff and Kothmann (1999) found considerable evidence of a link between protein content in the diet and feed intake regulation in ruminants. The new concept of intake regulation proposed by these authors is based on modeling realized performance potential of animals as a function of the interaction between genotypically determined performance potential and available nutrients in the diet. In this model, intake regulation can only be understood as an iterative process, with animals adjusting intake in an attempt to meet the requirements set by their anabolic program. Thus, genotypically specific potential energy demand is modified by environmental conditions and feed composition such that *realized* energy demand results. For growing animals, realized energy demand will be determined by the ability of the diet to supply protein and energy in an amount and balance required for growth. Animals are hypothesized to attempt to meet their protein requirements, with disregard for excess energy. In this context, the question of whether animals regulate intake to meet energy or protein requirements becomes key. Pittroff and Kothmann (1999) concluded from an extensive literature review that neither energy nor protein requirements alone can be defined as the object function for growing animals. Protein availability for synthetic processes is only one element in the regulation of utilization of metabolic fuels. The general principle should be assumed broader: animals eat according to regulation of use of metabolic fuels. For growing animals, the factor of overriding importance seems to be protein availability relative to potential demand levels. However, this contradicts Parks (1982) and Blaxter (1989). Blaxter (1989) stated (p. 283) that rats eat for energy, that is, they adjust their food energy intakes to meet their energy requirements for basal metabolism, muscular work, thermoregulation and growth. If their diets are diluted with an inert material they eat a

greater weight of food and keep constant their total energy intake (Blaxter, 1989). It is quite difficult to follow Blaxter's (1989) rationale because on p. 281-282 he cited experiments (Miller and Payne, 1962; Gurr *et al*., 1980) that actually presented strikingly clear evidence in support of Webster's (1993) contention that growing animals regulate intake in order to meet protein requirements for their anabolic program. It is particularly instructive to review Miller and Payne (1962) and Blaxter's (1989) discussion of this report. Miller and Payne (1962) reported two sets of experiments, one involving a large number of trials with growing rats, and one involving two growing pigs fed on alternating schedules with two types of diet (given to both species of experimental animals): a so-called high-calorie diet which was a normal rat chow diluted with fat and carbohydrates and given ad libitum, and the so-called low-calorie diet which was the normal rat chow fed at restricted amounts so as to maintain body weights. However, the energy density of the diets was not the most distinctive feature but the difference in protein concentration: N percentage of the 'high calorie' diet was one fifth of the 'low calorie' diet. Miller and Payne (1962) observed for the rat trials a substantial variability of calorie intakes, but only a small variation in protein intakes. Rats fed the 'high-calorie' diet ad libitum consumed about 30% more calories at weight constancy compared with the rats fed the 'low calorie' diet.

A similar experiment was conducted with growing pigs, with the difference of the 'high calorie' diet containing 10% of the nitrogen concentration of the 'low calorie' diet. The experiment involved two pigs which were fed both diets on alternating 40 day schedules. The 'pigs' fed the 'high calorie' diet (i.e., the diet with the low protein concentration) consumed on average 5 times more energy than the 'animals' fed the 'low calorie' diet (note that this study was a crossover design with alternating periods).

Blaxter (1989) correctly remarked that Miller and Payne (1962) failed to discuss the implications of a shift in body composition brought about by the two types of diet. Miller and Payne (1962) had ruled out this possibility and instead concluded that the animals fed the 'high calorie' diet exhibited a substantially increased heat production. However, Blaxter (1989) did not discuss the extreme variability of energy intake and remarkable constancy of protein intake observed in the rat experiments. Both growing rats and pigs evidently tried to regulate protein but not energy intake. Gurr *et al*. (1980) explicitly planned their experiments as a replicate and expansion of Miller and Payne (1962). They fed growing pigs to maintain their body weight at approximately 20 kg with two diets differing widely in protein content (26.8% vs. 2.4%). The high protein diet was fed in restricted amounts, the low protein diet was fed ad libitum. Animals fed the low protein diet consumed three times as much feed energy to maintain body weight as those fed the high protein diet. Consequently, they laid down considerable amounts of body fat. Over the experimental period of 42 days, animals reared on the low protein diet (which were fed ad libitum) consumed on average approximately 0.7 kg DM per day (calculated from data given by Gurr *et al*. (1980). This level of intake is considerably below normal ad libitum intake for pigs weighing 20 kg. That is, the low protein diet caused a reduction of feed intake compared with pigs fed diets with normal CP content. Blaxter (1989) took the results of Gurr *et al*. (1980) as proof of his conclusion that increases in heat production do not account for differences in energy consumption at body weight constancy. However, Blaxter (1989) did not discuss the third experiment reported by Gurr *et al*. (1980) with very young pigs raised to and held constant at 6 kg body weight. For these animals, the authors indeed showed considerable differences (40% increase in pigs fed the low protein diet) in heat production. Both calorimetry and energy balance estimation yielded this result. In conjunction with the other experimental results discussed so far, the suggestion that this increase in heat production was caused by the threefold increase of energy intake (compared with the high protein, restricted group) and associated increase in energy expenditure of splanchnic tissues seems to be indicated. The failure of Blaxter (1989) to discuss this observation is possibly understandable if the implication of inappropriateness of the net energy system for energy maintenance requirement calculation is considered.

Parks (1982) also addressed the effects of variable protein content in the diet on growth. His examples showed that the effects of protein content on efficiency of growth followed the law of diminishing returns. Unfortunately, for many of the examples he cited it is unclear whether the experimental animals were fed ad libitum. Parks (1982, p. 215) found that cumulative feed intake of rats (possibly reared on an ad libitum diet, Parks, 1970) did not differ when fed isocaloric diets ranging from 14% to 36.15% protein. He regarded this finding as proof that animals eat for energy and not protein. However, these data are contradictory to his interpretation of the rat feed intake results. This contradiction can be easily resolved when considering that 14% crude protein is not low enough to cause severe growth retardation in rats. That is, even though Parks (1982) could show that feed efficiency increased (with diminishing returns) by increasing CP content beyond 14%, the lower bound of this experiment was not low enough to reveal the mechanisms of feed intake adjustments shown by the experiments of Radcliffe and Webster (1978, 1979). Therefore, Parks (1982) clearly had insufficient evidence for his claim. Parks (1980) seemed to have been aware that there were open questions. He cited another experiment which showed that chicks fed a low protein diet grew excessively fat and criticized the experiments on the effects of protein on growth (including his own) which he used for fitting his Mitscherlich-type equation (growth efficiency vs. protein content of the diet) for not gathering data on body composition.

Summarizing it is concluded for monogastrics:

- Growing animals fed diets which are substantially below their requirements for protein reduce intake and have

drastically reduced protein deposition. Excess energy is deposited in lipid stores.

- As long as there are no adverse effects of severe protein limitation, growing animals increase intake in order to maintain maximum protein deposition at dietary protein levels marginally sufficient for growth. Excess energy has no effect on intake regulation under these conditions. The lower CP level of the diet at which intake begins to be decline is a critical threshold and is likely to differ between species and breeds. It cannot be a constant figure but must vary with age and physiological status.

There is no apparent reason to assume that these findings do not apply to ruminants. In fact, recent data (Pittroff and Keisler, unpublished) show that nutritional regimes designed to differentiate body composition in ewe lambs caused more variation in fat deposition than in protein deposition, although one of the diets was clearly growth limiting at below 8% CP (Figure 1).

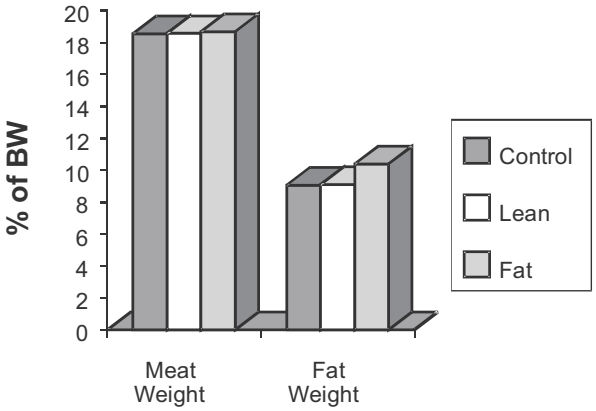

Figure 1. Protein and Fat Deposition in Growing Sheep Line 41 - High Growth (Pittroff *et al*., 1999).

However, intake responses of ruminants to variations in protein-energy ratio of diets are not responses to diet composition alone; they are responses to the ratio of absorbed macro-nutrients. This is clearly illustrated by the intragastric infusion studies conducted by Ørskov *et al*. (1983), Lindberg and Jacobsson (1990) (both studies summarized in Figures 2 and 3) and the intragastric and intraduodenal infusion studies of Black and Tribe (1973) and Black *et al*. (1973). This latter study is particularly interesting because it demonstrated the effects of energy-protein balance on prioritization of nutrient allocation. A high energy level combined with low protein supply led to a decline in wool production in sheep. This type of macro-nutrient imbalance would correspond to the human malnutrition condition *kwashiorkor*, which is associated with a reduced ratio of essential to non-essential amino-acids. Clearly, the substantial changes in metabolism induced by changing the protein-energy ratio of absorbed nutrients would have led to intake adjustments, if the experimental animals would have been under a normal feeding regime. However, in ruminants, the prediction of behavioral adjustments is far more complicated than in monogastrics because of the transformations occurring in the forestomachs. This is exemplified by the study of Redman *et al*. (1980) who studied ruminal, abomasal and ileal N kinetics in growing steers given a roughage base diet and four protein supplements widely differing in rumen degradability. At restricted intake, no significant differences in abomasal bacterial N flow could be found, even though ruminal digestion of organic matter substantially increased when readily degradable N sources were given. Because intake was restricted and a finely chaffed diet was fed, outflow rates were similar for all diets. Therefore, efficiency of microbial synthesis was probably limited by microbial recycling. Animal control of digesta removal, as a function of intake level and rumination activity must be considered when the results of such experiments are assessed, or more importantly, when predictive models are developed. When Redman *et al*. (1980) fed the same di-

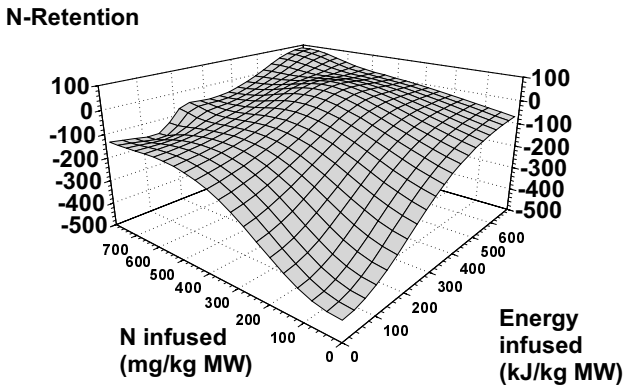

Figure 2. N retention as a function of level of intragastric infusion of N and energy substrates in dairy cows (data from Ørskov *et al*., 1983).

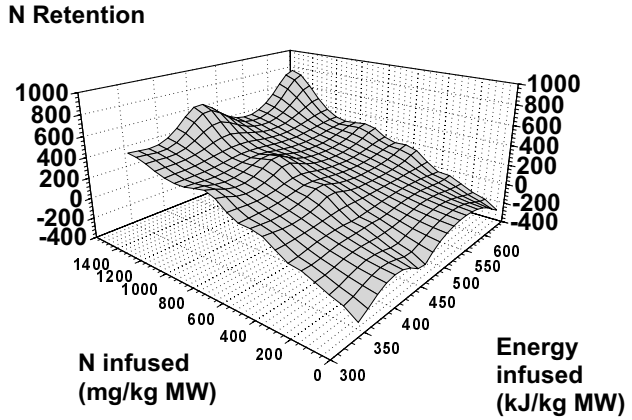

Figure 3. N retention as a function of level of intragastric infusion of N and energy substrates in sheep (data from Lindberg and Jacobsson, 1990).

ets to growing steers ad libitum, drastic intake effects of the protein supplements with corresponding increases in growth rate were observed. The increase in LW gain was up to 137%. Unfortunately, the authors did not collect data on digesta kinetics for this second experiment and did not determine digestibility of the undegradable N supplement (formaldehyde treated casein) that did not produce the highest growth effect.

We suggest that contradictory published data on the intake response of ruminants to varying energy-protein ratios of the diet are most likely the result of:

- imprecise estimate or measurement of absorbed nutrients;
- improper consideration of physiological state of the animal;
- species and breed differences in protein threshold (lower level of absorbed CP at which intake begins to decline).

Discussion and Conclusions

The preceding literature review intends to motivate a discussion on research priorities. It is clear that the physiological processes responsible for phenotypic variation among livestock cannot be quantified without appropriate prediction of nutrient fluxes. This means, nutrient intake must be understood. Research on intake regulation isolated from explicit consideration and quantification of potential and realized energy demand will not yield progress. Likewise, as long as there is no link to nutrient intake, experimental work on growth, lactation, and maintenance requirements cannot be expected to provide data meaningful for the construction of nutrient supply driven simulation models of animal production systems.

Debates as to what constitutes a proper mathematical form of a quantitative model of growth, or lactation are not helpful as long as no quantitative representation of the underlying performance potential, and its modification by available nutrients is included. Retrospective curve fitting produces little, if any biological insight.

The preceding review suggests that the amount of effort spent on purely 'curve fitting' exercises, in particular for growth and lactation, appears to be vastly disproportional to the effort required for conceptual understanding. We argue that conceptual understanding is the prerequisite for prediction.

On the other hand, attempts at dynamic description of lower level processes, for example detailed growth or lactation models, suffer from being uncoupled from the basic process of nutrient acquisition. They must fall back on empirical adjustments for nutrient fluxes, not unfittingly named 'fudge factors' in modeler's lingo, that threaten to compromise validation and transferability.

An interesting perspective on the modeling of maintenance requirements would be a proposal to not consider them at all (Pittroff, 1997; Oltjen and Sainz, 2001). This would be possible if the dynamics of all relevant body pools would be described in component models for growth and lactation. By necessity, this implies the abandonment of factorial approaches to the quantification of nutrient requirements of farm animals.

It seems to be further noteworthy that many rather fundamental issues in the understanding of the biology relevant for nutrient supply driven simulation models remain unresolved. The arguably most important ones are the relationship between rate of maturing and rate of fattening, the relationship between growth and onset of puberty, nutrient partitioning effects in lactation, and the catabolic response in relation to size of energy deficit.

These very basic knowledge gaps shed massive doubt on the capability of current genomics approaches to substantially contribute to the improvement of livestock production. Currently, the term 'functional genomics' is en vogue; we fail to see the focus on function, emphasizing that 'function' cannot be reduced to gene products. It is essential to point out that these knowledge gaps became apparent in attempts to develop functional, mechanistic, nutrient supply driven models of livestock systems, not because of advances in proteomics. Hence, there is a role of modeling of livestock systems in the advancement of animal science whose importance cannot be overstated.

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