

Simulating the partitioning of dietary amino acids: New directions

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ABSTRACT: In developing a mathematical model to allow prediction of growth in mammals, the simulation of amino acid metabolism is of particular importance because the predicted rate of protein deposition has a disproportionate influence on predicted body mass. In reality, the absorption and metabolism of amino acids in mammals is complex and highly integrated with continuous flux within and between body cells. To model amino acid transactions, however, a simplified construct of metabolism describing discrete physiological and metabolic processes must be developed. In the construct discussed here, a distinction is made between maintenance processes and those processes associated with growth. Growth is viewed as a function of nutrient deposition and support costs directly related to nutrient

deposition. Several processes are emphasized and discussed, including food and amino acid intake, amino acid absorption, amino acid losses at maintenance, net protein deposition, inevitable amino acid catabolism, gut endogenous amino acid loss correlated with food intake, the turnover of body protein associated with new protein synthesis, the synthesis of non-amino acid-, non-protein nitrogen-containing compounds and preferential amino acid catabolism. The modeling of animal growth has become mainstream over the last two decades and models are being used increasingly in research, teaching, and in commercial practice. As models become more causal and less empirical, their validity and utility will be enhanced.

Key Words: Amino Acids, Models, Pig, Protein, Simulation

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Introduction

Over the last three decades, considerable attention has been given by animal scientists to the development of causally based quantitative models describing the absorption and subsequent utilization of amino acids and other nutrients during animal growth. Such models are “representations” of the real system (and thus conceptual), and there are therefore different, arguably equally acceptable, approaches or “views.” Importantly, such models allow rigorous hypothesis testing around what is a relatively complex and highly interactive system. It is by the process of refutation and the formulation of new hypotheses to replace the older, less adequate ideas that modeling advances.

In this paper, a simplified conceptual framework is given that has been found useful for describing protein and amino acid transactions in the growing pig. The framework is discussed in the light of new directions that might be taken by modelers to increase the validity or application of their models. It is important to bear in mind, however, that the minimalist approach is an

inherent principle to modeling, and models should not be expanded simply for the sake of greater complexity, but rather should only be further developed if this will lead to a meaningfully enhanced causal understanding of the phenomenon.

Background to Models

Some of the simplest amino acid models developed were the early static factorial models, which summed the metabolic losses of absorbed amino acids (often bulked together into a crude estimate of a “maintenance” requirement) and the amino acids deposited in new proteinaceous tissue, and then corrected the sum to take into account the inefficiency of utilization of absorbed amino acids. Such models were particularly popular in poultry nutrition (Hurwitz and Bornstein, 1973; Smith, 1978; Hurwitz et al., 1983), and an example in pig nutrition is the early work of Whittemore and Fawcett (1974). These early models paved the way for the development of more sophisticated models that take into account not only the amino acids, but also the nonprotein dietary energy and the interaction between amino acids and ME. These biological models include parameters affected by the nutritional history of the growing animal and incorporate adaptive control processes and impose limits on physiological and biochemi-

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Table 1. Biological processes underlying amino acid utilization in the growing pig

1. Ingestion of dietary amino acids	
2. Amino acid absorption	
3. Maintenance ^a	<ul style="list-style-type: none"> • Turnover of body protein • Integumental amino acid loss • Gut endogenous amino acid loss • Synthesis of non-protein nitrogen-containing compounds • Urinary amino acid losses
4. Growth	<p>Body protein accretion</p> <p>Inevitable amino acid catabolism</p> <p>Gut endogenous amino acid loss</p> <p>Turnover of body protein</p> <p>Synthesis of non-protein nitrogen-containing compounds</p> <p>Preferential amino acid catabolism</p>
Support costs	<p>Inevitable amino acid catabolism</p> <p>Gut endogenous amino acid loss</p> <p>Turnover of body protein</p> <p>Synthesis of non-protein nitrogen-containing compounds</p>

^aA distinction is made between basal or maintenance processes (i.e., those occurring in the hypothetical state whereby body tissue is neither gained nor lost) and those processes associated with the accretion of new body tissue. The rate of a process at maintenance is defined as that rate commensurate with a daily food intake under which body weight is neither gained nor lost. Rates of the processes during growth are variable. It should be noted that for most of the metabolic processes, there is actually a natural continuum between maintenance and growth and that the distinction between states is arbitrary and reliant upon definition.

cal processes. Elements of these models are often causally based or deductive, and thus give considerable insight to the complex system being modeled.

A number of models simulating the uptake, metabolism, and partitioning of dietary nutrients in the growing pig have been developed (Whittemore and Fawcett, 1976; Moughan, 1981; Phillips and MacHardy, 1982; Tess, 1983; Whittemore, 1983; Moughan and Smith, 1984; Black et al., 1986; Stombaugh and Oko, 1980; Emmans, 1986; Moughan et al., 1987; Watt et al., 1987; Burlacu et al., 1988; Pettigrew et al., 1989; Pomar et al., 1991; Bridges et al., 1992; Ferguson et al., 1994; de Lange, 1995; Larduet and Savon, 1995; Knap, 1996; van Milgen et al., 2000).

As the field of pig growth modeling has developed, there has been a tendency for models to become more causal (less empirically based) and to further differentiate among the dietary nutrients and their ultimate metabolic fates (Boisen and Verstegen, 2000; Birkett and de Lange, 2001). This requires a detailed modeling of the ingestion, digestion, absorption and metabolism of amino acids.

A Framework for the Simulation of Amino Acid Metabolism

Clearly, the absorption and metabolism of amino acids in mammals is complex and highly integrated, with continuous flux within and between body cells. It is useful, however, and inherently necessary when constructing a model of metabolism, to consider amino acid metabolism as several discrete physiological processes (Table 1) that underlie or are causative to amino acid utilization. In the scheme presented here, the classical distinction is made between the “maintenance” or “basal” processes and those associated with growth. In

reality, however, these multiple sets of processes are highly interrelated. Nevertheless, it is considered useful to conceptualize and represent overall metabolism in two parts: maintenance and growth. At zero nitrogen retention, there are still costs associated with body protein metabolism, and these are the classical “basal” or “maintenance” costs. With positive nitrogen retention, there are extra costs incurred associated with maintaining the proteinaceous body tissues. These are referred to here as support costs for growth.

Much has been written recently about these various maintenance and growth processes (Moughan, 1999; Black, 2000; Whittemore 2001a; Moughan and Fuller, 2002) and the present contribution will not cover the same ground. Rather, possible new directions for modeling these components will be emphasized.

Modeling the Component Processes

Simulating Voluntary Food Intake and the Ingestion of Amino Acids

The amount of an amino acid ingested is a function of the quantity of food ingested and the amino acid composition of that food. The approaches and limitations to modeling voluntary food intake in the pig have been discussed (Emmans, 1995; Kyriazakis and Emmans, 1999; Black, 2000; Whittemore et al., 2001a).

A decision must be made by the modeler as to which of the dietary amino acids should be explicitly represented in the model. The usual approach has been to include selected dietary essential amino acids or all the traditionally considered dietary essential amino acids and the dietary nonessential amino acid component. However, it is becoming increasingly apparent (Ball et al., 1986; Fuller, 1994; Reeds and Beckett, 1996) that

under certain conditions, amino acids traditionally considered to be dietary nonessential can become rate-limiting, and the concept of conditional essentiality has been introduced. Thus, in some cases (e.g., modeling amino acid transactions in the young pig), it may be important to model the metabolism of amino acids, such as proline and arginine, explicitly. Particularly in relation to energetics, it may be useful to model the gut utilization of dietary glutamic acid, and the subsequent systemic body cell synthesis of glutamate, which is energy demanding (Reeds et al., 1998). For young animals, it may also be useful to directly model the interconversions of methionine to cysteine and phenylalanine to tyrosine. Further, in some models, the uptake and metabolism of lysine have been emphasized with other amino acids described by reference to an "ideal" amino acid balance. However, there is no single "ideal" dietary amino acid balance with the balance of amino acids associated with complete utilization by the animal being influenced by numerous diet and animal factors (Black and Davies, 1991).

Amino Acid Absorption

Most models of pig growth have relied upon the use of empirically derived amino acid digestibility coefficients for the constituent dietary ingredients to predict the uptake of dietary amino acids from the gut. This approach is static and does not allow for temporal dynamics to be simulated. Bastianelli and Sauvant (1995) and Rivest et al. (2000) have recently taken an alternative approach, where an attempt has been made to model the mechanisms known to underlie the digestive and absorptive processes. This allows the varying rate of flux of amino acids following a meal to be simulated and potentially allows description of factors (both dietary and animal) known to affect amino acid digestibility. The limitation of empirically derived amino acid digestibility coefficients in not accounting for the kinetics of amino acid absorption has been discussed by Rerat (1990), who correctly emphasizes the marked influence on efficiency of protein synthesis consequent upon synchronization of the dietary amino acid supply to the sites of protein synthesis, as well as the synchronization of the supply of amino acids and nonamino energy-supplying compounds. The degree of amino acid utilization is a function of both the extent of digestion and absorption of nutrients and the timeliness of the absorption. Thus, model components that can predict the kinetics of amino acid absorption stand to improve the accuracy of prediction of pig growth. There is an opportunity for such models to be causal, based around the physical laws governing fluid flow, rates of hydrolytic breakdown, and absorption related to gut surface area.

Whatever approach is used, it is still necessary to apply some factor, to account for food-related differences in the susceptibility to hydrolysis of molecular linkages. Usually, these factors will be based on differences in ileal amino acid digestibility (Moughan, 1995).

However, compelling evidence has recently been published from experiments using stable isotopes that essential amino acids are synthesized by gut microbes and are then absorbed (Fuller and Reeds, 1998; Metges, 2000). Such synthesis may make a quantitatively important contribution to amino acid supply; if so, it needs to be modeled. Studies are urgently needed to define what net contribution may accrue from intestinal bacterial proteolysis and amino acid synthesis and catabolism. If, overall, there is either a practically significant net synthesis or catabolism of amino acids in the upper digestive tract, then either digestibility coefficients will need to be refined or a description of the metabolism of the intestinal microflora be incorporated within models of the digestive process.

A final point is that when using ileal amino acid digestibility coefficients in modeling amino acid transactions, cognizance needs to be given to how apparent, true, and real coefficients (Boisen and Moughan, 1996b) should be applied, and in the case of processed proteins, the digestibility of reactive lysine should be incorporated in the model (Moughan, 2002) rather than conventional estimates of digestibility.

Maintenance

In the hypothetical state whereby a growing pig is neither gaining nor losing net body protein, metabolic processes are occurring that lead to the loss of proteinaceous material from the body, which must in turn be replaced by the diet. These processes are: 1) losses of amino acids via skin and hair; 2) losses of nitrogen of amino acid origin in urine reflecting inefficiency in the process of body protein turnover; 3) basal gut endogenous amino acid losses (mainly mucus, bile, desquamated cells); 4) the irreversible loss of amino acids in synthesizing essential non-amino acid nitrogenous metabolites (e.g., creatinine); 5) irreversible chemical alterations of amino acids (e.g., lysine to hydroxylysine); and 6) the loss of free amino acids in the urine.

The latter three processes are considered to be quantitatively minor at maintenance. The first three processes, however, are more important (Moughan, 1999) and need to be incorporated in models. Although in total, and for a rapidly growing animal, the maintenance amino acid requirement is only a small proportion (about 10% or less) of the total daily amino acid requirement, certain dietary essential amino acids are required disproportionately (e.g., cysteine loss in skin and hair, threonine loss in gut endogenous protein are relatively high).

Growth

For a rapidly growing animal, the actual net retention of amino acids into body protein explains a large part of the dietary amino acid need. However, the numerous support costs associated with this net protein accretion are certainly not insignificant and merit close

attention in the modeling of amino acid partitioning. It is possible to model amino acid retention as the net outcome of the two fundamental processes—protein synthesis and protein degradation—and this has been attempted (Pomar et al., 1991). Such an approach also allows protein retention to be related to cellular levels of DNA and messenger RNA. Although this approach has its attractions, protein synthesis and degradation have proven difficult to measure empirically, and sound data for modeling purposes are lacking. It has been more common to determine net rates of whole body protein retention under optimal dietary and environmental conditions as estimates of biologically maximal rates of body protein retention (**Pdmax**) for the particular type of animal. The intrinsic upper limit to whole body protein retention is an important constraint on growth since the cell has a finite capacity for protein synthesis and is unable to store free amino acids for later use. If, after a meal, the uptake of balanced amino acids required for protein synthesis exceeds the animal's capacity for protein synthesis, surplus amino acids are deaminated and the carbon skeletons eventually degraded. The Pdmax is influenced by genotype (breed and strain), gender, and age, and mean values reported in the literature range from as low as 90 g/d to values exceeding 200 g/d (Whittemore, 1983; Campbell, 1985). Recently, very thorough work has been conducted to determine upper limits to lean retention in North American pig populations (Schinckel, 1999). As animals are grown over progressively wider live weight ranges, it has become necessary to model the effect of age on Pdmax (Moughan, 1999; Whittemore et al., 2001b). It is also apparent that under practical farming conditions, pigs may not achieve the Pdmax value for their strain/breed as determined under breeding station or research center conditions, presumably because of effects due to factors such as subclinical disease, thermal environment, and social conditions (Baker and Johnson, 1999; Black et al., 1999; Burrin et al., 2001). For this reason the term "operational Pdmax" has been coined (Moughan et al., 1995), and operational Pdmax values have been determined on-farm (Morel et al., 1993; Moughan et al., 1995). The support costs for net protein accretion arise from processes such as endogenous gut amino acid losses, body protein turnover, inevitable amino acid catabolism, and the use of amino acids to synthesize essential non-amino acid nitrogenous compounds or in the irreversible structural alteration of amino acids. These support processes tend to be directly related to the rate of body protein retention, with higher net retentions incurring greater costs. There is extensive literature on gut endogenous amino acid losses (Boisen and Moughan, 1996a), with the method of determination being a central issue (Hodgkinson and Moughan, 2000). The traditional protein-free approach to determining gut endogenous amino acids has been discredited, and alternative approaches have been applied to yield more meaningful data. Gut amino acid losses are related to food dry matter intake and are

influenced by dietary composition, especially the amount and type of plant nonstarch polysaccharides (fiber) and antinutritional factors (e.g., tannins, lectins, trypsin inhibitors). It is now possible to model some of these effects directly. Indeed, the major (and disproportionate relative to its size) impact that the gut has on both energy and protein metabolism in growing animals suggests that gut turnover and growth may merit being modeled directly.

The study of Bikker et al. (1994) demonstrates an effect of both dietary energy and protein intakes on the proportion of whole body protein associated with either the carcass or the organs (including blood). Further, and given that the carcass component (and indeed components of the carcass) and the various organs have quite different amino acid compositions, the amino acid composition of whole body protein may vary with the level of nutrition and rate of growth, and thus there may be a case for modeling the growth of different body parts separately. It may also be useful to directly and explicitly model the degradation of amino acids in the hindgut with subsequent uptake of ammonia. Considerable quantities of ammonia are absorbed in the hindgut (confer ileal/faecal digestibility coefficient differences) and are synthesized to urea. Some of this urea is recycled into the gut, but much of the synthesized urea is excreted in the urine. These processes are important both energetically and in terms of nitrogen loss.

The use of amino acids as precursors for the synthesis of other non-amino acid (or irreversibly altered) nitrogenous compounds may also be quantitatively important in some cases (Reeds, 1988; Fuller, 1994) and should be considered for explicit representation in models. By way of example, the gut may use large amounts of cysteine to synthesize mucins and glutathione (Burrin et al., 2001), which may in turn lead to a metabolic demand for methionine. In fact, methionine is involved as a methyl donor in several different pathways. With respect to gut cysteine demand, it is interesting to note recent findings from the University of Alberta (Shoveller et al., 2000) of a 35% lower requirement of piglets for methionine under total parenteral nutrition as opposed to enteral feeding.

Body protein turnover increases with the rate of net body protein accretion (Milligan and Summers, 1986) and has an important potential impact upon nitrogen utilization since body protein turnover is unlikely to be completely efficient. The turnover of different body protein depots has been directly simulated in the recent work from Knap and Schrama (1996).

The concept of inevitable catabolism refers to the catabolism of the dietary first-limiting amino acid, which occurs during growth quite unrelated to energetic need, and is simply due to the existence of active catabolic enzyme systems in the cell. The recent work of Peter Reeds and his group has shed new light on the phenomenon of inevitable catabolism, in respect of first-pass gut metabolism. It is now clear (Fuller and Reeds, 1998; Burrin et al., 2001; Stoll et al., 1999) that the gut

tissues account for a considerable degree of the overall amino acid metabolism. Also, intriguing new data point to an important role for the enterocyte in the catabolism (oxidation) of absorbed dietary amino acids, including dietary essential amino acids and the often dietary first-limiting amino acid, lysine. The observed substantial degree of “first-pass” metabolism by the gut tissue may explain an important fraction of “inevitable catabolism.” In a pivotal study by Stoll et al. (1998), approximately one third of dietary lysine intake was metabolized by the gut tissues in 28-d-old pigs, with only 18% of the first-pass metabolism being accounted for by gut protein synthesis (i.e., incorporation of lysine in mucosal tissue). This infers that there is a considerable degree of first-pass lysine catabolism and that the enterocyte may be an important site for the catabolism of lysine and other dietary essential amino acids. It may be that the enterocyte has a specific catabolic requirement for certain dietary essential amino acids. This is an important new finding. From the results of a subsequent study (van Goudoever et al., 2000), it appears that the first-pass utilization of lysine is influenced by nutritional state. For 4-wk-old pigs given a high-protein (23% crude protein) diet, there was considerable lysine metabolism by the gut, but lysine use by the portal-drained viscera was derived almost entirely from arterial input. The relatively low amount of dietary lysine used in the first-pass was almost entirely oxidized (representing one third of whole body lysine oxidation or about 5% of dietary lysine intake). When a low-protein diet (about 9% crude protein) was given, overall lysine metabolism was not affected, but now, and in contrast to the high protein finding, both dietary and arterial lysine were used by the portal-drained viscera in nearly equal amounts, and intestinal lysine oxidation was suppressed. An overview of both studies (Stoll et al., 1998; van Goudoever et al., 2000) suggests that gut tissue lysine metabolism in the pig may be quantitatively important and compartmentalized, and it may be influenced by the level of nutrition, with increased catabolism accompanying higher levels of amino acid uptake. The data suggest a high obligatory visceral need for lysine, with protein intake influencing gut lysine catabolism. The results of the two studies discussed above are not entirely consistent with each other and further confirmatory studies are needed. However, the studies do suggest an important role for the gut tissues in lysine (and other dietary essential amino acids) metabolism and catabolism. This is further evidence supporting the need to model the metabolic transactions of the gut separately.

A common approach in developing pig growth models has been to describe gut endogenous amino acid losses directly, but to lump the other support costs together into a single measure of “catabolic” losses. The magnitude of such losses can be determined experimentally (after correcting for maintenance losses) by determining the difference between the amount of the absorbed first-limiting amino acid and the amount of that amino

acid deposited in tissue for animals fed protein below their maximal rate of retention and given a high-energy diet (so that nonprotein energy sources are not limiting). Sound empirical data to thus describe the efficiency of utilization of the first-limiting amino acid are lacking, and this is a major weakness in modeling amino acid partitioning. Recent studies, however, have addressed this issue and useful information is beginning to emerge (Moughan, 1999; Edwards et al., 1999).

Kees de Lange and coworkers at the University of Guelph have recently conducted a series of carefully controlled serial slaughter studies that provide useful new information. Growing pigs (mean live weight of 50 to 60 kg) were fed a highly digestible casein and cornstarch diet, wherein a prescribed dietary essential amino acid was clearly first-limiting. Animals were screened, in a preliminary nitrogen balance study, for their upper limits to body protein retention (P_{dmax}) to allow selection of a cohort of animals with similar P_{dmax} . Feeding levels were varied such that the first-limiting amino acid was supplied at set proportions of the amount of the amino acid needed to meet the estimated requirement to support P_{dmax} . Metabolizable energy intake exceeded the determined requirement to support P_{dmax} . In the first study (Möhn et al., 2000) in which lysine was the first-limiting amino acid, the marginal efficiency of using absorbed (true ileal digestible reactive lysine) lysine for protein deposition was 0.75 and was not affected by ME intake or available lysine intake. Thus, approximately 25% of the absorbed available lysine was unaccounted for, presumably largely lost to catabolism. In a further study in which threonine was the first-limiting amino acid (de Lange et al., 2001), a marginal efficiency of utilization of absorbed threonine of close to 75% was determined, and the efficiency of utilization was not affected by live weight, but there was some indication that threonine utilization was highest at the lowest threonine intake. Threonine disappearance was relatively constant at 23.7% of available threonine intake, when threonine intake varied between 70 and 100% of the threonine requirement to support P_{dmax} . Conversely, Reijmers et al. (2000) reported that the efficiency of utilization of available methionine plus cysteine (above maintenance), decreased with increasing methionine plus cysteine intake from a high of 90% (60% of P_{dmax}) to a low of 71% (100% of P_{dmax}).

Whittemore et al. (2001b) have attempted to provide a basis for inefficiency in the utilization of the dietary first-limiting amino acid by relating the efficiency of utilization of ideal protein to protein (amino acid) losses associated with body protein turnover, which are considered to increase with the rate of protein retention. Although such an approach undoubtedly explains part of the inefficiency of utilization of the absorbed first-limiting amino acid, losses from turnover may not be the only or the most important source of inefficiency. For example, there may be, as discussed above, quite substantial “first-pass” catabolism of dietary essential

amino acids by the gut enterocytes. There is also likely to be some degree of catabolism (inevitable) of absorbed dietary amino acids by other body cells. However, losses due to body protein turnover are likely an important contribution to overall inefficiency of utilization. As more information becomes available, it would appear possible to model the inefficiency of dietary first-limiting amino acid utilization based on a component related to inevitable catabolism plus other inevitable losses, and to a possibly more variable component related to rate of body protein turnover.

The processes leading to postabsorptive inefficiency of utilization of the absorbed first-limiting amino acid collectively account for a loss of around 20 to 25% of the absorbed first-limiting amino acid. The efficiency of utilization may vary among the different absorbed amino acids and may be affected by the amount of the absorbed amino acid relative to the amount required for maximal body protein synthesis (Moughan, 1989; Seve and Henry, 1995), though this is a contentious issue (Möhn et al., 2000; de Lange et al., 2001).

There is a well-understood nutritional interaction between dietary protein and energy (nonprotein energy supplying nutrients) stemming from the fact that protein synthesis is an energy-demanding process with amino acids being both substrates for protein synthesis and compounds capable of yielding energy. In situations where the nonprotein fraction of the diet is insufficient to yield the required ATP, then amino acids will be oxidized to supply ATP. This phenomenon needs to be modeled. The first approach is to use empirically derived functions to describe the relationship between protein retention and energy intake when overall dietary energy is limiting, and thus predict the rate of body protein retention, with lipid retention being calculated as a residual function. An alternative approach is to assume that there can be long-run zero or negative body lipid retention in support of protein accretion and to then calculate energy demands and yields based on these premises. Some researchers (Whittemore and Fawcett, 1976) have assumed that there is a minimal daily rate of body lipid retention (Ld_{minimum}) that must be supported, whereas others (e.g., Moughan et al., 1987) have assumed that other than under conditions of severe starvation (whereby particular adaptations occur), the body has a desired minimal lipid content, and thus have modeled a minimal level of body lipid (Lt_{minimum}). The concept of a minimal whole body lipid to whole body protein ratio ($Lt:Pt$) minimum has also been advanced by Whittemore (1995). Emmans and Kyriazakis (1997) have discussed a further approach to the problem, which also has some biological appeal. They have proposed that the net material efficiency (i.e., the slope of protein retention on protein supply above maintenance) of using ideal protein for protein retention (**ep**) is directly proportional to the ratio of metabolizable energy to digestible crude protein of the food, up to a critical value at which it attains its maximal value ep^* . The value ep^* is analogous to "inevitable

catabolism." The values for ep and ep^* are assumed (based on some experimental evidence) to be constant across genotypes and for pigs of different liveweight. These empirical and deductive approaches have been recently reviewed (Emmans and Kyriazakis, 1997; Moughan, 1999).

Conclusion

The modeling of animal growth has become mainstream over the last two decades, and important causal theories of growth have been developed and demonstrated to be useful in practice. At the same time, there has been considerable progress made in our understanding of amino acid digestion and postabsorptive metabolism. The challenge for modelers is to use this new information to further develop their models of growth to enhance validity and applicability. It seems likely that the next generation of pig growth models will model the biochemical utilization of individual nutrients (including the individual amino acids) more closely and will begin to directly model amino acid transactions in the gut and other subcomponents of total body protein (e.g., liver, connective tissue vs. muscle). The modeling of protein turnover in these subcomponents, and the description of a hierarchy of amino acid use for protein deposition will become increasingly important.

Implications

The mathematical modeling of animal growth has become an important tool in animal science research and teaching. Moreover, the use of animal growth models in commercial practice offers, among other applications, a new and situation-specific approach to nutrient requirement estimation and to the development of feeding regimens. As basic knowledge is developed on the growth and metabolism of the pig, pig growth models will become causal, thus having enhanced validity and utility.

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