Variation in maintenance requirements of growing pigs in relation to body composition. A simulation study.

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Variation in maintenance requirements of growing pigs in relation to body composition. A simulation study.

Pieter W. Knap

Proefschrift

ter verkrijging van de graad van doctor
op gezag van de rector magnificus
van Wageningen Universiteit,
dr C.M. Karssen,
in het openbaar te verdedigen
op maandag 29 mei 2000
des namiddags te vier uur in de Aula.

CIP-DATA KONINKLIJKE BIBLIOTHEEK, DEN HAAG

Knap, Pieter W.

Variation in maintenance requirements of growing pigs in relation to body composition. A simulation study / Pieter W. Knap, – [S.I. : s.n.]
Thesis Wageningen. – With ref. – With summary in Dutch.
ISBN 90-5808-170-2
Subject headings: maintenance requirements / body composition; variation; simulation; pigs

Knap, P.W. (2000) Variation in maintenance requirements of growing pigs in relation to body composition. A simulation study. [Variatie van de onderhoudsbehoeften van groeiende varkens in afhankelijkheid van hun lichaamssamenstelling. Een simulatiestudie.]

Existing dynamic models for the simulation of growth metabolism in pigs were extended with routines to predict the energy requirements of protein turnover and thermoregulation. Protein turnover was modeled by distinguishing six body protein pools with different turnover rates and different growth curves. Thermoregulation was modeled by assessing minimum and maximum heat loss, and heat production, deciding by comparison of these whether the pig is cold or hot, and taking appropriate metabolic action. Model output compared satisfactorily with independent data. Pig populations were modeled by stochastic simulation, imposing between-animal variation on growth potential parameters and therefore on body (growth) composition. Because protein turnover, and heat production and thermal insulation, in the model depend on body (growth) composition, between-animal variation was generated in the associated energy requirements. This leads to variation in maintenance requirements as a function of variation in body composition. The simulated output was analysed to provide an answer on the question "to what extent can differences in maintenance requirements be attributed to differing proportions of the different organs and tissues of the body, each having different metabolic rates" ? The conclusion from this analysis is that the contribution of variation in body (growth) composition to the variation of total maintenance requirements in growing pigs is very limited, probably less than 10 % of the total variance. Experimental verification of this conclusion is desirable; the design of the required experiments is discussed, making use of the simulation results.

PhD thesis, Wageningen Agricultural University (Department of Animal Sciences), Wageningen Institute of Animal Sciences (WIAS), Postbus 338, 6700 AH Wageningen, The Netherlands.

Stellingen

1. De energetische onderhoudsbehoefte van gezonde groeiende varkens vertoont een fenotypische variatiecoëfficiënt van ongeveer 10 %. Dit proefschrift

2. De fenotypische variantie van de onderhoudsbehoefte van gezonde groeiende varkens is voor ongeveer 30 % van genetische aard, en hangt voor minder dan 10 % samen met de lichaams(groei)samenstelling. Dit proefschrift

3. De volwassen lichaamseiwitmassa van slachtvarkenvaderlijnen is sinds de jaren '60 niet noemenswaard veranderd; hun volwassen lichaamsvetmassa is met een factor drie of vier verminderd. Dit proefschrift

4. Het modelleren van dierlijke groei zou meer moeten worden gericht op een gedetailleerde beschrijving van de onderhoudsprocessen dan op "nieuwe" regels voor potentiële eiwit- en vet-aanzet. Dit proefschrift

5. Gepubliceerde resultaten van experimenten aan varkens kunnen sterk aan algemene bruikbaarheid winnen als het (geno)type van de dieren wordt gedocumenteerd. Een bescheiden eerste aanzet daartoe zou zijn het vermelden van de sexe, de leeftijd en de spekdikte bij een bepaald gewicht.

6. De acceptatie van nuttige strategieën in de veefokkerij wordt vaak vertraagd doordat men *case studies* van alom gerespecteerde wetenschappers interpreteert als algemeen geldend, en nalaat de sommen over te doen met de eigen kengetallen. In de ernstigste gevallen leidt dit tot goeroeïsme.
C. Smith (1962) *Animal Production* 6:337-344
C.R. Henderson (1973) *Animal Breeding and Genetics Symposium in honor of J.L. Lush.* ASAS/ADSA, Champaign; pp. 10-41
J.J. Rutledge (1980) *Journal of Animal Science* 51:871-874
B.W. Kennedy *et al.* (1993) *Journal of Animal Science* 71:3239-3250
K. Meyer (1997) *Proceedings of the Association for Advancement of Animal Breeding and Genetics* 12:470-473

7. *They are little machines for converting protein into fat. This is the original Mediterranean hog, before genetic engineers began crossing the beast with oriental pigs to get bigger and fatter carcasses. It still lives a good life for a pig. The British hog at nine months is too fat to walk, weighing in at 200 kg.*[1]
Een bedrijfssector die aanleiding geeft tot het publiceren van dit soort onsamenhangende flauwekul verdient het niet om door de samenleving serieus te worden genomen.
1: G. MacDonogh (1997) *Financial Times* 02-02-1997; p. 15

8. De algemene conclusie van dit proefschrift is *Luiting*[1] *Was Right*, afgekort *LWR*. De logische extrapolatie is *LIAR*.[2]
1: P. Luiting (1991) *The use of feed consumption data for breeding of laying hens.* Proefschrift LU Wageningen
2: G.C. Emmans & M.W.A. Verstegen (1997) Personal communication

9. De opkomst van moleculaire genetica als werktuig voor het meten van evolutie is een zegen voor de traditionele wetenschappen, want het leidt tot nieuwe sexy paradigma's.[1] Zo blijken eikelwormen opeens nauwer verwant te zijn aan zeekomkommers dan aan mensen.[2]
1: naar S.B. Hodges & L.L. Poling (1999) *Science* 283:998-1001
2: A. Adoutte *et al.* (1999) *Trends in Genetics* 15(3):104-108

10. Europese eenwording vereist dat iedere inwoner van de EU tweetalig wordt opgevoed met voor iedereen dezelfde tweede taal, en dat vrijwel iedereen het daarmee even moeilijk krijgt; de beste keus voor die tweede taal is daarom het Baskisch.

11. Je eerste perfecte vanillepudding kost minstens zoveel moeite als je eerste gepubliceerde tijdschriftartikel.

Stellingen bij het proefschrift

Variation in maintenance requirements of growing pigs in relation to body composition. A simulation study.

Pieter W. Knap, Wageningen, 29 mei 2000.

Zij die [...] een proefschrift schrijven [zijn] meer te beklagen dan te benijden, daar ze zich eindeloos aftobben. Ze voegen toe, veranderen, schrappen, herstellen weer, herzien, werken het weer geheel en al om, laten het graag anderen zien, houden het negen jaar in portefeuille en zijn nooit tevreden met het resultaat. De beloning die ze er tenslotte voor krijgen [...] is wel heel duur betaald met al hun zwoegen, zweten en gebrek aan [...] slaap. Voeg hierbij nog dat dit alles gaat ten koste van hun gezondheid, dat ze daardoor humeurig, lelijk, bijziende of zelfs blind worden, tot armoede vervallen, bij ieder uit de gunst zijn, dat ze alle genoegens moeten verzaken, dat ze vóór hun tijd oud zijn, ontijdig sterven en wat dies meer zij. Doch al deze opofferingen getroosten zij zich gaarne om de goedkeuring weg te dragen van één of twee geleerde boekenwurmen.

Desiderius Erasmus (1515) *De lof der zotheid* (vert. A. Dirkzwager & A.C. Nielson, 1971). Paris-Manteau, Amsterdam; pp. 189-191.

Preface

This thesis was conceived in April 1978, when I was discussing my choice of subjects with Carel Richter, my MSc coach at Wageningen Agricultural University. Carel approved of my plans for majors in Aquaculture and in Animal Breeding, but was less enthusiastic about a minor in Information Technology. He suggested to go for Theoretical Production Ecology instead: "They work a lot with computers too, they simulate crops and ecosystems and so on". People at the TPE department seemed to like my ideas, but that I would want to simulate *animals* was clearly a bit bizarre; I had to appoint an extra supervisor to deal with the 'animal aspects'. That supervisor was found in the person of Aren van Es, by whom I got grilled every Thursday afternoon from September to December about two growth models that had just been published, and about their shortcomings. Between those Thursdays, Jan Goudriaan of TPE took care of the modeling aspects; at the end, my thesis was titled *Comments on two models of animal growth and energy metabolism*, and much later I discovered that most of those comments had been unjustified (no wonder, when the punchcards with your program code spill over the floor every now and then). But anyway, simulation is a strongly addictive thing. Especially when you try to model growing pigs.

So it was not so strange to become involved with the Wageningen/Guelph postgraduate course on *Modeling of growth in the pig*, where I presented my ideas about stochastic simulation of growing pigs to groups of innocent nutritionists and physiologists on a Friday morning in May, each year from 1992 to 1996. That way I got to know John Black, Gerry Emmans, Cees de Lange, Paul Moughan and Jim Pettigrew (I had met Paul five years earlier, when Martin Verstegen had borrowed me to coordinate a course that Paul came to give about his Massey Model). The first two chapters of this thesis have grown from those five Friday mornings and the subsequent dinner parties, in combination with Johan Schrama's MSc thesis that Martin made me co-supervise in 1989. Ella Luiting, with whom I had built up a colourful array of personal and professional relationships in the meantime, exploited one of those dinner parties to talk Martin and Gerry into volunteering as promotors for this PhD project. That was very useful, for the thing tended to lie around and gather dust for months on end when I was too occupied with re-designing pig breeding programs, re-decorating bathrooms and/or re-thinking *demi-haute cuisine*.

Many Zodiac PhD theses have a preface with a long list of people who helped the author with his research chores. Not so in this one, the project has been largely a private enterprise, or to put it a better way: pure hobby (it helped that much of the work could be done on a laptop computer, and that most airports supply free electricity if you know how to find those carefully hidden power sockets *and* carry a worldwide connector plug). But in these times of jealously guarded Intellectual Property, the handful of scientists who without any hesitation shared their precious data with me deserve to be acknowledged. In that respect thanks are due to Helmuth Pfeiffer, Andreas Susenbeth, Henry Jørgensen and the late Prins van der Hel. Gertruud Bakker hunted down some poorly accessible data and Søren Andersen dug deep into his impressive database to provide me with decades-old pedigree information. Vielen Dank, Helmuth und Andreas; tusind tak, Henry og Søren; hartelijk bedankt, Prins en Gertruud. Other people who contributed one way or another are acknowledged in the respective chapters.

But it is fair to say that Martin, Gerry and Ella have been the driving force behind this booklet. And considering that many a Wageningen PhD graduation has that wonderful unique operetta touch, but I like opera better, it seemed right to acknowledge each of them with a tiny bit of the latter.

For Gerry, who turned out to have published many of my discoveries years ago, who taught me things like the difference between a constant and a variable, how to judge a sigmoid, why one generally doesn't want one's assumptions to be 'strong' and much more, and who enriched my English vocabulary with terms like 'commensurate', 'wunch' and 'intellectually bereft': *Questo birbo mi toglie il cervello, tutto è un mistero per me.*[1] And I am not unique in that.

For Martin, who can make the impression that he hardly noticed what you told him before dinner, but then during the washing-up neatly dissects the flaws from your earlier reasoning: *Il dottore Dulcamara in ogni arte è professor!*[2] As much as Van Es is his spiritual father, Martin is my elder brother. Of course, Mariet does a pretty good job as the big sister that I never used to have…

For Gerry and Martin together, a virtual management team that wants you to make your texts much shorter but at the same time a bit longer, which leads to a strong tendency towards self-control: *Se vuol ballare, signor contino, il chitarrino le suonerò.*[1] These gentlemen double-book their diaries as shamelessly as an American airline company does its seats; the fact that people still try to arrange meetings with them is a clear illustration of their Added Value Potential.

Ella is not only a stimulating impresario; there is no-one better than her for merciless scrutiny of algebra and computer code. For my best friend and gravest critic, who between our PhD theses bravely overcame the monstrous disease that made me often wonder *Che farò senza Euridice?*[3], the part from the Habanera that says all there is left to say: *L'amour est enfant de bohème, il n'a jamais, jamais, connu de loi.*[4]

[1] Mozart & Da Ponte (1786): *Le nozze di Figaro*
[2] Donizetti & Romani (1832): *L'elisir d'amore*
[3] Gluck & Calzabigi (1762): *Orfeo ed Euridice*
[4] Bizet, Meilhac & Halévy (1875): *Carmen*

Contents

Pig farming is a wonderful mixture of scientific precision and massive ignorance.

C. Clark (1998) *International Pigletter* **18**(3):13.

The fact that 'efficiency' can mean many different particular things does not reduce its usefulness; it is no different, in this sense, from words like 'animal', 'plant', 'aeroplane' or 'book'.

C.R.W. Spedding *et al.* (1981) *Biological efficiency in agriculture.* Academic Press, London; p. 3.

Introduction

1. Describing maintenance requirements

This thesis deals with certain aspects of the maintenance energy requirements in growing pigs. This is a risky topic to write about: almost everyone seems to have some intuitive feeling for it, but the scientific literature is full of imprecise and contradictory descriptions and definitions of "maintenance", and of its requirements. It is therefore important to stress that this study does not attempt to clarify that particular issue. On the contrary, we assume some common understanding of the matter (rightly or wrongly), and look in more detail at some components of the aggregate. This section considers the various ways the aggregate has been approached, in order to put those components in their proper perspective.

There are at least three operational definitions of when an animal can be regarded to be "at maintenance". Close and Fowler (1982) (i) state that "the concept of maintenance [...] relates to an animal in energy equilibrium, neither losing nor gaining energy", (ii) stress that this would mean that the sum of the energetic equivalents of body protein and lipid deposition (the energy retention) is zero, but not necessarily that *both* these deposition rates would be zero, and (iii) continue with references to studies that have shown that immature animals have the tendency to deposit protein and catabolise lipid when fed at that particular level. The USA National Research Council (NRC, 1996) writes in its nutritional recommendations for beef cattle: "energy maintenance does not necessarily equate to maintenance of body fat, body protein, or body weight". This is the most common approach.
Others have restricted "the concept of maintenance" to the true steady state situation where "strictly there should be no translocation of material within the animal" (Armsby and Moulton, 1925), so that both protein and lipid deposition rates are zero (e.g. Kielanowski, 1965; Emmans, 1994).
Because it is much more difficult to monitor the body energy balance than to monitor body weight, animals are often assumed to be fed "at maintenance" when their body weight does not change (long-term trials such as by Taylor and Murray, 1991; short-term trials such as by Kolstad and Vangen, 1996, and by Ball *et al.*, 1998a) although this may be accompanied by considerable changes in their body energy balance.

Similar to many other authors, the USA National Research Council (NRC, 1988), when dealing with maintenance requirements of pigs, seems to have chosen not to commit itself to any of the above options, and to leave the choice to the reader. They immediately focus on the *requirements*: "Maintenance energy requirements include needs for all body functions and moderate activity. Many factors influence these requirements, including environmental temperature, activity level, group size, stress [...] and body composition". This is in contrast to, for example, Stephens's (1991) description, which would not allow for any heat increment of feeding or activity in its specification of "maintenance": "The term *maintenance requirement* as it is used in nutrition and metabolism literature is essentially conceptual, and represents that portion of heat production which is not attributable to productive processes such as growth, gestation, and lactation, or to other identifyable energy costs such as the heat increment of feeding or activity". In fact, this specification comes close to the fasting heat production.

Such specifications of maintenance requirements are important in animal science because applied feeding levels are often related to the presupposed maintenance requirement, *e.g.* "animals were fed at 2 or 3 times maintenance". The base level is commonly adopted from rec-

ommendations such as ARC (1981) and NRC (1988).

A more precise and quantitative description of maintenance energy requirements (ME_{maint}, in $kJ.d^{-1}$) was presented by Emmans (1994). When ignoring methane production in monogastrics, his equation builds upon fasting heat production (FHP; the energy expended when maintenance requirements are met by metabolism of body tissue) as follows:

$$ME_{maint} = FHP + [w_d \times FOM + w_u \times (UN - FUN)] \quad (1)$$

FOM, the fecal organic matter produced from the diet, and UN, the nitrogen excreted in the urine (both in $g.d^{-1}$), relate to the steady state with zero protein and zero lipid deposition. FUN represents UN at fasting. The constants w_d and w_u (estimated at 3.8 and 29.2 $kJ.g^{-1}$, respectively) translate mass into energy. The heat increment of feeding "at maintenance" corresponds to the term in square brackets in equation (1), but any heat increment associated with protein or lipid deposition is explicitly excluded from ME_{maint} as both are zero in this approach; as mentioned above, this does not hold for the majority of maintenance studies. Walker and Young (1993) made the same explicit distinction between "energy used for vital processes" (analogous to Emmans's ME_{maint}) and "extra energy costs associated with the productive state", and refer to the aggregate as *support costs*: "the machinery costs necessary to support the animal in a productive state, which have been shown to vary with growth rate".

The main picture that emerges from all this is one of confusion. There is neither general agreement about what ME_{maint} actually represents nor about its components, and most descriptions are of a qualitative nature. This is not likely to change in the foreseeable future. For the purposes of this thesis, the main issue is which metabolic processes should be included in the aggregate.

There is no disagreement about "physiological service functions" (Gill and Oldham, 1993) such as circulation, coordination, respiration and excretion. The levels of these in the absence of production are commonly included in the "basal metabolic rate" together with cell maintenance functions such as the active transport of ions through cell membranes (further referred to as "membrane transport") and turnover of the established body protein mass. It is also common to make some allowance for "basic activity", which in monogastrics includes little more than just standing upright rather than lying down, but in ruminants sometimes allows for grazing activity.

But actions such as thermoregulation, immune response and coping with other stressors are often excluded from the specification of the aggregate, although the literature is full of references to the apparent maintenance requirements of animals that were not kept in thermoneutral, pathogen-free and welfare-friendly conditions. A similar situation applies to all physical activity beyond the basic level, especially in young animals, and to physiological service and cell maintenance functions "above maintenance". Much of the disagreement about the proper definition of maintenance processes seems to stem from the difficulty of separating the costs involved with the above mentioned functions out of the measured heat production.

Taken together, these views would seem to allow for the quantitative description of the maintenance requirement of a mature animal in metabolic "steady state" that does not have to cope with any kind of stress on its system and that is engaged in only a basic level of physical activity (*cf.* Van Es, 1972; Webster, 1988; McCracken, 1992). Naturally, this steady state would

require the absence of dynamic processes such as growth, reproduction, lactation or physical work.

Maintenance costs of mature animals have received much scientific attention in the extensive meat production sectors (in the western world mainly sheep and beef cattle). Because of the low prolificacy of these species, a relatively large proportion of the total nutrient input into such production systems is required for "maternal overhead", *i.e.* for maintaining the parental generation rather than for bringing the progeny generation to its required slaughter point. The classical study of this issue is by Dickerson (1978), who made use of mid-seventies USA performance trait levels to parameterise his bio-economic model (Dickerson, 1982), and calculated that maintenance plus replacement of the parental generation of sheep and beef cattle requires 50 to 58 % of the total feed energy input per kg of edible meat protein produced from the slaughter progeny generation. Webster (1989b; table 1) gives (undocumented) corresponding values of 52 to 70 %. The maintenance costs of the progeny itself play a much less important role in such production systems (17 to 23 % from those same calculations). Hence the latter issue has been the subject of serious scientific study only recently (*e.g.* Ball *et al.*, 1998, and references provided there).
By contrast, in the intensive meat production systems based on broiler chickens, turkeys or pigs the maternal overhead requires a much smaller proportion of the total feed energy input (6.5 to 20 % according to Dickerson, 1978; 4 to 20 % according to Webster, 1989b). The 20 % figures are for pigs; its current value would be much lower due to increased reproductive rates since that time (see also Large, 1976). The maintenance costs of the slaughter progeny have a considerable impact on the overall energetic system efficiency (31 to 60 % from those same calculations, which seem unrealistically high values nowadays). Hence this issue merits, and has attracted, more scientific attention than in the extensive production systems.

We have to consider, then, the maintenance requirements of a growing immature animal that, by definition, is not in metabolic steady state. This makes it again difficult to decide what should be included in our conceptual maintenance processes. Young *et al.* (1989) measured oxygen consumption in fetal, neonatal, growing and adult sheep, and found significantly elevated metabolic rates per $kg^{0.81}$ metabolic body weight during the stage of highest relative growth rate (28 to 74 days of age). These authors conclude that the elevated metabolic intensity associated with production processes makes the scaling of metabolic rate with a common body weight exponent inappropriate. Hence when maintenance requirements are expressed as a function of metabolic body weight with a fixed exponent (*e.g.* α $kJ.kg^{-0.75}.d^{-1}$), they (or rather, α) would become inflated during rapid growth.
This seems to build upon one of Stephens's (1991) surmises: "in immature animals that are in positive energy balance, the physiological processes which make up maintenance requirement are running at elevated levels (Milligan and Summers, 1986)". In order for this to make literal sense, "maintenance" would have to be partly defined as the direct result of production processes, which goes far beyond more consistent definitions such as Emmans's in equation (1). But although Stephens's "maintenance" is clearly confounded with production-related metabolic processes, it represents one of the dominant views on the issue in current animal science (Cleveland *et al.*, 1983; Tess *et al.*, 1984b; Summers *et al.*, 1986; Baldwin and Hanigan, 1990).

Part of the associated confusion may be eliminated by using the terms *metabolic intensity* (Turner and Taylor, 1983) or *support functions* (Walker and Young, 1993) instead. For example, the former authors' statement "an animal at equilibrium is in a 'tuned-down' physiological state. The food used in such a state can hardly be equated quantitatively to the food used for basic vital functions in a productive animal" conveys roughly the same information as Stephens above, but it is unambiguous because the term "maintenance" is avoided.
The elevated physiological processes referred to by Stephens are mainly the functions related to increased nutrient intake (foraging and feed intake activity; digestion and its associated enzyme production and wear and tear on digestive tissues; excretion) summarised in increased heat increment of feeding, and intensified cellular functions such as membrane transport and protein turnover (*cf.* Milligan and Summers, 1986). Obviously, metabolic intensity would be more strongly elevated by the process of growth when that growth is more intense, due to two possible factors: (i) the rate of growth, *e.g.* in kg per day, and (ii) its composition, in terms of the ratio of protein to lipid deposition.

2. Explaining maintenance requirements

Both growth rate and growth composition have been the subject of substantial genetic change through artificial selection in commercial pig and poultry populations, especially in the second half of the twentieth century (see McKay *et al.*, 2000, and Merks, 2000, for examples). Although this genetic change has dramatically increased the gross production efficiency of pig and poultry meat, the growth-related elevation of metabolic intensity in young growing pigs, turkeys and broiler chickens makes the individual animal more and more expensive to maintain (or more appropriately, to support) on a daily basis. A large part of this apparent trend is caused by the widely established habit to express maintenance requirements per $kg^{0.75}$ metabolic body weight: modern lean genotypes contain more protein per $kg^{0.75}$, and it is mainly the proteinaceous tissues that generate the maintenance-related metabolic functions.

For example, Campbell and Taverner (1988) and Rao and McCracken (1992) measured maintenance requirements of growing males of "high lean growth" pig genotypes by extrapolation of energy intake at various feeding levels towards zero energy retention, and report estimates of 600 to 610 kJ per $kg^{0.75}$ metabolic body weight per day (further denoted as $kJ.kg^{-0.75}.d^{-1}$). These estimates are much higher than the levels between 420 and 460 $kJ.kg^{-0.75}.d^{-1}$ recommended for growing pigs by the UK Agricultural Research Council (ARC, 1981) and the USA National Research Council (NRC, 1988). These recommendations were compiled from much earlier sources, which form a mixture of (i) studies similar to Campbell's and Rao's, extrapolating energy intake to zero energy retention, and (ii) factorial analyses according to model (2) in section 3. Following the above reasoning, a considerable part of this difference would disappear when maintenance requirements were expressed per unit of body protein mass, as stressed by Whittemore (1983), Webster (1983, 1988) and Emmans and Fisher (1986), among others.

But the metabolic intensity of the "lean" tissue varies considerably between tissue pools as well. This issue was recently reviewed by Archer *et al.* (1999) who focused mainly on ruminants, but a convenient example in normally growing pigs is from Pekas and Wray (1991). These authors subjected pigs to indirect calorimetry to measure FHP, and related the results to the mass of several tissues by cluster analysis. Strong relations were found between FHP and

the mass of the gastro-intestinal tract (particularly the small intestine), the liver, pancreas and kidneys. Likewise, the maintenance requirements of immature pigs were related to muscle mass and viscera mass by Van Milgen *et al.* (1998) and Van Milgen and Noblet (1999), who report a contribution (per unit of tissue mass) of the viscera to FHP (in fasted pigs) and to the maintenance requirements (in pigs fed *ad libitum*) three to four times as high as the contribution of muscle. As Webster (1988) noticed, this raises the question as "to what extent differences in maintenance requirements [can] be attributed to differing proportions of the different organs and tissues of the body, each having different metabolic rates". It is this question that leads to the study carried out in this thesis.

At the same time, the voluntary feed intake of growing pigs has decreased considerably over the past few decades, as illustrated in Figure 1. Much of this decrease is simply due to reduced energy requirements because of reduced lipid deposition (as illustrated in section 5.4 of the the General Discussion chapter), but part of it may reflect a reduced intake *capacity*.

McCracken *et al.* (1994) subjected pigs of a modern "high-lean growth" genotype to forced feeding and failed to measure an increased protein deposition rate in these overfed pigs relative to *ad libitum*-fed controls. This result does not support the notion of a reduced intake capacity in at least that particular genotype. Nevertheless, in genotypes where it would occur, the options for coping with unexpected loads on the system (which would require extra resources) may be compromised.

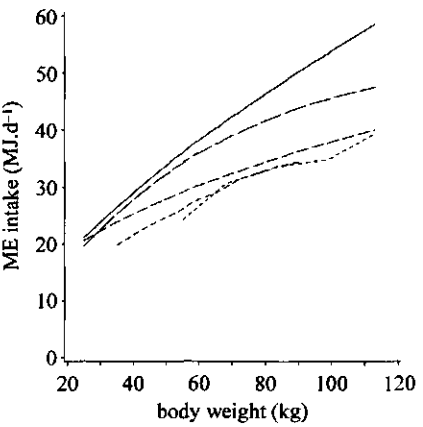

Figure 1 Daily *ad libitum* ME intake patterns of growing pigs, in relation to body weight, as reported by (from top to bottom) Cole *et al.* (1967), NRC (1988), Cole and Chadd (1989), Labroue (1996), and Von Felde (1996). Updated from Close (1994).

The notion has been developed in animal ecology that "investment into production [is] traded off against investment into maintenance" (Wieser, 1994), specifically in "conditions of ecological stress [where] an environmental change [...] disturbs the balance between maintenance and production". The author continues with examples of reduced maintenance functions (protein turnover and membrane transport) with increased production levels in a wide variety of animal species, and concludes that high levels of maintenance functions naturally lead to high levels of metabolism and the associated energy requirements but at the same time provide a "greater [...] range over which the activity of cells can be controlled" and a "greater flexibility and richer behavioural repertoire".

From that point of view, it is more appropriate to use the term "maintenance *processes*" than "maintenance *requirements*", the latter term emphasising the implied costs rather than the functionality of the processes involved. Insight in this matter would benefit from "maintenance" being regarded as a set of fitness-related functions rather than merely as a cause of nutritional inefficiency. The allocation of sufficient resources to these fitness-related functions is crucial for homeostasis, and since evidence is slowly emerging that this allocation is at least partly genetically regulated (*cf.* Knap and Luiting, 1999), the consideration of maintenance

requirements as a full-fledged component of breeding objectives becomes more and more relevant.

3. Measuring maintenance requirements

Although in section 2 we cited studies that "measured maintenance requirements of growing pigs", this measurement is by no means straightforward, mainly because the statistical partitioning of many physiological service functions, and of the metabolic costs of protein synthesis and membrane transport, into maintenance- and growth-related processes is not feasible. It is notoriously difficult to obtain unconfounded estimates of the maintenance energy requirements (ME_{maint}) and the energetic efficiencies of protein and lipid deposition (k_P and k_L) without consistent specifications of ME_{maint} such as in equation (1).

The straightforward approach towards measuring a process that requires a zero energy retention in order to be consistently defined, would seem to be to subject an immature animal to a feeding level that keeps it in that state, and measure its metabolic intensity as a direct estimate of ME_{maint}. However, it has been argued that the resulting measurements (as reported for immature pigs by Jentsch *et al.*, 1989, and Hoffmann *et al.*, 1992, and by Vangen, 1980, and Kolstad and Vangen, 1996) would reflect the animal's metabolic intensity at maturity (which it has not attained yet) rather than the elevated intensity in its undisturbed growing state. Stephens (1991) used evidence from an experiment by Taylor *et al.* (1981) on immature cattle to conclude that "immature animals not in positive energy balance are likely to make metabolic adjustments which render estimates of maintenance requirements suspect" and "maintenance requirements per unit body weight at artificially imposed equilibria [are] identical to those at maturity; [...] a sufficiently long equilibration period [allows] the animal's metabolism to settle at the same base level as it would ultimately reach if development were allowed to proceed normally".

Van Milgen and Noblet's (1999) analysis of deposition data measured in growing pigs fed *ad libitum* suggests that if these pigs would be fed "at maintenance", they would deposit some body protein and catabolise body lipid. These authors are uncomfortable with that result (for reasons not relevant here), and give three reasons why it may be an anomaly: erroneous data, an inadequate statistical model, or "probably most important, the concept of maintenance [involving zero energy retention] may not be appropriate for growing animals". The latter notion (which is supported by Close and Fowler, 1982, and Walker and Young, 1993, among others) would again imply that the result of extrapolation of observations on growing animals towards their state of energy equilibrium (or *vice versa*) should not be treated as a meaningful physiological characteristic. As Moe (1992) put it, "it is possible to extrapolate [...] to zero growth rates to identify a maintenance component. If this hypothetical maintenance component is accepted as a mathematical entity rather than a physiological one, many conceptual problems can be avoided". Webster (1988) characterised maintenance in growing animals as "an operational description".

Interestingly, Dawson and Steen (1998) estimated ME_{maint} in growing immature sheep and beef cattle, and found the results to be much higher than the corresponding ARC (1980) and AFRC (1990) recommendations. They attribute the difference not to genetic changes in growth intensity (as in the pig studies cited in section 2) but to changes in measurement con-

ditions: the earlier estimates derive from trials that attempted to keep the animals in steady state and it "would be expected that heat production by the visceral organs would be lower than in fully fed animals" because "higher maintenance requirements associated with higher rates of gain appear to be due to the increased mass of metabolically active organs such as the liver, intestines, heart and kidneys". Walker and Young (1993) and Van Milgen *et al.* (2000) notice the same trend in growing pigs kept on various feeding levels.

An alternative, and widely used, approach to measuring ME_{maint} is by extrapolation of observations on animals in positive energy balance, applying Kielanowski's (1965) "factorial analysis" to regress ME intake on protein and lipid deposition:

$$ME_{intake} = ME_{maint} + \frac{23.8}{k_P} \times P_{dep} + \frac{39.6}{k_L} \times L_{dep} \quad (2)$$

(where P_{dep} and L_{dep} denote protein and lipid deposition in $kg.d^{-1}$, respectively; k_P and k_L denote the energetic efficiencies of these deposition processes; the constants 23.8 and 39.6 $kJ.g^{-1}$ are the net combustion energy contents of protein and lipid). The estimate for ME_{maint} follows as the intercept of the regression analysis, usually from extrapolation.
It has been noticed that this multiple linear regression approach has the disadvantages of intercorrelated independent variables (*e.g.* Kielanowski, 1976a; Close and Fowler, 1982; Tess *et al.*, 1984b; Walker and Young, 1993; Noblet *et al.*, 1999) and larger measurement errors on the independent variables than on the dependent one (Emmans and Kyriazakis, 1997). This causes the associated parameter estimates to be confounded and biased, respectively, which makes it statistically hazardous to interpret them independently from each other. In accordance with this, Tess *et al.* (1984b) reviewed literature estimates of k_P and k_L for growing pigs (all obtained with models like (2)), which they found to range from 0.36 to 0.76 and from 0.58 to unity, respectively, and which they found to depend strongly on ME_{maint} which was either estimated as a parameter in the same analyses or assumed fixed. The common practice of relating ME_{maint} to metabolic body weight with a fixed rather than a simultaneously estimated exponent is likely to cause even more interdependence of the estimates (Noblet *et al.*, 1999).
Hence the factorial analysis estimates its three parameters (k_P, k_L and ME_{maint}) rather inappropriately. Van Milgen and Noblet (1999) present an alternative statistical model with two simultaneous nonlinear equations that relate protein and lipid deposition, respectively, to ME intake above maintenance. This produces estimates for the same three parameters as above plus the fraction of ME intake above ME_{maint} that is designated for protein (as opposed to lipid) deposition, and the "change in energy gained as protein (and lost as lipid) relative to the change in BW for animals fed at zero energy retention". The statistical improvement of this approach over the factorial one is the simultaneous solution of the protein- and lipid-related processes, which allows for taking into account the relation between these so that the estimates are less confounded and therefore more reliable. The price to pay is the necessity to estimate two extra parameters.

In another attempt to avoid the above mentioned confounding between parameter estimates, Emmans (1994) proposed an alternative (and this time internally consistent) arrangement of the ME-requiring body functions. In contrast to Van Milgen and Noblet (1999), whose main contribution is in the statistical processing of the data, this approach involves a change of model. It re-defines ME_{maint} according to equation (1) in section 1, and allows explicitly for

the heat increment of feed intake "above maintenance". In this extended model, k_p and k_L are true and unconfounded constants for a given diet composition. This approach is further described in Chapter 3 of this thesis.

4. Partitioning maintenance requirements

Much of the increase in gross efficiency of pig meat production mentioned in section 1, was brought about by a genetic change towards increased leanness. This effect will reach a plateau when the economically optimum levels of pig carcass leanness are achieved, which will not take long in the western world. When a further increase of efficiency is desired, it will then have to come from a reduction of overhead costs, either by a further increase of growth rate (reduction of the time to slaughter) or by a reduction of the overall maintenance requirements per unit of metabolic body weight per day. As discussed in section 1, this in itself is likely to reduce metabolic scope and make the system more sensitive to environmental instability. A prerequisite for control of such, more environmentally sensitive, production systems would be to study the maintenance-related processes and their metabolic costs and benefits in more detail. To quote NRC (1996) again: "Successful management of beef cattle, whether for survival and production in poor nutritive environments or for maximal production, depends on knowledge of and understanding their maintenance requirements".

In order to achieve that, we would have to consider the physiological service functions and processes like body protein turnover, cell membrane transport, thermoregulation, immune response, coping with other stressors (most notably, social ones), and physical activity. Because there is a considerable between-animal variation (largely of a genetical nature) in body composition, the maintenance functions related to body composition must be expected to show such variation as well, certainly as long as maintenance requirements are expressed in relation to metabolic body weight. This would hold for protein turnover and membrane transport, for thermoregulation, and possibly also for some immune response functions (see section 7.1 of the General Discussion chapter). The other functions, not obviously related to body composition, have been found to vary between individuals too. This holds for many immune response functions (see Knap and Bishop, 2000), for physical activity (Dunnington *et al.*, 1977; Heckl-Ensslin *et al.*, 1991), and for response to social stressors (Jonsson, 1985; Hohenboken, 1986; Koolhaas and Van Oortmerssen, 1998). It follows that we must expect ME_{maint} to vary between animals within a genotype, and that this variation is partly of a genetical nature.

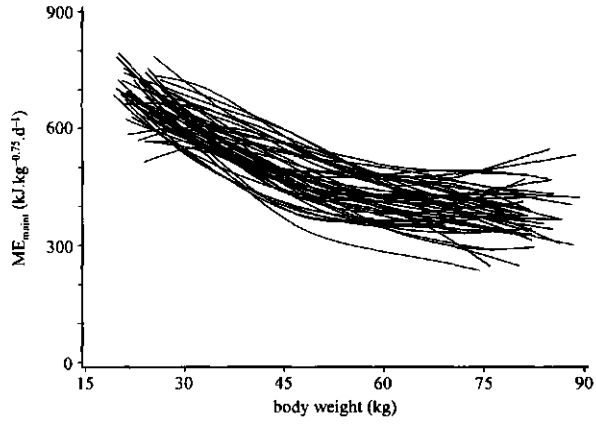

Figure 2 Estimated maintenance energy requirements (ME_{maint}) of 48 growing pigs in relation to body weight. Spline interpolation curves through data from Thorbek (1975).

The classical source of infor-

mation on maintenance requirements in individual growing pigs is Thorbek (1975). Her experimental results have been summarised in Figure 2, which shows the estimated ME_{maint} of 48 scale-fed growing pigs in relation to body weight. Each pig was subjected to indirect calorimetry eight times between 23 and 80 kg BW, and k_P and k_L were estimated (at 0.48 and 0.77) on the data while assuming a constant value for ME_{maint} at a given body weight. Estimates for ME_{maint} were then obtained for each of the 48 × 8 records as $ME_{maint} = ME_{intake} - \frac{23.8}{k_P} \times P_{dep} - \frac{39.6}{k_L} \times L_{dep}$ (using the above estimates for k_P and k_L, but with k_L=1 in case of lipid catabolism). Re-analysis of these data produced a REML estimate for the between-animals variance component of ME_{maint} at 0.24.

The literature provides some more references for variation in the maintenance requirements of mature cattle, chickens and mice, and of growing cattle, mice and pigs, and many more for variation in residual feed intake in those same species. This will be dealt with in more detail in section 3.2 of the General Discussion chapter.

5. Modeling maintenance requirements

Black *et al.* (1995) referred to the various maintenance-related processes as discussed in section 4 when they wrote: "ideally, these components of maintenance should be represented also within a comprehensive model of animal growth". Similarly, "in order better to address this variation [in ME_{maint}], several groups have developed mechanistic models which attempt to capture cause and effect relationships underlying maintenance energy expenditure as they vary across physiological states, environmental conditions, breed and other factors. A number of physiological/metabolic functions which contribute to variance in apparent maintenance requirements have been identified; these functions have been characterized, at least partially, using mechanistic models" (Baldwin and Hanigan, 1990). These authors claim that many physiological service functions, and also the metabolic costs of protein synthesis and membrane transport, "manifest themselves as components of both maintenance energy expenditures and costs of production [...] As a result, mechanistic models are increasingly deviating from [the] use of the classical concept of depicting costs of maintenance and production separately". This coincides with the surmise of Van Milgen and Noblet (1999) quoted in section 4, and with the reasoning on support costs by Walker and Young (1993). It also illustrates the potential value of the use of mechanistic simulation models (Thornley and France, 1984), rather than empirical statistical models, for studying the above mentioned processes. Of course, the increasing complexity of simulation models when mechanistic routines are added to them has its disadvantages as well; we come back to that in section 3.4 of the General Discussion chapter.

For the simulation studies initiated in this thesis, it is useful to distinguish between three partially overlapping groups of maintenance-related processes: (i) the physiological service functions, (ii) processes triggered by environmental factors (thermoregulation, immune response, reactions on social stressors), and (iii) processes related to body composition (protein turnover, membrane transport, thermoregulation and possibly some immune response functions). Group (iii) is of the most immediate interest in a pig breeding context, because it is body composition that is influenced most by pig breeding activities. But given the "advanced" stage of the current production genotypes in some meat-producing species, group (ii) is of rapidly

increasing interest to animal breeders, because some of these traits seem to be primarily responsible for the environmental sensitivity of highly productive genotypes that leads to genotype by environmental interactions. Of course, in the animal breeding context, the interest is as much directed to the between-animal variation of these processes as to their mean levels. The novel contribution to science that is attempted at in this thesis is the mechanistic modeling of between-animal variation in some of the processes of the above groups (ii) and (iii). We focus on protein turnover and thermoregulation because there is quantitative information available (albeit incompletely) that makes it possible to describe these processes as functions of body composition. For membrane transport and immunocompetence, this is not (yet) the case. These functions are discussed in some detail in sections 6 and 7 of the General Discussion chapter.

6. Contents of this thesis

Chapter 1 describes how we extended an existing growth model with a routine to simulate protein turnover as a function of body composition. That extended model is used in chapter 2 to find out to what extent we may expect maintenance requirements to vary as a result of variation in body composition, mediated through protein turnover.

Likewise, chapter 3 describes how we extended an existing growth model with a routine to simulate thermoregulation as a function of body composition. That extended model is used in chapter 4 to find out to what extent we may expect maintenance requirements to vary as a result of variation in body composition, mediated through thermoregulation.

The stochastic models used in chapters 2 and 4 had to be parameterised with variation coefficients of the distribution of body protein over pools, and body lipid over depots, but such variation is poorly documented. Chapter 5 presents evidence for the fact that there actually *is* animal-intrinsic variation in those traits.

The models that were extended in chapters 1 and 3 make use of a few parameters to characterise the genotype of the pigs that they simulate, with the implicit assumption that the values of these parameters differ between pig populations (and within them, but that is not the point here). Chapter 6 describes five pig genotypes in terms of these parameters, showing the magnitude of these differences among real-life populations.

The General Discussion deals with some details that were not included in chapters 1 and 2 or in chapters 3 and 4, either because there was no room for that in the associated journal articles, or because this information was discovered after publication.
After that, we make some short comments on the analyses of chapters 5 and 6, which can only be regarded as "the best we can do" with the data currently available. There is a strong need for better data if this kind of analysis is ever to become serious, and we briefly describe the trials that would be necessary to produce such data.
Finally, we discuss the aspects of membrane transport and immunocompetence that are relevant for modeling of these functions, and attempt to place our findings in a broader perspective, including the use of this technology in animal breeding.

Who could say that this entity on the photograph was the creature he had met this evening? Physically, biologically, they were not the same. He had read somewhere that the body cells renewed themselves every seven years. Identity did not continue. Former times, former people. The identity was new, endlessly new, endlessly evolving. Wonderful.

Lionel Davidson (1968) *Making good again.* Mandarin, London; pp. 44-45, 199. Condensed.

Chapter 1 is based on

P.W. Knap and J.W. Schrama (1996) Simulation of growth in pigs: approximation of protein turnover parameters. *Animal Science* **63**:533-547

Chapter 1

Approximation of protein turnover parameters

A dynamic model for simulation of growth in pigs was extended by a module to describe protein turnover in six body protein pools (muscle, connective tissue, liver, blood plasma, gastro-intestinal, and "other" proteins). The model describes protein deposition in these pools following different growth curves and differential rates of turnover. Growth curve parameters and turnover rates were obtained from the literature.

In growing animals, experimentally measured turnover rates represent a combination of turnover of already present body protein and fractional (repeated) synthesis of newly deposited protein. An attempt was made to distinguish between these processes by varying the values of the fractional rate of synthesis of newly deposited protein (FRS_{dep}) and of the proportion of maintenance energy requirements not related to protein turnover ($FrcME_{maint}$), and comparing the simulated output to the output from the original model without the protein turnover module.

The turnover rate (TR_{pres}) of already present connective tissue protein reached unrealistic values for $FRS_{dep} > 2.5$ per day, which puts an upper limit to FRS_{dep}.

The output from the extended and the original models showed similar patterns for certain combinations of FRS_{dep} and $FrcME_{maint}$, dependent on the levels of model input variables. For $FRS_{dep} \leq 2.5$ per day, these similar patterns have their optimum at $FrcME_{maint} = 0.65$, coinciding with $FRS_{dep} = 2.0$ per day. The corresponding TR_{pres} values were 0.060, 0.019, 0.585, 1.492, 0.582, and 0.016 per day for the above mentioned pools.

Introduction

Consumed energy is partitioned over the requirements of body maintenance, protein deposition, and lipid deposition. In a large part of the animal production cycle, the energy requirements of body maintenance are quantitatively at least as important as those of the deposition processes. When the term "maintenance" is defined as energy intake minus the calculated energy requirements of deposition processes, and is adjusted for metabolic body weight, a considerable amount of genetic variation appears to be present in the residual term in pigs (Foster *et al.*, 1983; De Haer *et al.* 1992; Mrode and Kennedy, 1993) as well as in other livestock species. Therefore, maintenance requirements form an important source of genetic variation in feed efficiency, possibly to be exploited by animal breeding.

At the same time, however, maintenance is by far the least documented process of the abovementioned three, which causes a possibly interesting source of genetic variation to be largely neglected because of lack of knowledge of the system. It may be worthwhile to study the phenomenon in more detail, further separating the underlying physiological processes of this composite trait, e.g. body protein turnover, ion transport over cell membranes, thermoregulation, immune response, coping with other stressors, and physical activity.
Although maintenance is commonly regarded as being independent of deposition processes, it must be noted that the energy requirements of body protein turnover and thermoregulation are naturally dependent on body composition characteristics, especially the body protein to lipid ratio. In this way, part of the maintenance requirements may be influenced by deposition-related traits. This means that a change in these traits will affect a correlated change in maintenance requirements. Breeding activities have resulted in major changes in protein and lipid deposition in pigs; the typical 100 kg slaughter pig contains nowadays about 15 kg less fatty tissue than 35 years ago. And although pig breeding organisations are now gradually reducing the selection pressure on leanness, we may expect this trend to generally continue for the next decade or so.

The starting point for this study was the algorithm described by Moughan and Smith (1984) and reviewed by Moughan and Verstegen (1988) for simulation of the protein and energy metabolism of growing pigs. Details of a modified version of this model, as far as these are essential for the understanding of the present text, are in Appendix I.
In the original model (Moughan and Smith, 1984), after maintenance energy requirements (given as a simple function of metabolic body weight) have been fulfilled, energy metabolism is determined largely by the amount of energy required for body protein deposition and the minimum required lipid deposition. The conversion of ingested amino acids into body protein is the driving force behind the simulated processes.

It is our purpose to extend this model with a more elaborate quantification of maintenance energy requirements, separating "maintenance" into its major constituent processes. For this purpose we will explicitly consider, in Chapters 1 to 4 of this thesis, the energy requirements of protein turnover and thermoregulation. The idea is that when these processes can be modelled separately, the interrelationships between maintenance energy requirements and deposition-related traits can be described in more detail.
The aim of this Chapter is then to derive reasonable approximations for key parameters to quantify the energy requirements of body protein turnover. In Chapter 2 we attempt to quan-

tify the turnover-dependent relations between body composition and maintenance requirements.

Protein turnover

To maintain homeostasis, body protein is continually being degraded into amino acids and resynthesised. In this way, substrate and enzyme levels can be quickly adapted to changing external influences, and protein with erroneous amino acid sequences or damaged structure can be replaced before adversely affecting the organism. The functions of cells and tissues can be adapted, thus coping with long-term external changes. The aggregate of catabolic and anabolic processes involved in this amino acid flow is referred to as "protein turnover". It generally involves a large fraction of amino acids in the body; typically about half the amino acids daily entering the extracellular pool of a young mammal stem directly from degraded body protein, the remainder being directly supplied by the diet (Riis, 1983b).
Protein catabolism does not appear to require large amounts of energy; but resynthesis of body protein requires approximately 5 mol ATP per mol arranged peptide bonds (Van Es, 1980). This means that the energy requirements involved with protein turnover are ususally quite large.

Quantitatively, protein turnover is a poorly documented process. Three parameters are used to describe turnover: the *fractional rate of synthesis* (FRS) describes the fraction of total body protein that is daily (re)synthesised from free amino acids; the *fractional rate of catabolism* (FRC) describes the fraction that is daily degraded into free amino acids; the *turnover rate* (TR) describes the fraction that is daily degraded and resynthesised. In growing animals, TR = FRC, whereas TR = FRS in animals that lose weight. When net body protein balance is zero, TR = FRS = FRC.
FRS and FRC can be measured *in vivo* by making use of radio-actively labeled amino acids, supplied to the animal in the feed or by infusion (see Waterlow *et al.*, 1978, and Schreurs *et al.*, 1992). FRS is quantified by determining the amount of labeled amino acid incorporated into the body protein shortly after administration. After prolonged administration of labeled amino acid, resulting in saturation of body protein with the marker, FRC can be measured as the amount of labeled amino acid that is released from the body protein. Usually, the total amount of body protein is assumed constant, which implies that TR = FRS = FRC. This assumption is obviously violated in growing animals, where resynthesis of degraded present body protein (which is the "base level" of TR) is accompanied by protein deposition. Hence, measured FRS exceeds true TR in these animals.
Published values for FRS and FRC (reviewed by Waterlow *et al.*, 1978; Riis, 1983a; Simon, 1989) show large variation between the various body tissues. Muscle and connective tissue proteins have generally lower turnover rates than most organs and, especially, blood plasma proteins.

In growing animals, a considerable fraction of the body protein that is deposited daily in addition to the already present amount is broken down and resynthesised before final deposition is accomplished, probably several times (Van Es, 1980; Reeds, 1989; Klein and Hoffmann, 1989). This would allow for corrections of erroneously created amino acid sequences.
This means that overall FRS, as measured in a growing animal, is a combination of the turn-

over rate of the present body protein (TR_{pres}) and the fractional rate of synthesis of newly deposited protein (FRS_{dep}). In other words: "protein turnover can be regarded as consisting of two components: an essentially inevitable turnover associated with the maintenance of cell function and a variable turnover associated with growth" (Reeds, 1989), related to TR_{pres} and FRS_{dep}, respectively.
Values published in the literature seem to refer to overall FRS; FRS_{dep} and TR_{pres} are hard to quantify separately in growing animals. Of course, FRS would reflect TR_{pres} in mature non-growing animals.

Model

In order to accommodate protein turnover processes, the model (Appendix I) was extended as follows.

Costs of protein synthesis

The above mentioned energy requirement for the resynthesis of body protein (5 mol ATP per mol peptide bonds) was considered equivalent to an energy requirement of 410 kJ metabolisable energy (ME) per mol arranged peptide bonds. This assumes that one mol ATP is, on average, equivalent to 82 kJ ME; this is the average of the 74, 78, and 93 kJ ME from glucose, fatty acids, and volatile fatty acids quoted by Van Es (1980).

Protein pools

To describe the ME requirements for turnover of body protein, six body protein pools were distinguished, each accounting for a major part of the daily protein synthesis: (i) *muscle*; (ii) *connective tissue* (bone, cartilage, skin, claws, hair, and fatty tissue); (iii) *liver*; (iv) *blood*

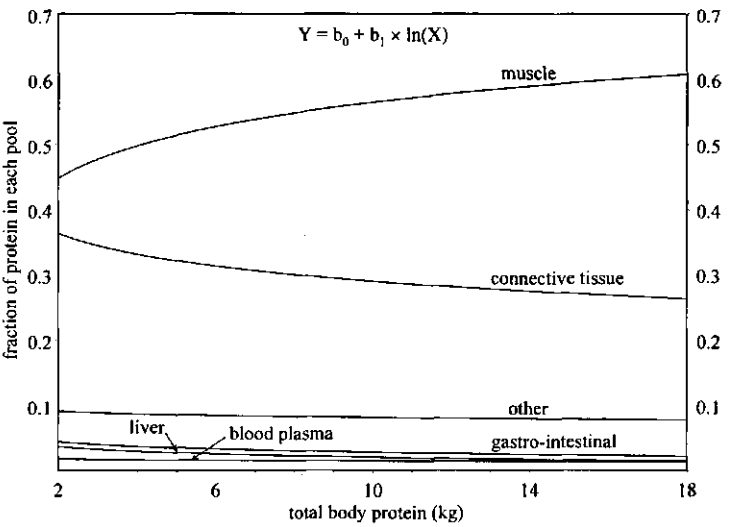

Figure 1 The simulated course of distribution of body protein pools, in relation to total body protein mass. Coefficients for muscle protein: $b_0 = 0.3965$, $b_1 = +0.07314$; connective tissue: $b_0 = 0.3943$, $b_1 = -0.04525$; "other": $b_0 = 0.0955$, $b_1 = -0.00601$; gastro-intestinal: $b_0 = 0.0511$, $b_1 = -0.01$; liver $b_0 = 0.0435$, $b_1 = -0.01$; blood plasma protein: $b_0 = 0.0191$, $b_1 = -0.00188$.

plasma; (v) *gastro-intestinal* (including smooth muscle, epithelium, and digestive enzymes); and (vi) *"other"* proteins (thoracic and abdominal organs, the central nervous system, blood cells, etc.). Riis (1983a, table 5.5) suggested that these pools account for approximately 35, 10, 12, 21, 17 and 5 %, respectively, of daily protein synthesis in a 50 kg pig.

The distribution of body protein over these pools varies with the animal's degree of development (muscle protein grows relatively faster than the other pools, especially in young animals). Figure 1 shows the average course of the proportions of several protein pools. These patterns were obtained by relating data from Oslage (1965), from Metz *et al.* (1980), from Tullis (1981), from Jørgensen *et al.* (1985), from Susenbeth and Keitel (1988), and from Pfeiffer *et al.* (1990) to total body protein mass, and generalising the results as described in Appendix II.

The number of peptide bonds per gram of protein in each of the pools (NPB_i, in $mol.g^{-1}$, i=1,...,6) was derived from the composition of each pool's protein in terms of 18 amino acids (the essential ones, and ala, glu, gly, pro, ser, asp, and others) and their molecular weights, as follows:

$$NPB_i = \sum_{j=1}^{18} \frac{frcAA_{ij}}{molwtAA_j - 18.028} \qquad (1)$$

The fractions of protein i that are made up by amino acid j ($frcAA_{ij}$) have been derived from Riis (1983a, table 5.1). The constant 18.0128 is the molecular weight of the water that is released at peptide bond formation; $molwtAA_j$ denotes the molecular weight of amino acid j. The resulting values are NPB_i = 9.27, 10.68, 9.35, 9.35, 9.59, and 9.08 millimol peptide bonds per gram protein in the above mentioned pools, respectively.

Turnover of present body protein.

For each of the six protein pools, the turnover rate ($TR_{pres,i}$) of already present body protein was approached as follows. Riis (1983a, table 5.5) gives values for overall FRS_i of the proteins in these pools as estimated for a growing pig of 50 kg body weight. These values are "approximations obtained by interspecies comparisons of the values" that were derived from a literature survey (Riis, 1983a, table 5.3). As such, they represent a combination of TR_{pres} and FRS_{dep}:

$$FRS_{overall,i} = \frac{TR_{pres,i} \times P_{pres,i} + FRS_{dep} \times P_{dep,i}}{P_{pres,i} + P_{dep,i}} \qquad (2)$$

As the animal matures, P_{dep} decreases and P_{pres} increases, which leads to an increasing proportion of $FRS_{overall}$ being due to TR_{pres}. Because $TR_{pres,i}$, as rate variables, are usually much lower that FRS_{dep}, $FRS_{overall}$ decreases towards maturity. Equation (2) can be rearranged as

$$TR_{pres,i} = FRS_{overall,i} - \frac{P_{dep,i}}{P_{pres,i}} \times (FRS_{dep} - FRS_{overall,i}) \qquad (3)$$

Thus TR_{pres} may be derived from available $FRS_{overall}$ values by subtracting from these a factor comprising the ratio between the amounts of daily deposited (P_{dep}) and present (P_{pres}) protein

(i.e. the specific growth rate of protein), and the difference between the fractional rate of synthesis of newly deposited protein (FRS_{dep}) and $FRS_{overall}$.

Values for the amounts of protein present in each pool ($P_{pres,i}$) follow from Figure 1. The daily amount of protein deposited in each pool ($P_{dep,i}$) was quantified simply by subtracting the value of $P_{pres,i}$ obtained in the last simulation time step from the value in the present one. Differentiation of the corresponding formulae with respect to time was not really feasible because of the employed time step of one full day.
Then, permissible (possible, but not necessarily realistic) values for $TR_{pres,i}$ may be derived from equation (3) by varying FRS_{dep} over its likely range, as described below.

Finally, the ME requirements for turnover of already present body protein (in $kJ.d^{-1}$) follow from:

$$ME_{turn,pres} = 410 \times \sum_{i=1}^{6} TR_{pres,i} \times P_{pres,i} \times NPB_i \quad (4)$$

Turnover of newly deposited body protein.

The ME requirement for the repeated breakdown and resynthesis of newly deposited protein (connected to FRS_{dep}) was described by a similar procedure as for the turnover of present protein, distinguishing the same six protein pools.
For each pool, the number of peptide bonds that will be rearranged as a result of the repeated synthesis of deposited protein can be estimated from the NPB_i values (equation (1)) times the amount of daily deposited protein $P_{dep,i}$. The associated ME requirements follow from:

$$ME_{turn,dep} = 410 \times FRS_{dep} \times \sum_{i=1}^{6} P_{dep,i} \times NPB_i \quad (5)$$

In fact, the above procedure makes explicit the part of the overall ME requirements of protein deposition (ME_{Pdep}, assumed to be 53 $MJ.kg^{-1}$ by Moughan and Verstegen, 1988) that results from the (repeated) arrangements of peptide bonds. Because 5 mol ATP per mol peptide bonds is supposed to correspond to $410 \times \frac{1}{6} \sum_{i=1}^{6} NPB_i = 3.92$ MJ ME per kg protein, the value for ME_{Pdep} used during simulation must be reduced by $3.92 \times FRS_{dep}$.

Energy expenditure.

The "growth loop" in the simulation programme (Appendix I) was extended as follows.

The ME requirements for body maintenance (ME_{maint}) were originally simulated as a function of metabolic body weight (Figure 2; De Greef, 1987). These were reduced to "maintenance *independent* of protein turnover" by multiplication with $FrcME_{maint} < 1$.

This assumes that the remaining maintenance-related processes (thermoregulation, immune response, physical activity and "basal metabolism") are correctly described by the same type of function:

$$ME_{maint,indep} = FrcME_{maint} \times (495 - 0.75 \times BW) \times BW^{0.75} \quad (6)$$

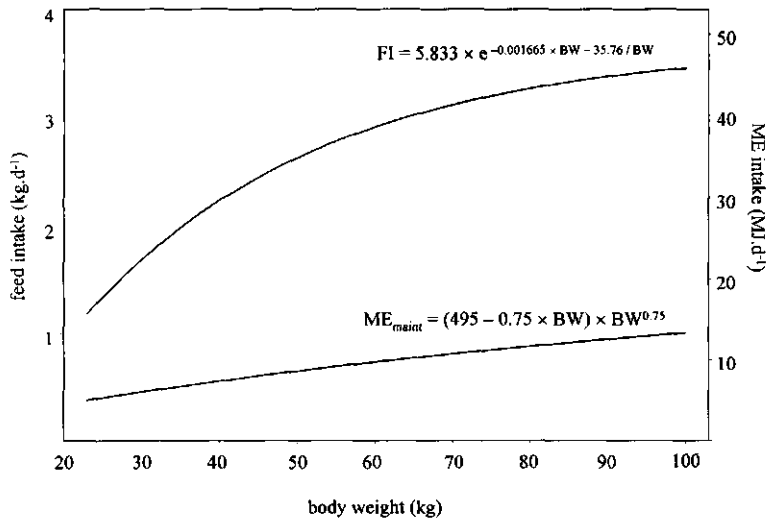

Figure 2 The course of simulated feed intake (FI, after Kanis and Koops, 1990) and ME intake, and of simulated maintenance ME requirements (ME_{maint}, after De Greef, 1987), in relation to body weight. Diet is assumed to contain 13.26 $MJ.kg^{-1}$.

The amount of non-protein energy available for production is now calculated as:

$$nonprotME_{prod} = ME_{+deam} - ME_{maint,indep} - ME_{turn,pres} \quad (7)$$

Then, after P_{dep} has been determined, the amount of energy left available for production can be calculated as:

$$ME_{prod} = nonprotME_{prod} + 23.6 \times P_{dep} - ME_{turn,dep} + ME_{P_{dep}} \times (P_{prod} - P_{dep}) \quad (8)$$

This process separates ME_{maint} into two parts: a fixed one ($ME_{maint,indep}$) and one ($ME_{turn,pres}$) that varies with body protein content and composition. Moreover, the initially fixed value for ME_{Pdep} of 53 $MJ.kg^{-1}$ is made dependent on the composition of P_{dep}, which changes during development. Deviations from the average value of this variable portion ($ME_{turn,dep}$) would be allocated to ME_{maint} in a fixed system. In equations (7) and (8), the original ME_{maint} has effectively been replaced by $ME_{maint,indep} + ME_{turn,pres} + ME_{turn,dep}$.

When the simulated value of the ratio of lipid to protein deposition L_{dep}/P_{dep} turns out to be lower than its required physiological minimum, P_{dep} is recalculated in an alternative way; the denominator term of the corresponding formula (expressing the gross ME requirements per gram deposited protein) was extended by a factor $ME_{turn,dep} / P_{dep}$ to allow for the ME costs of repeated synthesis of newly deposited protein.

Parameterisation

As a result of the limited availability of quantitative information on protein turnover processes, the above described extensions of the model (Moughan and Smith, 1984) require three mutually dependent key parameters to be quantified before the model can be implemented: the fractional rate of synthesis of newly deposited protein (FRS_{dep}), the turnover rates of already

deposited protein in the various pools ($TR_{pres,i}$), and the part of ME_{maint} that is allocated to protein turnover processes (1 – $FrcME_{maint}$). Together with parameters such as the maximum daily protein deposition ($P_{dep,max}$) and the minimum lipid to protein deposition ratio, these parameters define a multidimensional space of values that should each be restricted within certain boundaries. This "permissible" subspace restricts each of the parameters to a range (ideally, a narrow one) within which it is consistent with the values of the other ones. The extended model may be parameterised by quantifying these ranges. This can be done by varying these parameters between (and somewhat beyond) their likely extreme values, and evaluating the critical output variables of the model (such as L_{dep}, P_{dep}, and heat production [HP]) in order to determine which combinations lead to realistic results.

For that purpose, the extended model was evaluated repeatedly over a (cumulative) body weight range from 23 to 100 kg. The parameters to be approximated are FRS_{dep} and $FrcME_{maint}$. FRS_{dep} was varied, from its theoretical minimum of unity to a value that was

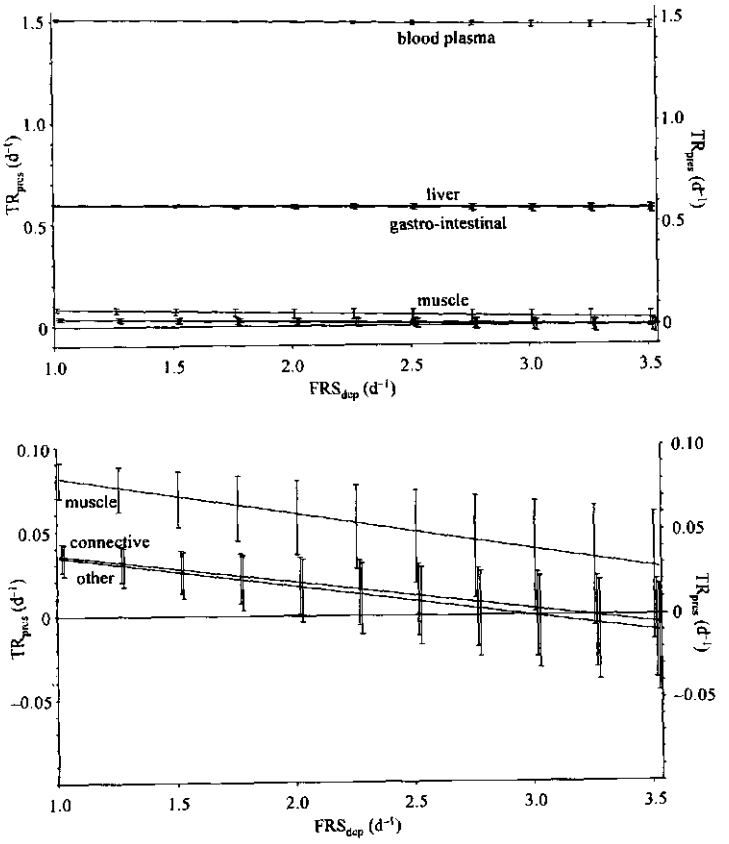

Figure 3 The course of simulated TR_{pres} values for the various protein pools as a function of simulated FRS_{dep} values. The vertical bars indicate the ranges resulting from different simulated feeding levels and $FrcME_{maint}$ and $P_{dep,max}$ values. The lower plot magnifies the bottom part of the upper plot.

found in preliminary runs of the model to be too high, over the range [1.00, 1.25, ..., 3.25, 3.50] d^{-1}. The same preliminary runs suggested likely values for $FrcME_{maint}$ around 0.7; hence this parameter was varied over the range [0.50, 0.55, ..., 0.80, 0.85].

Simultaneously, the values of $P_{dep,max}$ and of the feeding level (the model variables that are most important for determination of nutrient availability and allocation) were varied over [100, 125, ..., 225, 250] g d^{-1} and [0.6, 0.7, ..., 1.1, 1.2] times the originally simulated intake, respectively. This originally simulated feed intake level corresponds to the unrestricted intake level of individually housed growing (Yorkshire × Landrace) castrates that was fitted by Kanis and Koops (1990) as a continuous function of body weight: see Figure 2. Over the simulated body weight range from 23 to 100 kg, this intake covered the simulated maintenance requirements more than threefold.
The function is clearly invalid outside this range: its predicted feed intake is lower than ME_{maint} below 11 kg body weight.
There were thus 11 × 8 × 7 × 7 = 4312 replicates. All other model parameters were kept constant at values taken from Moughan and Smith (1984), Moughan (1985), and Moughan and Verstegen (1988); the minimum lipid to protein deposition ratio was set to unity, and for $FRS_{overall,i}$ the values of Riis (1983a, table 5.5) were adopted (0.10, 0.05, 0.60, 1.50, 0.60 and 0.05 d^{-1} for the above mentioned protein pools, respectively).

$TR_{pres,i}$ versus FRS_{dep}

The simulated values for $TR_{pres,i}$ at a total body protein mass of 7.3 kg (for comparability with the values from Riis, 1983a, used in equation (3)) have been plotted against the corresponding ones for FRS_{dep} in Figure 3.

During the "day" in which this protein mass was reached, the simulated values of $P_{dep,i}$ / $P_{pres,i}$ (equation (3)) for the various pools showed distributions as given in Table 1. At this stage of development, daily protein deposition turns out to comprise 1 to 2 % of the already present body protein mass. About 77 % of the variation in this fraction between replicates was caused by the variation in feeding level and in $P_{dep,max}$, and to a lesser extent in $FrcME_{maint}$.

Table 1 Distribution characteristics of ($P_{dep,i}$ / $P_{pres,i}$) at 7.3 kg body protein mass over all simulation replicates

Protein pool	minimum	average	maximum
Muscle	0.0100	0.0210	0.0339
Connective tissue	0.0075	0.0158	0.0256
Liver	0.0051	0.0107	0.0174
Blood plasma	0.0077	0.0163	0.0264
Gastro-intestinal	0.0060	0.0126	0.0204
Other	0.0082	0.0172	0.0278

Figure 3 reveals a clearly negative relation between $TR_{pres,i}$ and FRS_{dep}: these variables have to fit together into the space provided by $FRS_{overall}$. The value for blood plasma protein was affected most: the simulated differences in FRS_{dep} caused ca 85 % of the variation in TR_{pres} for this pool; for the other pools, this value ranged from 62 to 71 %.
The simulated variations in $P_{dep,max}$ and the feeding level caused together 20 to 27 % of the variation in $TR_{pres,i}$ values (8 % for plasma proteins); this variation is visualised in Figure 3 by

the vertical bars at each point. A relation between $FrcME_{maint}$ and $TR_{pres,i}$ was virtually absent.

It follows from Figure 3 that TR_{pres} for connective tissue reaches a substantial proportion of very low values when FRS_{dep} exceeds 2.5 d^{-1}. For FRS_{dep} around 3.25 d^{-1}, this pool's TR_{pres} becomes even negative on average. Although the turnover of present connective tissue protein is expected to take place at a very low rate, the turnover rate can clearly not be (less than) zero. Hence the 2.5 d^{-1} value must be regarded as an operational upper level for FRS_{dep}.
The simulated values for the pool of "other" proteins provide even more rigourous limitations.

Deposition and heat production versus FRS_{dep} and $FrcME_{maint}$

As a tentative reference, the original model (Moughan and Smith, 1984) has been evaluated over the same ranges of the "independent" variables ($P_{dep,max}$ and feeding level, amounting to $7 \times 7 = 49$ replicates). Using the original model as a reference implies that corresponding parameter combinations within the extended model are required to result in the same partitioning of ME over deposition processes and maintenance as predicted by the model with constant ME_{maint}.

The growth curves of P_{dep}, L_{dep} and HP served as evaluation criteria. For this comparison, these variables and simulated body weight on each "day" were combined into quadratic regression coefficients of P_{dep}, L_{dep} and HP on body weight for the 4312 "extended" plus 49 "original" replicates. At each of the 78 points in a body weight range over [23, 24, ..., 99, 100] kg, these coefficients were then used to calculate P_{dep}, L_{dep} and HP values for the extended model and for the original model with the same $P_{dep,max}$ and feeding level input (leading to HP_{extd} and HP_{orig}, respectively, etc.).

This leads to sums of squares like $SS(HP) = \sum_{i=23}^{100} (1 - \frac{HP_{extd,i}}{HP_{orig,i}})^2$, and analogous expressions for $SS(P_{dep})$ and $SS(L_{dep})$. Distribution characteristics of these variables are in Table 2. $SS(P_{dep})$ and especially $SS(L_{dep})$ showed very skewed and tailed distributions. $SS(P_{dep})$ shows much lower mean values than $SS(L_{dep})$ and SS(HP); this may reflect the fact that the latter entities are, more or less, derivations of P_{dep} in the simulation model.

The parameter MeanDev was derived for summarisation:

$$MeanDev = \frac{\frac{1}{3} \times SS(P_{dep}) + \frac{1}{3} \times SS(L_{dep}) + \frac{1}{3} \times SS(HP)}{100 - 23} \qquad (9)$$

Low values for MeanDev indicate a high degree of similarity in the output variables of the extended *versus* the original model. The distribution of this parameter was found to be very skewed with a heavy tail, as shown in Table 2.
Its relation to FRS_{dep} and

Table 2 Distribution characteristics of variables from replicates of the extended model compared to corresponding values from the original model

Variable	minimum	median	maximum	average
$SS(P_{dep})$	0	0.0007	0.0953	0.0054
$SS(L_{dep})$	0	0.0066	93	0.3144
SS(HP)	0	0.0056	0.4955	0.0221
MeanDev	0	0.0072	31	0.1140

$FrcME_{maint}$ depends on the input values of $P_{dep,max}$ and feeding level. Our problem is then to find the MeanDev minimum in a four-dimensional response surface, two dimensions of which (FRS_{dep}, $FrcME_{maint}$) are of primary interest for our present purposes. As is illustrated in Figure 4, the other two (feeding level, $P_{dep,max}$) cause a large variation which is, again for our present purposes, nuisance variation; most of this variation occurs outside the area of interest (i.e. outside the area of minimum MeanDev values), so for a proper estimation of surface minima the data should be adjusted for it.

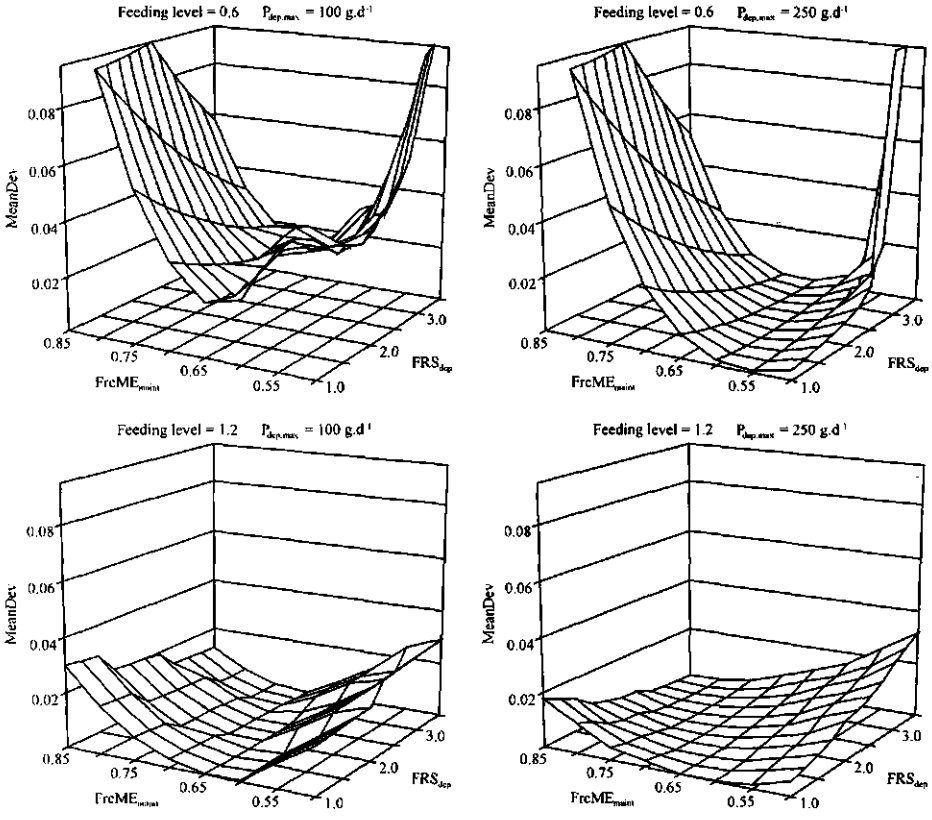

Figure 4 The course of simulated MeanDev values in relation to simulated $FrcME_{maint}$ and FRS_{dep} values, for extreme combinations of the feeding level and $P_{dep,max}$. The cases with the 5 % highest MeanDev values are not shown.

The response surfaces were analyzed following Neter *et al.* (1985) with the RSREG procedure of SAS (1990c). A first analysis used the regression model

$$\begin{aligned} \text{MeanDev} = {} & b_0 + b_1 \times FRS_{dep} + b_2 \times FrcME_{maint} + b_3 \times P_{dep,max} + b_4 \times \text{feeding level} + \\ & + b_5 \times (FRS_{dep})^2 + \ldots + b_8 \times (\text{feeding level})^2 + \\ & + b_9 \times (FRS_{dep} \times FrcME_{maint}) + \ldots + b_{14} \times (P_{dep,max} \times \text{feeding level}) \end{aligned} \quad (10)$$

Canonical analysis revealed that the response surface has one moderately weak "hill" orienta-

tion (with an eigenvalue (EV) of –0.21, associated with the feeding level) and three "valley" orientations (a strong one with EV = 0.50, positively associated with $P_{dep,max}$ and negatively with $FrcME_{maint}$; a weak one with EV = 0.11, positively associated with both $P_{dep,max}$ and $FrcME_{maint}$; and an essentially flat one with EV = 0.003, associated with FRS_{dep}).
In accordance with this, the linear and quadratic terms of FRS_{dep} and most of its crossproducts were non-significant ($0.31<P<0.99$), whereas the linear term of $FrcME_{maint}$ was non-significant ($P=0.56$) but its quadratic term and crossproducts were strongly significant ($P<0.002$).

To adjust for the "nuisance variation" caused by $P_{dep,max}$ and the feeding level, the inverse values of Cook's distance values produced by model (10) were used as weighting factors in the regression model

$$\begin{aligned} MeanDev = {} & b_0 + b_1 \times FRS_{dep} + b_2 \times FrcME_{maint} + \\ & + b_3 \times (FRS_{dep})^2 + b_4 \times (FrcME_{maint})^2 + \\ & + b_5 \times (FRS_{dep} \times FrcME_{maint}) \end{aligned} \qquad (11)$$

This weighted regression fitted the data well ($R^2 = 0.97$), and predicted an overall minimum value of –0.016 for MeanDev at $FrcME_{maint} = 0.6447$ and $FRS_{dep} = 2.86\ d^{-1}$. The latter value does not agree with the above derived requirement that FRS_{dep} should be below $2.5\ d^{-1}$; apart from that, the non-significant effect of FRS_{dep} on MeanDev makes the estimate quite meaningless.
Therefore each subset of $P_{dep,max}$ by feeding level was analysed separately with model (11) without weighting. This resulted in 49 combinations of $FrcME_{maint}$ and FRS_{dep} where MeanDev attains its predicted minimum. Nine of these combinations required FRS_{dep} to be either negative or much higher than the 3.5 upper limit of our evaluation range, which is in line again with the lack of significance of this effect. Within our evaluation range, the linear regression equation $FRS_{dep} = -3.088 + 7.811 \times FrcME_{maint}$ was obtained ($r = 0.83$). Solving this equation for $FrcME_{maint} = 0.6447$, as found above, gives a FRS_{dep} value of $1.948\ d^{-1}$.

Parameterisation: FRS_{dep}, $TR_{pres,i}$ and $FrcME_{maint}$

In view of the above mentioned results, it seems to be a reasonable compromise to parameterise the extended model with values $FrcME_{maint} = 0.65$ and $FRS_{dep} = 2.0\ d^{-1}$. The corresponding average values for $TR_{pres,i}$ are 0.060, 0.019, 0.585, 1.492, 0.582, and $0.016\ d^{-1}$ for muscle, connective tissue, liver, blood plasma, gastro-intestinal and "other" proteins, respectively.

Discussion

In this study, we attempted to integrate present (far from complete) quantitative knowledge of protein turnover into a dynamic growth simulation model. The possibility is thereby created to evaluate a wide range of combinations of values of the model parameters. Ideally, this may point to a narrow subspace of such combinations from which the model predicts a realistic set of output variables; it may be reasonable to hold that this subspace reflects the true state of nature.
The simulation results in Figure 3 indicate that FRS_{dep} is likely to be smaller than $2.5\ d^{-1}$:

beyond that value, TR_{pres} values of some protein pools (most notably, connective tissue) are required to be unrealistically low in order to make the energetic budget balance.
When comparing the extended model to the original one with respect to the simulated P_{dep}, L_{dep}, and HP growth curves, it can be concluded that the two sets of output variables show similar patterns for certain combinations of FRS_{dep} and $FrcME_{maint}$, and that these combinations differ for different values of $P_{dep,max}$ and, especially, the feeding level.
These similar patterns occur for the various combinations of the feeding level and $P_{dep,max}$ around an $FrcME_{maint}$ value of 0.65, coinciding with an FRS_{dep} value of 2.0 d^{-1}
The method followed to obtain these parameters was quite straightforward; however, some remarks must be made.

Turnover rates of protein pools

As a consequence of the scarcity of quantitative information on protein turnover rates, it is difficult to asses the extent to which the initial $FRS_{overall}$ values (taken from Riis, 1983a) reflect the true state of nature. Compared to other reports (e.g. Simon, 1989, table 9.18), the adopted values for muscle, liver, and gastro-intestinal proteins seem high, whereas the value for "other" proteins seems low.
Riis (1983a) comments on his own approximations as follows: "The whole body synthesis shown in table 5.5 [...] is larger than the values reported by Reeds *et al.* (1978) for 34-kg pigs [...] The calculated values may be an overestimation. The value used for fractional rate of muscle protein synthesis is presumably at the upper limit of turnover rates for this pool. On the other hand, the values obtained by Reeds *et al.* appear low when compared with other values in table 5.3 and may be underestimations [...] Turnover rates of muscle and presumably liver proteins show large diurnal variations in meal-fed animals [...] Differences in time of start and termination of infusion may contribute to apparent discrepancies in turnover rate values."
Simon (1989) stresses that "the constant infusion method underestimates the protein synthesis rates in organs with high secretory activities like liver, pancreas, and tissues of the digestive tract; this is related to the fact that it is mainly proteins retained in the tissues that are measured. By contrast, when the large dose technique, which measures also the synthesis of secretory proteins, is used fractional synthesis rates close to 100 % for liver and small intestinal tissues were calculated. These values were at least 50 % higher than estimates with the continuous infusion technique under comparable conditions [...] In addition, protein synthesis in [secretory organs] seems to be influenced by various external factors; when tissue protein synthesis rates estimated by almost the same method in pigs in different experiments [...] are compared, a high variability of the fractional protein synthesis rates in liver, pancreas, and tissues of the upper digestive tract and reasonable agreement for the values estimated in other tissues may be observed."
And Reeds (1991) points to "uncertainties as to the quantitative accuracy of the currently available measurements of protein synthesis *in vivo*. When problems associated with structural and kinetic heterogeneity of amino acid pools are considered [...] in addition to the difficulty of measuring the synthesis of rapidly turning over proteins [...], one is led to conclude that current estimates of protein synthesis *in vivo* may be underestimates."
To obtain some idea of the sensitivity of the derived parameters for changes in the initial

$FRS_{overall}$ values we have run the above procedure with (possibly more "conservative") $FRS_{overall}$ values of 0.05, 0.05, 0.40, 1.50, 0.40, and 0.20 d^{-1} for muscle, connective tissue, liver, blood plasma, gastro-intestinal, and "other" proteins, respectively. Following the same steps as before, TR_{pres} of muscle protein showed some negative values beyond an FRS_{dep} level of 2.0 d^{-1}. Plots equivalent to Figure 4 were shaped very much the same as in that figure, but were shifted to somewhat higher $FrcME_{maint}$ levels. The most likely approximations of FRS_{dep} and $FrcME_{maint}$ turned out to be 1.76 d^{-1} and 0.73, respectively; the corresponding mean TR_{pres} values were 0.017, 0.025, 0.387, 1.496, 0.384, and 0.175 d^{-1} for the respective protein pools. The true state of nature will probably be somewhere between these figures and the ones derived initially; but in view of the above considerations of Riis (1983a), Simon (1989) and Reeds (1991), the initial set may be a reasonable compromise.

Size of protein pools

It is almost inevitable in simulation models such as the one used here that the minor classes of some attribute of the simulated system is collected together into a class of "miscellaneous items"; the choice that has to be made is one between loss of information *versus* unnecessary detail. Riis's (1983a) pool of "other" proteins forms a good example of such a residual class that suffers from some lack of realism. The pool contains just below 10 % of total body protein mass; this might be regarded as a quite large "miscellaneous" group. Riis (1983a) assumed its turnover rate to be low, but this may be debatable in view of the above discussion by Simon (1989). So, the fact that it is this pool of "other" proteins that first imposes restrictions on the value of FRS_{dep} in Figure 3 may throw little light on the matter; it may be wise to ignore the pool's data, rather than to draw restrictive conclusions from them.

It must be stressed once again that the pool growth curve parameters in Figure 1 do not pretend to have general validity. The five literature sources from which they have been compiled differ greatly, for example with regard to the muscle protein to connective tissue protein ratio; in Jørgensen *et al.* (1985) the amount of muscle protein at the beginning of the considered trajectory is equal to the amount of connective tissue protein, whereas in Pfeiffer *et al.* (1990), it is about twice as large. Likewise, the pools' growth curves are almost linear in Metz *et al.* (1980) and Tullis (1981), whereas they are strongly curved in Susenbeth and Keitel (1988) and Pfeiffer *et al.* (1990). Thus, the values in Figure 1 may give a satisfactory description of some "average" genotype only, which is convenient for our present purposes; when simulating a specific pig population, it is likely to be worthwhile to estimate these parameters explicitly.

A very weak point in this study is the incomplete approximation of the growth curves of liver and especially gastro-intestinal proteins. The one available reference to liver protein (Pfeiffer *et al.*, 1990) may hardly be considered to be generally valid (see the above paragraph), and information on gastro-intestinal proteins is even more scarce. The adopted values for the regression coefficients of –0.01 per ln(kg) are quite likely to be inaccurate, but they seemed to be the best estimate we were able to make at present. More or less surprisingly, the simulated system turned out to be quite insensitive to the values of these regression coefficients; when using regression coefficients derived from the first three of the quoted references, approximated turnover parameters were about the same as the ones reported above.

Turnover of present and newly deposited protein

It may seem strange that a positive relation between FRS_{dep} and $FrcME_{maint}$ (as found from the subset analysis with model (11), see above) is required to delimit the subspace where the MeanDev parameters reach their minima: high FRS_{dep} levels lead to high ME requirements per unit of protein deposition (equation (5)), which may be expected to have to "compete" with the turnover-*independent* maintenance requirements when the amount of energy available for production is determined (equations (7) and (8)). Thus, one might expect a negative relation between FRS_{dep} and $FrcME_{maint}$.
But ME_{Pdep} has been reduced in the extended model to compensate for the explicit effects of equation (5); moreover, $ME_{turn,pres}$ (equation (4)) is two to three times as large as $ME_{turn,dep}$ (equation (5)) because P_{pres} is so much larger than P_{dep} (Table 1), and the relation between FRS_{dep} and TR_{pres} is strongly negative (Figure 3). As far as any "competition" is going on, it is between the requirements for turnover of present protein on the one hand and those for repeated synthesis of deposited protein and turnover-independent maintenance processes on the other (equation (7)). Of course, this is a *contradictio in terminis*, resulting from the parameterisation constraints. It is a consequence of assuming a fixed ME_{maint} formula that turnover-independent maintenance and the total turnover requirements are more or less required by the model to fit into the same space. In reality they are expected to be independent. This will be considered in more detail later; the present study focuses on estimation of values of the model parameters.

A central assumption in our approximation is the concept of a constant value for FRS_{dep} for all considered protein pools: for example, we assume that the deposition of a unit of connective tissue protein is accompanied by as many repeated synthesis-and-breakdown processes as the formation of a molecule of blood plasma protein. There seems to be little, if any, experimental evidence to support or reject this assumption; it seems simply logical that the probability of erroneous peptide bond formations is as large for one protein type as it is for another, irrespective of the life cycle of the protein in a later stage.

Some more insight into this phenomenon is provided by the variation in simulated TR_{pres} values (which is variation over the various $P_{dep,max}$ levels and feeding levels) at a given FRS_{dep} level, as visualised by the vertical bars in Figure 3. As is illustrated in Figure 5 for connective tissue protein, TR_{pres} values close to or below zero appear exclusively at combinations of high $P_{dep,max}$ and high feeding levels (i.e. circumstances under which P_{dep} / P_{pres} in equation (3) may become large). This effect is more pronounced at the high FRS_{dep} level. The latter suggests that genotypes with a high potential for protein deposition (i.e. high P_{dep} / P_{pres} levels) may derive this partly from a more efficient protein deposition metabolism, in terms of reduced FRS_{dep}, in at least some protein pools.

Of course, the approximations that were derived for $FrcME_{maint}$ and FRS_{dep} are bounded within the ranges that were evaluated during simulation (e.g. values for FRS_{dep} above 3.5 d^{-1} would not have been found). The literature is very scarce with experimental values for comparison.
Total simulated ME requirements for protein turnover (i.e. $ME_{turn,pres} + ME_{turn,dep}$) comprised 16 % (at $P_{dep,max}$ = 100 $g.d^{-1}$ and the 1.2 feeding level) to 26 % (at 250 $g.d^{-1}$ and 0.6) of total

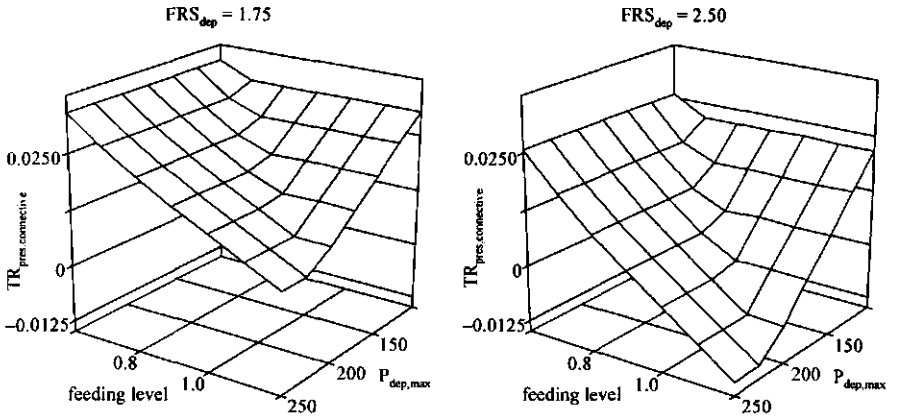

Figure 5 The course of simulated TR_{pres} values for connective tissue protein, in relation to the feeding level and $P_{dep,max}$, for two levels of FRS_{dep}.

HP; similar fractions were obtained by Gill *et al.* (1989) when simulating protein metabolism of growing lambs of 20 kg body weight.

Simulated $ME_{turn,pres}$ values (equation (4)) ranged from 139 (at 100 $g.d^{-1}$ and 1.2, see above) to 165 (at 250 $g.d^{-1}$ and 0.6) $kJ.kg^{-0.75}.d^{-1}$. After division by the 3.92 MJ ME per kg protein required for the arrangement of peptide bonds (see the **Model** section), this suggests an average protein synthesis of 36 to 42 g $kg^{-0.75}$ d^{-1} for turnover of already present body protein. This value is about twice as high as the "total body protein synthesis at nitrogen equilibrium" in underfed immature rats, pigs, and cattle summarised by Reeds (1989, table 1). Over our simulated body weight range of 23 to 100 kg, the average metabolic body weight was about 21 $kg^{0.75}$; the body contains about 9 kg protein at that stage, i.e. about 430 g protein per $kg^{0.75}$. Our simulated values thus imply that somewhat less than 10 % of total body protein mass is recycled daily, which corresponds neatly, of course, to the weighted average of the TR_{pres} parameters we obtained, which is 0.092 at 9 kg body protein mass.
This suggests that either our TR_{pres} parameters are gross overestimates (although even the weighted average of the more "conservative" set described in the second section of this **Discussion** section is 0.073), or the turnover of already present body protein in growing pigs is grossly underestimated by measuring the protein synthesis in immature, maintenance-fed animals. As Reeds *et al.* (1980) put it, when discussing the above mentioned results on underfed immature pigs, "the physiological significance of results obtained in such animals is debatable".

The derived value of FRS_{dep} = 2.0 d^{-1} may seem to be the simple result of the fact that its range was effectively restricted to lie between FRS_{dep} = 1.0 and 2.5 d^{-1}, and to be just about the mean of these extremes. But the surface analysis algorithm was free in its choice of the minimum MeanDev levels (Figure 4), irrespective of the corresponding $FrcME_{maint}$ or FRS_{dep} values. MeanDev values close to zero just turn out to be found over the whole simulated FRS_{dep} range, and in the subset analysis with model (11) even beyond it (see above). $FrcME_{maint}$ is a much more restrictive parameter; its allowed range is not fully covered.

The simulated values of $P_{dep,max}$ are expected to cover the range found in common European (and derived) pig breeds. $P_{dep,max}$ values reported in the scientific literature have often been measured in experimental populations that had been isolated from commercial genetic improvement for veterinary reasons (or simply for convenience). For such genotypes, $P_{dep,max}$ is reported to vary roughly between 90 and 150 $g.d^{-1}$ (Moughan and Verstegen, 1988). In contrast, Campbell and Taverner (1988), Whittemore *et al.* (1988), Rao and McCracken (1991), and De Greef and Verstegen (1992) report P_{dep} values between 180 and 210 $g\ d^{-1}$, measured in stock from commercial breeding programmes.

The simulated growth curves of the protein pools and the approximated values for FRS_{dep} and

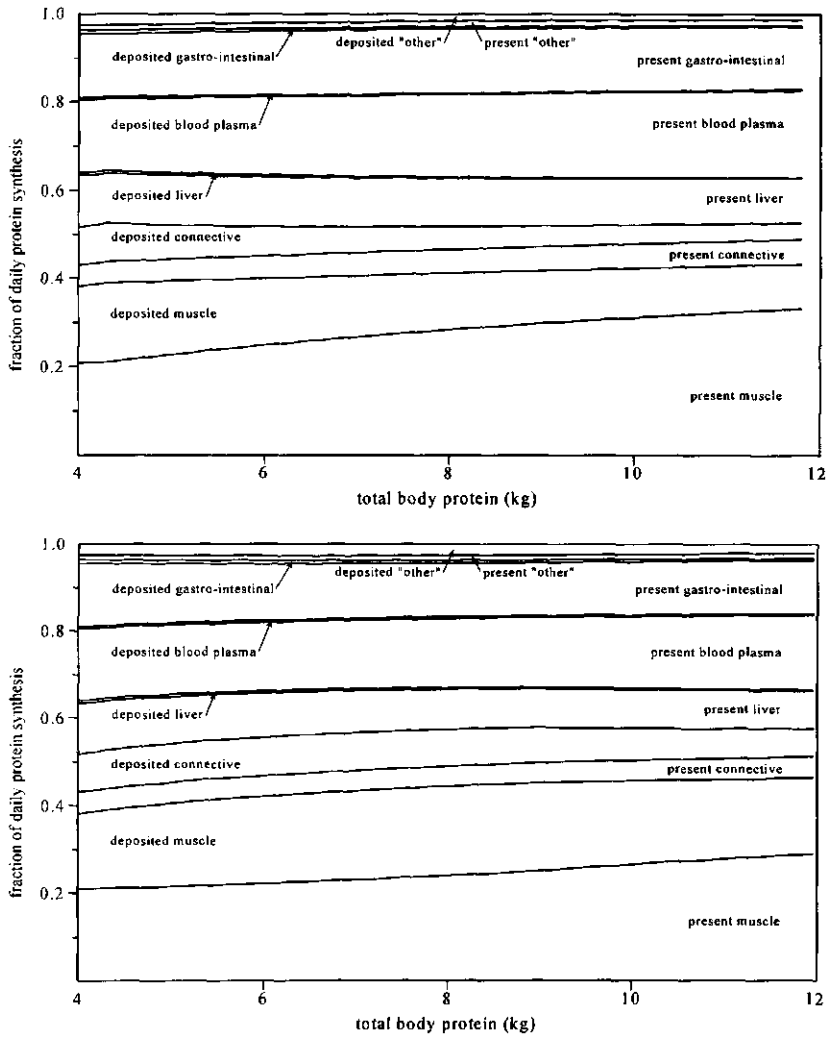

Figure 6 The cumulative distribution of protein synthesis over turnover of present protein pools and repeated synthesis of newly deposited protein, in relation to total body protein mass and $P_{dep,max}$. Top: $P_{dep,max} = 100\ g.d^{-1}$, bottom: $P_{dep,max} = 200\ g.d^{-1}$.

TR_{pres} can be used to produce Figure 6. In this cumulative plot, the proportion of total daily protein synthesis that is caused by the turnover of the already present amount of each pool protein, or by the repeated synthesis-and-breakdown connected to its growth, is shown in dependence of the total body protein mass. It is clear that the deposition processes are much more important (in a quantitative sense) at the high $P_{dep,max}$ level. And while the turnover of present muscle protein covers the largest fraction, it appears that the turnover of present blood plasma and gastro-intestinal proteins pose at least the same requirements as the deposition of new muscle protein.

When recognising that P_{dep} is more or less negligible in comparison to P_{pres} (Table 1), equation (2) can be simplified to $FRS_{overall,i} \approx TR_{pres,i} + \frac{P_{dep,i}}{P_{pres,i}} \times FRS_{dep}$. The term $\frac{P_{dep,i}}{P_{pres,i}} \times FRS_{dep}$ ranges from 0.02 to 0.04. Compared to $TR_{pres,i}$, which range from 0.02 to 1.49, this is very large to negligibly small. Figure 6 illustrates that the major part of overall turnover of gastro-intestinal, liver, and blood plasma proteins, as it can be measured experimentally in growing pigs, is caused by resynthesis of already present protein; for muscle and connective tissue proteins, a large part of it is caused by repeated synthesis of newly deposited protein.

Feeding level

The feeding level appeared to be an "independent variable" with much influence on most of the evaluated model parameters. However, it must be noted that the feed allowance simulated in this study is nothing more than a continuous system of rationing to body weight; true *ad libitum* feed intake is a quite different phenomenon, as it requires between-animal variation of intake, independent of body weight. Actually simulating *ad libitum* feed intake in a realistic way, if possible at all, is far beyond the scope of this study.

Acknowledgements

This study was started in 1989 as JWS's MSc thesis, which was supervised by Martin Verstegen. Subsequently, valuable contributions were made by Paul Moughan, John Woolliams, Ella Luiting, Gerry Emmans and Gertruud Bakker. Henry Jørgensen, Andreas Susenbeth and Helmuth Pfeiffer kindly made available the data used in Appendix II.

Appendix I. An outline of a generalised pig growth simulation model.

The model used in this study (Moughan and Smith, 1984) was referred to in the review by Moughan and Verstegen (1988) as a "deterministic model", describing "the basic nutrient partitioning process as being regulated by a few simple biochemical and physiological control points". They state that models of this type "can be used to predict commercially important measures of animal performance, in face of variation in factors such as genotype, sex of the animal, level and quality of nutrition and physical climate". The adapted "generalised flow diagram" in Figure 7 depicts the nutrient digestion and utilisation processes (and some of the physiological interactions) that are considered in this model.

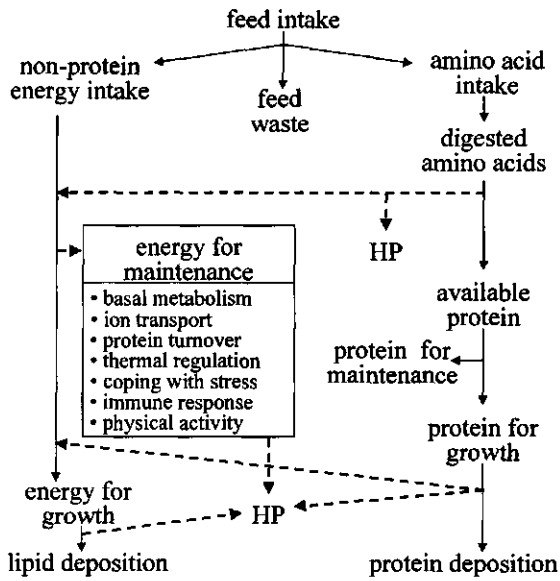

Figure 7 A generalised flow diagram of the Moughan simulation model (adapted after Moughan and Verstegen, 1988).

Aspects of the computer algorithm of our modified version of this model that are necessary for the understanding of the present text have been coded below.

start
read feed composition (ME, CP, amino acids)
read feed intake curve parameters (a_0, a_1, a_2)
read amino acid pattern of "ideal" protein
read ME requirement parameters for body maintenance (b_1, b_2)
read ME costs of protein and lipid deposition (ME_{Pdep}, ME_{Ldep})
read ME yield of protein deamination (ME_{Pdeam})
read maximum protein deposition ($P_{dep,max}$)
read minimum lipid to protein deposition ratio ($R_{L/P,min}$)
read body weight at start and end (BW_{start}, BW_{end})
determine chemical score of feed CP, ME in feed CP
determine amounts of balanced and deaminated protein
determine non-protein ME per g feed
set start values for body protein, lipid, inorganic matter (P, L, ash)
$BW = BW_{start}$
days = 0

while $BW < BW_{end}$ *do*

$FI = a_0 \times e^{a_1 \times BW - a_2 / BW}$

determine intake of "balanced" and deaminated protein

determine non-protein ME intake

determine protein requirements for body maintenance (P_{maint})

P_{prod} = balanced protein – P_{maint}

$ME_{maint} = (b_1 \times BW + b_2) \times BW^{0.75}$

$P_{dep} = min(P_{prod}, P_{dep,max})$

ME_{+deam} = non-protein ME + deaminated protein × ME_{Pdeam}

$ME_{prod} = ME_{+deam} - ME_{maint} + 23.6 \times P_{dep} + ME_{Pdeam} \times (P_{prod} - P_{dep})$

$$L_{dep} = \frac{ME_{prod} - P_{dep} \times ME_{P_{dep}}}{ME_{L_{dep}}}$$

if $L_{dep} / P_{dep} < R_{L/P,min}$

$$P_{dep} = \frac{ME_{+deam} - ME_{maint} + P_{prod} \times ME_{P_{deam}}}{ME_{P_{dep}} - 23.6 + ME_{L_{dep}} \times R_{L/P,min} + ME_{P_{deam}}}$$

$L_{dep} = P_{dep} \times R_{L/P,min}$

end if

$HP = ME - 23.6 \times P_{dep} - 39.7 \times L_{dep}$

determine ash deposition (ash_{dep})

days = days + 1

$P = P + P_{dep}$

$L = L + L_{dep}$

$ash = ash + ash_{dep}$

determine water mass (H_2O)

$BW = P + H_2O + ash + L$

end do

end

Appendix II. Partitioning of body protein of growing pigs over protein pools.

Tullis (1981) lists in the appendices of her thesis the 29 individual observations on the size of the various protein pools that were measured in pigs with a total body protein mass up to 18 kg (the more or less arbitrary range we consider here). The individual data behind the results published by Jørgensen *et al.* (1985), by Susenbeth and Keitel (1988), and by Pfeiffer *et al.* (1990) were provided by H.H. Jørgensen (personal communication, 1991; n=244 for muscle, connective tissue, blood, and entrails), A.J.J. Susenbeth (personal communication, 1992; n=40 for muscle and connective tissue), and H. Pfeiffer (personal communication, 1992; n=36 for muscle, connective tissue, blood, liver, stomach, and entrails). The data of Metz *et al.* (1980; n=21) comprise muscle, connective tissue, and entrails.

Bone, fatty tissue, skin, hair, ears, and claws were combined into "connective tissue". "Muscle" includes intermuscular fatty tissue in Susenbeth and Keitel's (1988) data; it does not in the data from Metz *et al.* (1980), Jørgensen *et al.* (1985), and Pfeiffer *et al.* (1990), whereas in Tullis's (1981) data, intermuscular fat could be separated out by making some assumptions on its lipid content.

The percentages of body protein in muscle, connective tissue, blood and entrails were regressed on the natural logarithm of total body protein mass; the estimated regression coefficients are in Table 3.

Table 3 Regression coefficients (% per ln[kg]) of percentage of body protein in various protein pools on the natural logarithm of total body protein mass

	source[†]				
Protein pool	Metz	Tullis	Jørgensen	Susenbeth	Pfeiffer
Muscle	+3.186	+2.712	+7.658	+5.493	+8.521
Connective tissue	–0.371	–1.717	–4.885	–2.449	–3.787
Entrails	–2.110	–1.756	–2.541	–2.308	–4.690
Blood		+0.761	–0.232	–0.693	–0.047

[†] Metz *et al.*(1980); Tullis (1981); Jørgensen *et al.* (1985); Susenbeth and Keitel

The proportion of body protein that is present in the form of blood protein (which includes both plasma and blood cell proteins) shows little change when total body protein mass increases; it is the only pool for which both negative and positive regression coefficients are reported, and all these are close to zero.

The body fraction that is summarised with the term "entrails" includes not only the liver and the gastro-intestinal tract, but also the major part of the pool of "other protein" sources. Some studies include also the tongue (which is mainly muscle) and the ears, hairs, and/or abdominal fatty tissue (which is connective tissue) into this fraction. Nevertheless, the share of total body protein that is accounted for by this pool (and its course with increasing body protein mass) is rather uniform over the literature sources.

The muscle and connective tissue pools seem to complement each other, although the exact course with increasing total body protein varies greatly.

We have combined the data from these sources together and performed an overall regression analysis, weighting the records from each subset proportionally to the inverse of the standard error of the estimated regression coefficient for muscle protein. The results are in Table 4. These regression parameters are not fully consistent, because the data sets used did not all contain information on the same pools. But different weightings for the combined regression led to only marginally different results.

Table 4 Overall regression coefficients (% per ln[kg]) of percentage of body protein in various protein pools on the natural logarithm of total body protein mass

Protein pool	intercept	regression
Muscle	39.652	+7.314
Connective tissue	39.431	–4.525
Entrails	17.123	–2.581
Blood	3.751	–0.188

The courses of liver and gastro-intestinal protein growth are poorly documented. Oslage (1965) distinguished two fractions: *Innereien* ("innards", comprising not only the liver but also brain and spinal chord, tongue, heart, lungs and oesophagus, spleen, kidneys and abdominal fat) and *Abfall* ("offal", comprising not only the gastro-intestinal tract but also eyes and ears, claws, gall bladder, urine bladder and sexual organs); these two subsets show very similar courses with increasing total body protein mass: the equivalent regression coefficients are –2.068 and –2.321 % per ln(kg), respectively. In contrast, Pfeiffer *et al.* (1990) provided data on liver and stomach protein leading to equivalent regression coefficients of –0.856 and –0.162 % per ln(kg), respectively.

Muscle and connective tissue represent pools that are considered as such in this study, but the other ones are not. These figures must be recombined, sometimes in an arbitrary way, to derive realistic values for our parameterisation.

Describing a pig with a total body protein mass of 7.3 kg, Riis (1983a, Table 5.5) gives approximate figures for the distribution of this protein over the six pools: muscle (50.7 %), connective tissue (28.8 %), liver (2.74 %), gastro-intestinal (4.11 %), blood plasma (2.05 %), and "others" (11.6 %).
For the same total of 7.3 kg body protein, the regression parameters from Table 4 lead to corresponding values of 54.2 % for muscle, 30.4 % for connective tissue, 12.0 % for entrails (covering 7.0 % for *Innereien* and 5.0 % for *Abfall*), and 3.4 % for blood.
Riis's latter four pools correspond to our "entrails" plus "blood" pools, which contain 15.4 % of total body protein in our example pig. Within this set of "non-muscle, non-connective tissue" pools (= 100 %), 13.33, 20, 10, and 56.67 % are liver, gastro-intestinal, plasma, and "other" proteins, respectively (from Riis); *Innereien*, *Abfall* and blood cover 45.7, 32.3, and 21.8 %, respectively (from Table 4, and from Oslage, 1965).

Thus, Riis's pool of "other" proteins would contain (blood [Table 4] minus blood plasma [Riis]) + (*Innereien* [Oslage] minus liver [Riis]) + (*Abfall* [Oslage] minus gastro-intestinal [Riis]) = 56.7 % of this fraction, which is 8.7 % of total body protein mass (from Table 4). Liver, gastro-intestinal tract and blood plasma proteins would cover 2.1, 3.1 and 1.5 % of total body protein mass, respectively. Calculating the pool (*Innereien* minus liver) using the data for liver from Pfeiffer *et al.* (1990) would lead to 2.7 % and 8.1 % of total body protein mass covered by liver and "other" proteins, respectively. We will use the means of both sets here

(2.4 % and 8.4 %).

We have used the regression coefficients in Table 4, applying those of "blood" for blood plasma protein. The regression coefficients of *Innereien* [Oslage] and liver protein [Pfeiffer], and those of *Abfall* [Oslage] and stomach protein [Pfeiffer] were combined arbitrarily into round values of –1 % per ln(kg), both for liver and for gastro-intestinal protein. The corresponding intercept values can be derived as given in Figure 1; the values for "other" proteins follow from the constraint that intercepts should sum to 100 % and regression coefficients to zero.

The genotypes covered by these references vary widely with respect to fattening and slaughter characteristics; correspondingly, the differences between the regression parameters are sometimes quite large. It may be critical to determine these parameters specifically for the pig population to be simulated. The values in Figure 1 should be seen as generalisations, serving solely for parameterisation of this general study.

You know they say every tiny part of your body is replaced every seven years? Right. So... I've got a tattoo on my arm, right? Had it done eight years ago. So... how come it's still there? I mean, OK, new tiny bits of skin float in, but that means it ought to be all new and pink by now.

Terry Pratchett (1998) *Jingo*. Corgi, London; p. 250. Condensed.

Chapter 2 is based on

P.W. Knap (1996) Stochastic simulation of growth in pigs: protein turnover-dependent relations between body composition and maintenance requirements. *Animal Science* **63**:549-561

Chapter 2

Protein turnover-dependent relations with body composition

A dynamic model for simulation of growth in pigs, that was extended by a module to describe protein turnover, was made stochastic in order to simulate groups of pigs with between-animal variation in the maximum daily protein deposition ($P_{dep,max}$), in the minimum lipid to protein deposition rate ($R_{L/P,min}$), and in the distribution of body protein over protein pools (muscle, connective tissue, and other proteins). As a result, these simulated pigs show between-animal variation in body and body protein composition. This in turn leads to between-animal variation in energy requirements for protein turnover; this causes between-animal variation in maintenance requirements (ME_{maint}) as a result of variation in body composition.

Simulated population means for $P_{dep,max}$ were varied in seven steps from 100 to 250 $g.d^{-1}$, with a between-animal variation coefficient of 10 %; the feeding level was also varied in seven steps. Dependent on the levels of these input variables, 100 kg pigs showed within-population standard deviations in body protein and lipid content of 0.31 to 0.54 kg and 1.22 to 2.17 kg, respectively. ME_{maint} showed a protein-turnover-related within-population coefficient of variation of 1.4 to 2.0 %. Comparison over populations suggests that a 150 % increase in $P_{dep,max}$ (from 100 to 250 $g.d^{-1}$) would increase protein-turnover-related ME_{maint} by 11 to 15 %, from between 470 and 486 $kJ.kg^{-0.75}.d^{-1}$ to 541 $kJ.kg^{-0.75}.d^{-1}$.

The inferences that can be made from this with regard to experimental design are discussed.

Introduction

The algorithm described by Moughan and Smith (1984) and discussed more generally by Moughan and Verstegen (1988) for simulation of the protein and energy metabolism of growing pigs was extended in Chapter 1 to make explicit the ME requirements for body protein turnover. Using this extended model to simulate pigs growing from 23 to 100 kg body weight, likely values were approximated for the fraction of maintenance ME requirement independent of protein turnover processes ($FrcME_{maint}$ = 0.65), for the fractional rate of synthesis of newly deposited protein (FRS_{dep} = 2.0 d^{-1}), and for the turnover rate of already present protein (TR_{pres} = 0.060, 0.019, 0.585, 1.492, 0.582, and 0.016 d^{-1} for muscle, connective tissue, liver, blood plasma, gastro-intestinal and "other" proteins, respectively).

It was implicitly assumed in Chapter 1 that these turnover rates are constants that show no variation among individual animals, and there is little evidence to the contrary.
But total body protein mass is well known to show variation among animals of equal body weight. In addition, animals with the same total body protein mass do show variation in the proportions of total body protein present in the various protein pools (see the Appendix).
Therefore, the ME requirements for body protein turnover will vary among animals simply as a result of variation in body protein mass and its distribution over pools with different growth curves and turnover rates. When these ME requirements are regarded as part of the energy requirements for body maintenance (ME_{maint}), it follows that ME_{maint} will show protein turnover-related variation among animals as a result of variation in body composition.
In order to quantify this ME_{maint} variation, Moughan and Smith's (1984) model as extended in Chapter 1 was used in the present study for stochastic simulation of growing pigs with variable body composition and, as a result, variable ME_{maint}.

Methods

Introducing stochasticity

To simulate variable body composition, the simulation routine described in Chapter 1 was executed repeatedly with stochastic values for the model parameters $P_{dep,max}$ (the maximum attainable level of daily protein deposition, in $g.d^{-1}$) and $R_{L/P,min}$ (the minimum ratio of daily lipid to daily protein deposition, in $kg.kg^{-1}$). $P_{dep,max}$ values were obtained for each replicate (i.e. for each simulated animal) as

$$P_{dep,max} = \mu_{Pdep,max} + rannor_1 \times \sigma_{Pdep,max}$$

where $rannor_1$ is a random drawing from the standard Normal distribution (obtained using the RANNOR function supplied by SAS, 1990a), and $\mu_{Pdep,max}$ and $\sigma_{Pdep,max}$ are the assumed $P_{dep,max}$ population mean and standard deviation, respectively.

$R_{L/P,min}$ was varied accordingly as

$$R_{L/P,min} = \mu_{RL/P,min} + rannor_2 \times \sigma_{RL/P,min} \qquad (rannor_2 > 0)$$

$$R_{L/P,min} = \mu_{RL/P,min} \qquad (rannor_2 \leq 0)$$

which creates a skewed frequency distribution, as appropriate for a ratio.
Standard deviations were quantified by assuming constant coefficients of variation: $\sigma_{Pdep,max} = 0.10 \times \mu_{Pdep,max}$ and $\sigma_{RL/P,min} = 0.05 \times \mu_{RL/P,min}$.

The simulated fractions of total body protein present in various protein pools (muscle, connective tissue, liver, blood plasma, gastro-intestinal, and "other" proteins) were made dependent on the logarithm of total body protein mass as

$$FrcP_i = a_i + b_i \times \ln(P) \quad (1)$$

The intercept values (a) and the regression coefficients (b) follow from Table 4 of Chapter 1. $FrcP_1$ and $FrcP_2$ (for muscle and connective tissue protein, respectively) were made stochastic (and correlated to each other) by adding the terms $rannor_3 \times \sigma_{e1}$ and $(rannor_3 \times r_{12} + rannor_4 \times \sqrt{1-r_{12}^2}) \times \sigma_{e2}$, respectively. Here, $rannor_3$ and $rannor_4$ are independent random drawings from the standard Normal distribution, the standard deviations σ_{e1} and σ_{e2} are in Table 6 (Appendix), and the correlation coefficient r_{12} is in Table 7 (Appendix); these parameters have been summarized in Table 1.

Table 1 Parameters of equation (1), describing the distribution of body protein mass over pools

Protein pool i	a_i	b_i	σ_e †	r_{12} ‡
1 Muscle	0.3965	+0.07314	0.0244	–0.838
2 Connective tissue	0.3943	–0.04525	0.0190	
3 Liver	0.0955	–0.00601		
4 Blood plasma	0.0511	–0.01		
5 Gastro-intestinal	0.0435	–0.01		
6 "Other"	0.0191	–0.00188		

† Residual standard deviation among the regression lines
‡ Correlation coefficient between pools 1 and 2

The remaining protein pools are poorly documented; therefore, no attempt was made to treat them as was done for the muscle and connective tissue protein pools. Instead, the fraction of body protein that remains after $FrcP_1$ and $FrcP_2$ have been accounted for was divided over these pools proportionally to the levels that follow from their a and b values (Table 1); this leads to a stochastic proportion of "high-turnover" (liver, blood plasma and gastro-intestinal) plus "other" proteins, with fixed ratios among these four pools.

Forty-nine simulation runs were carried out with this stochastic model to generate 500 replicates (i.e. simulated animals, grown from 23 to 100 kg body weight) for each of the $7 \times 7 = 49$ combinations of $\mu_{Pdep,max}$ = [100, 125, ..., 225, 250] $g.d^{-1}$ and feeding levels at [0.6, 0.7, ..., 1.1, 1.2] times the "reference" feed intake (FI) of Chapter 1. The latter is a function of body weight (Kanis and Koops, 1990) that covers the maintenance energy requirement more than threefold (figure 2 of Chapter 1).
Each of the 49 simulations used the same set of $500 \times 4 = 2000$ random standard Normal drawings ($rannor_1$ to $rannor_4$); hence each of the 49 sets of replicates represents the same population of 500 simulated animals in terms of their deviations from the population means of $P_{dep,max}$, $R_{L/P,min}$, and body protein distribution over pools. To put it loosely, we compare otherwise identical populations of 500 animals at different $\mu_{Pdep,max}$ values (and $\sigma_{Pdep,max}$ values proportional to these) and fed at different feeding levels.

Simulation output of primary interest for the present study are the frequency distributions of protein deposition in the above mentioned pools and of lipid deposition, and the frequency distributions of ME requirements for turnover of already present and newly deposited body

protein ($ME_{turn,pres}$ and $ME_{turn,dep}$, respectively, which are parts of ME_{maint}).
Because mean levels and variation of protein and lipid deposition can be expected to be dependent on the simulated feed protein quality, this whole evaluation was carried out twice, with different simulated feed protein quality levels, as shown in Table 2. The amino acid composition of the feed protein used in Chapter 1 was, together with most of the other model parameters, based on Moughan (1985); as these figures may be regarded as insufficient for modern high-performance genotypes, this diet will further be referred to as the "low-quality protein diet". The contrasting "high-quality protein diet" was obtained by increasing the proportions of all essential amino acids in the feed protein by 62.5 % (which reduces the proportion of non-essential amino acids). The ME content of each diet was kept at 13.3 $MJ.kg^{-1}$, the crude protein content of each diet was kept at 19.8 % up to 50 kg body weight and 17.8 % afterwards.

Table 2 Gross amino acid contents (g per g feed) of the two simulated diets

	protein quality	
amino acid	low	high
lys	0.0088	0.0143
met+cys	0.0054	0.0088
try	0.0017	0.0028
his	0.0038	0.0062
phe+tyr	0.0125	0.0203
thr	0.0064	0.0104
leu	0.0113	0.0183
ile	0.0047	0.0076
val	0.0064	0.0104

Results

Comparison of the results from the simulated "low-quality" and "high-quality" protein diets revealed, as expected, somewhat higher (and more variable) P_{dep} and hence lower (and more variable) L_{dep} levels on the latter diet. As a result, $ME_{turn,pres}$ and $ME_{turn,dep}$ are somewhat higher and more variable as well. However, all relations among output variables and between input and output variables were very similar between the two diets. Because of this, and for the sake of clarity, only the results of the simulations using the "high-quality" protein diet are presented in the following text (Tables 3 and 4). However, because of the lower variation in P_{dep}, the results of the simulations using the "low-quality" protein diet are more graphically illustrative (Figures 1 and 2).

Body composition traits.

Table 3 gives the simulated population means and standard deviations of total body protein deposition from 23 to 100 kg body weight (P_{dep}, in kg), total muscle and connective tissue protein deposition ($P_{dep,mus}$ and $P_{dep,con}$, respectively, in kg; these are included in P_{dep}) and total body lipid deposition (L_{dep}, in kg) for each of the 49 simulations on the "high-quality" protein diet. Initial (23 kg body weight) body protein and lipid mass was 3.84 and 1.86 kg, respectively
The relationships between these deposition traits are visualized in Figure 1 for the "low-quality" protein diet. The three extremes depicted here (the lowest $\mu_{Pdep,max}$ in combination

Table 3 Means and standard deviations of deposition traits[†] (kg) in the $\mu_{Pdep,max}$ by feeding level subclasses on the high-quality protein diet

feeding level	trait	$\mu_{Pdep,max}$ (g.d^{-1}) 100 mean	100 sdev	125 mean	125 sdev	150 mean	150 sdev	175 mean	175 sdev	200 mean	200 sdev	225 mean	225 sdev	250 mean	250 sdev
0.6	P_{dep}	13.76	0.309	14.02	0.099	14.03	0.084	14.03	0.084	14.03	0.084	14.03	0.084	14.03	0.084
	$P_{dep,mus}$	8.84	0.387	9.01	0.354	9.02	0.357	9.02	0.358	9.02	0.357	9.02	0.357	9.02	0.357
	$P_{dep,con}$	3.38	0.268	3.43	0.267	3.43	0.266	3.43	0.266	3.43	0.266	3.43	0.266	3.43	0.266
	L_{dep}	15.36	1.219	14.34	0.373	14.29	0.314	14.29	0.315	14.29	0.314	14.29	0.314	14.29	0.314
0.7	P_{dep}	12.57	0.478	13.51	0.384	13.96	0.192	14.04	0.096	14.05	0.092	14.05	0.091	14.05	0.091
	$P_{dep,mus}$	8.03	0.441	8.67	0.418	8.97	0.363	9.03	0.357	9.04	0.361	9.04	0.360	9.04	0.360
	$P_{dep,con}$	3.12	0.259	3.32	0.267	3.42	0.269	3.44	0.267	3.44	0.267	3.44	0.267	3.44	0.267
	L_{dep}	20.15	1.905	16.42	1.491	14.66	0.736	14.33	0.344	14.31	0.314	14.31	0.314	14.31	0.314
0.8	P_{dep}	11.46	0.519	12.55	0.473	13.34	0.405	13.83	0.288	14.03	0.145	14.06	0.098	14.07	0.094
	$P_{dep,mus}$	7.28	0.445	8.02	0.442	8.55	0.426	8.89	0.389	9.02	0.359	9.05	0.358	9.05	0.362
	$P_{dep,con}$	2.87	0.245	3.11	0.258	3.28	0.266	3.39	0.269	3.43	0.268	3.44	0.267	3.44	0.267
	L_{dep}	24.65	2.086	20.30	1.880	17.21	1.592	15.26	1.088	14.51	0.543	14.35	0.337	14.33	0.319
0.9	P_{dep}	10.51	0.536	11.66	0.510	12.55	0.473	13.21	0.420	13.69	0.336	13.96	0.223	14.07	0.136
	$P_{dep,mus}$	6.64	0.441	7.42	0.446	8.01	0.441	8.47	0.432	8.79	0.406	8.98	0.372	9.05	0.357
	$P_{dep,con}$	2.65	0.232	2.91	0.248	3.11	0.259	3.26	0.266	3.36	0.269	3.42	0.270	3.44	0.270
	L_{dep}	28.54	2.157	23.91	2.044	20.41	1.860	17.77	1.631	15.90	1.295	14.84	0.808	14.47	0.449
1.0	P_{dep}	9.70	0.540	10.87	0.529	11.80	0.503	12.54	0.471	13.13	0.429	13.57	0.370	13.88	0.281
	$P_{dep,mus}$	6.11	0.431	6.89	0.444	7.51	0.447	8.01	0.442	8.41	0.434	8.71	0.416	8.92	0.386
	$P_{dep,con}$	2.47	0.220	2.74	0.237	2.94	0.249	3.11	0.258	3.24	0.265	3.34	0.269	3.40	0.271
	L_{dep}	31.90	2.174	27.13	2.121	23.41	2.014	20.48	1.851	18.20	1.657	16.45	1.405	15.28	1.042
1.1	P_{dep}	9.00	0.531	10.18	0.535	11.13	0.522	11.91	0.505	12.54	0.472	13.06	0.437	13.48	0.387
	$P_{dep,mus}$	5.64	0.416	6.42	0.437	7.06	0.446	7.58	0.450	8.01	0.445	8.37	0.439	8.65	0.422
	$P_{dep,con}$	2.31	0.209	2.58	0.227	2.79	0.240	2.97	0.251	3.11	0.258	3.22	0.264	3.31	0.269
	L_{dep}	34.78	2.162	29.99	2.169	26.16	2.097	23.06	1.972	20.55	1.843	18.53	1.674	16.92	1.477
1.2	P_{dep}	8.39	0.518	9.56	0.536	10.53	0.536	11.33	0.518	12.00	0.499	12.55	0.476	13.01	0.440
	$P_{dep,mus}$	5.25	0.400	6.01	0.428	6.66	0.442	7.19	0.447	7.64	0.449	8.02	0.447	8.33	0.443
	$P_{dep,con}$	2.17	0.199	2.44	0.218	2.66	0.233	2.84	0.243	2.99	0.252	3.11	0.258	3.21	0.263
	L_{dep}	37.31	2.123	32.53	2.174	28.65	2.133	25.46	2.073	22.82	1.965	20.61	1.832	18.79	1.691

[†] P_{dep}: total protein deposition between 23 and 100 kg body weight. $P_{dep,mus}$ and $P_{dep,con}$: total muscle and connective tissue protein deposition. L_{dep}: total lipid deposition.

with the highest feeding level, and the highest $\mu_{Pdep,max}$ in combination with the lowest and the highest feeding levels) encompass the results from all 49 simulations, both in terms of mean levels and in terms of variation.

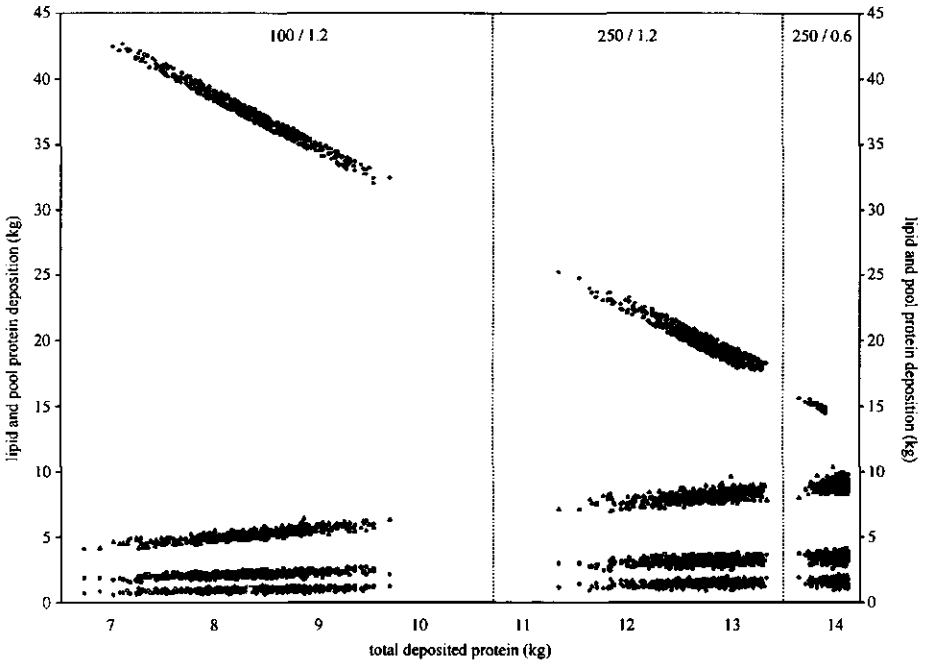

Figure 1 Simulated deposition of body lipid, muscle protein, connective tissue protein, and all other proteins (top to bottom), in relation to total body protein deposition (23 to 100 kg body weight), for three extreme combinations of $\mu_{Pdep,max}$ (100 and 250 $g.d^{-1}$) and feeding level (0.6 and 1.2 times the reference value) as indicated in the top of the graphs.

As expected, the mean levels of P_{dep}, $P_{dep,mus}$, and $P_{dep,con}$ increase (and those of L_{dep} decrease) with increasing $\mu_{Pdep,max}$, more markedly so at the higher feeding levels.
Although the $\sigma_{Pdep,max}$ value of each simulation was set proportional to its $\mu_{Pdep,max}$ level, simulated standard deviations of the traits in Table 3 are not proportional to their population means. For a given $\mu_{Pdep,max}$ value, increasing the feeding level results in a higher and more variable L_{dep}, and as a result (because of the fixed body weight trajectory) in a lower and more variable P_{dep}. Alternatively, at a given feeding level, increasing $\mu_{Pdep,max}$ results in a higher and less variable P_{dep} and in a lower and less variable L_{dep}.

Simulated mean $P_{dep,mus}$ levels are at about 64 % of the corresponding P_{dep} levels, with standard deviations of 0.33 to 0.53 kg. Assuming a protein content of muscle tissue of 20 % (as reported by most of the sources quoted in the Appendix) this would correspond to a standard deviation of muscle mass in a 100 kg pig of 1.65 to 2.65 kg. Standard deviations of estimated lean percentage in commercial slaughter pigs are usually higher than that (around 4 percentage points, *e.g.* tables 10 and 13 in Fisher, 1990) because of environmental variation that was not included in our simulations, and because the "lean" fraction that is measured in

dissection trials contains substantial amounts of intermuscular fat (plateauing to a proportion of 0.26 of "lean" mass around 115 kg body weight, based on appendices 1.1 and 1.2 in Tullis, 1981).

Mean $P_{dep,con}$ levels are at about 25 % of the corresponding P_{dep} levels, with standard deviations of 0.20 to 0.27 kg.

Maintenance-related traits.

For each of the 49 simulations on the "high-quality" protein diet, Table 4 gives simulated population means and standard deviations (all in kJ $ME.kg^{-0.75}.d^{-1}$) of the total calculated maintenance requirements (ME_{maint}) and its constituents: maintenance requirements independent from protein turnover processes ($ME_{maint,indep}$), ME requirements for turnover of already present body protein from 23 to 100 kg body weight ($ME_{turn,pres}$) and those for turnover of newly deposited protein ($ME_{turn,dep}$). The relationships of the turnover-related traits with P_{dep} have been visualised for the "low-quality" protein diet in Figure 2. Again, the three extremes depicted here (the same as in Figure 1) encompass the results from all 49 simulations, both in terms of mean levels and in terms of variation.

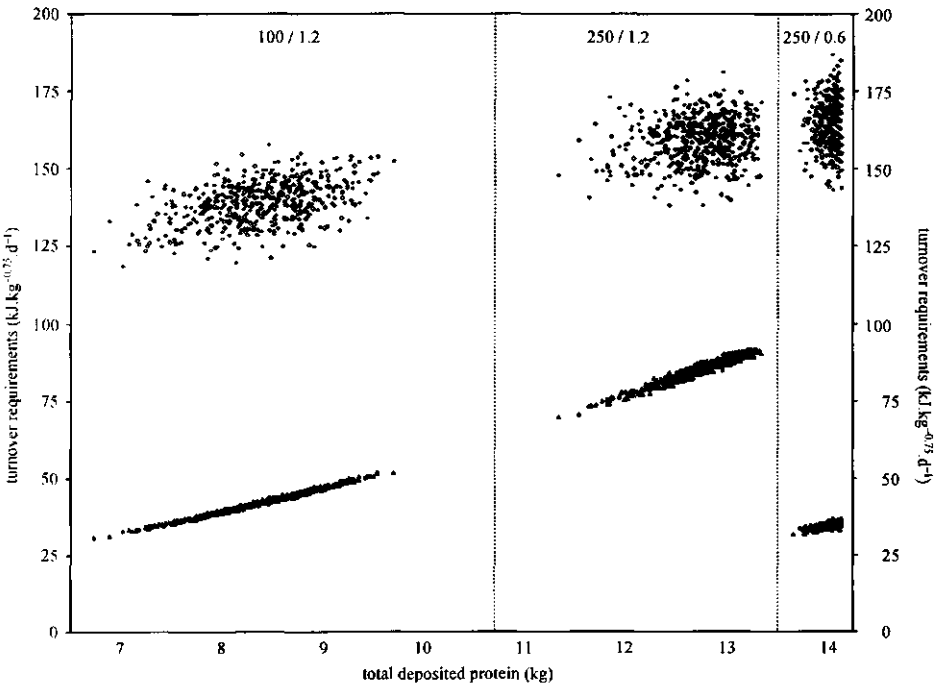

Figure 2 Simulated ME requirements for turnover of present (top) and newly deposited body protein (bottom), in relation to total body protein deposition (23 to 100 kg body weight), for three extreme combinations of $\mu_{Pdep,max}$ (100 and 250 g d^{-1}) and feeding level (0.6 and 1.2 times the reference value) as indicated in the top of the graphs.

Table 4 Means and standard deviations of maintenance-related traits† ($kJ.kg^{-0.75}.d^{-1}$) in the $\mu_{Pdep,max}$ by feeding level subclasses on the high-quality protein diet.

		$\mu_{Pdep,max}$ ($g.d^{-1}$)													
feeding		100		125		150		175		200		225		250	
level	trait	mean	sdev	mean	sdev	mean	sdev	mean	sdev	mean	sdev	mean	sdev	mean	sdev
0.6	ME_{maint}	489.2	7.60	491.6	6.93	491.9	6.86	492.0	6.87	492.1	6.87	492.1	6.87	492.1	6.87
	$ME_{maint,indep}$	291.8	0.16	291.9	0.06	291.9	0.05	291.9	0.05	291.9	0.05	291.9	0.05	291.9	0.05
	$ME_{turn,pres}$	163.9	7.51	164.5	7.40	164.5	7.39	164.5	7.39	164.5	7.39	164.5	7.39	164.5	7.39
	$ME_{turn,dep}$	33.4	1.54	34.7	0.81	34.7	0.81	34.7	0.81	34.7	0.81	34.7	0.81	34.7	0.81
0.7	ME_{maint}	488.8	8.58	498.0	8.08	502.4	7.27	503.4	6.94	503.6	6.90	503.7	6.90	503.7	6.90
	$ME_{maint,indep}$	290.8	0.09	291.1	0.18	291.3	0.13	291.3	0.07	291.3	0.06	291.3	0.06	291.3	0.06
	$ME_{turn,pres}$	160.3	7.58	163.3	7.49	164.3	7.41	164.5	7.38	164.5	7.37	164.5	7.37	164.5	7.37
	$ME_{turn,dep}$	37.7	2.73	43.4	2.46	46.4	1.39	47.0	0.93	47.0	0.93	47.0	0.93	47.0	0.93
0.8	ME_{maint}	485.5	9.00	497.7	8.91	506.6	8.50	512.2	7.79	514.5	7.17	515.1	6.97	515.2	6.93
	$ME_{maint,indep}$	290.2	0.09	290.2	0.09	290.4	0.16	290.6	0.17	290.7	0.11	290.7	0.08	290.7	0.07
	$ME_{turn,pres}$	155.6	7.54	160.3	7.53	162.9	7.48	164.1	7.42	164.4	7.39	164.5	7.37	164.5	7.36
	$ME_{turn,dep}$	39.7	3.26	47.2	3.41	53.1	3.22	57.2	2.38	58.8	1.37	59.2	1.08	59.2	1.07
0.9	ME_{maint}	481.6	9.22	495.5	9.39	506.6	9.27	515.2	8.92	521.4	8.30	524.9	7.57	526.2	7.15
	$ME_{maint,indep}$	289.9	0.16	289.7	0.10	289.6	0.09	289.7	0.13	289.9	0.17	290.0	0.15	290.1	0.11
	$ME_{turn,pres}$	150.9	7.44	156.6	7.51	160.4	7.51	162.6	7.47	163.8	7.43	164.3	7.40	164.5	7.37
	$ME_{turn,dep}$	40.9	3.60	49.3	3.97	56.6	4.08	62.7	3.91	67.3	3.31	70.1	2.21	71.0	1.43
1.0	ME_{maint}	477.6	9.31	492.7	9.69	505.2	9.81	515.5	9.67	523.8	9.35	530.2	8.81	534.6	8.10
	$ME_{maint,indep}$	289.7	0.22	289.3	0.17	289.1	0.11	289.0	0.09	289.1	0.12	289.2	0.15	289.3	0.17
	$ME_{turn,pres}$	146.5	7.31	152.8	7.45	157.3	7.49	160.4	7.49	162.4	7.46	163.6	7.43	164.2	7.40
	$ME_{turn,dep}$	41.5	3.82	50.6	4.35	58.8	4.66	66.0	4.75	72.2	4.59	77.2	4.13	80.7	3.19
1.1	ME_{maint}	473.8	9.30	489.6	9.88	503.1	10.17	514.6	10.23	524.3	10.10	532.4	9.79	539.0	9.31
	$ME_{maint,indep}$	289.6	0.24	289.1	0.22	288.7	0.17	288.6	0.14	288.5	0.11	288.5	0.11	288.5	0.14
	$ME_{turn,pres}$	142.4	7.15	149.1	7.36	154.1	7.45	157.8	7.48	160.4	7.47	162.3	7.45	163.4	7.43
	$ME_{turn,dep}$	41.8	3.96	51.4	4.63	60.3	5.08	68.3	5.34	75.4	5.41	81.6	5.26	86.8	4.88
1.2	ME_{maint}	470.3	9.21	486.4	9.98	500.6	10.40	513.0	10.66	523.8	10.69	533.1	10.54	541.1	10.26
	$ME_{maint,indep}$	289.4	0.25	288.9	0.26	288.5	0.23	288.2	0.19	288.0	0.15	287.9	0.12	287.9	0.12
	$ME_{turn,pres}$	138.8	6.97	145.7	7.26	151.0	7.39	155.1	7.45	158.2	7.47	160.5	7.47	162.1	7.44
	$ME_{turn,dep}$	42.0	4.01	51.9	4.83	61.2	5.39	69.8	5.79	77.6	6.02	84.7	6.06	90.9	5.92

† ME_{maint}, $ME_{maint,indep}$, $ME_{turn,pres}$, $ME_{turn,dep}$: ME requirements for maintenance (total and independent from protein turnover), and for turnover of already present and newly deposited body protein, respectively.

The turnover rate of already present body protein was treated as pool-specific in this study (TR_{pres} varies from 0.016 to 1.492 d^{-1} over the various pools, see the Introduction). Hence the plots of $ME_{turn,pres}$ (the upper ones in Figure 2) show a substantial variation at each P_{dep} level: quadratic regression within simulation sets of $ME_{turn,pres}$ on P_{dep} produced residual standard deviations (RSD) between 1.9 and 6.6 $kJ.kg^{-0.75}.d^{-1}$; when $P_{dep,mus}$ and $P_{dep,con}$ were added to the regression model, RSD was reduced to below 0.39 $kJ.kg^{-0.75}.d^{-1}$.

By contrast, the fractional rate of synthesis of newly deposited protein was assumed to be constant for all pools (FRS_{dep} = 2.00 d^{-1}). Hence the plots of $ME_{turn,dep}$ against P_{dep} (the lower ones in Figure 2) are very close to straight lines. A quadratic regression model of $ME_{turn,dep}$ on P_{dep} produced RSD values between 0.10 and 0.25 $kJ.kg^{-0.75}.d^{-1}$; addition of $P_{dep,mus}$ and $P_{dep,con}$ to the regression model further reduced these to 0.01 to 0.18 $kJ.kg^{-0.75}.d^{-1}$.

The information on among-animal variation in Table 4 can be summarized as follows.

Naturally, $ME_{maint,indep}$ varies only as a result of slight variations in metabolic body weight during the growth period. Hence its standard deviations within the $\mu_{Pdep,max}$ by feeding level subclasses are very low (ranging from 0.04 to 0.25 $kJ.kg^{-0.75}.d^{-1}$), just as its means differ hardly among subclasses.

$ME_{turn,dep}$ is proportional to daily body protein deposition, and hence its variation follows the variation of P_{dep} in Table 4. At the low feeding levels its standard deviation is therefore very limited (especially when $\mu_{Pdep,max}$ is high, of course), whereas at the higher feeding levels its coefficient of variation ranges from 5 to 10 %. It is especially the different feeding levels that cause the standard deviation of $ME_{turn,dep}$ to range widely from 0.81 to 5.13 $kJ.kg^{-0.75}.d^{-1}$.

When expressed on a daily basis, $ME_{turn,pres}$ is proportional to body protein mass, and the standard deviation of P_{dep} was seen in Table 4 to vary widely over $\mu_{Pdep,max}$ subclasses and especially over feeding levels. Nevertheless, the standard deviations of $ME_{turn,pres}$ (Table 4) are quite uniform over subclasses: they range from 6.96 to 7.58 $kJ.kg^{-0.75}.d^{-1}$. This discrepancy is related to the fact that $ME_{turn,pres}$ is expressed on a daily basis whereas P_{dep} is a cumulative figure, and to the large influence of the pool protein distribution on $ME_{turn,pres}$ (see above): the standard deviations of $P_{dep,mus}$ and $P_{dep,con}$ vary much less over $\mu_{Pdep,max}$ subclasses and feeding levels than P_{dep}'s do.

As an overall consequence of the above, ME_{maint} shows standard deviations from 6.86 to 10.69 $kJ.kg^{-0.75}.d^{-1}$. Higher ME_{maint} standard deviations generally coincide with the higher feeding levels and lower $\mu_{Pdep,max}$ values.

Discussion

In Chapter 1 it was attempted to integrate present quantitative knowledge of protein turnover parameters into a dynamic growth simulation model, and to derive likely values for these parameters. In the present study, this extended simulation model was made stochastic to generate replicates (animals) with variable body composition and, hence, variable energy requirements for protein turnover.

Adequacy of the model

This study was directed specifically to the among-animal variation (in body composition and turnover requirements) in the simulation output. Before this output variation is interpreted, it should be recognized that it has been generated entirely as a result of the simulated variation in the input variables $P_{dep,max}$ and $R_{L/P,min}$. Real-life data on the variation of the input variables are not available. However, some of the output variation can be related to experimental data, which can give some idea about the realism of the whole system.

In the data of Jørgensen *et al.*(1985) and Susenbeth and Keitel (1988) (see the Appendix), pigs with body weights between 80 and 120 kg showed standard deviations of total body protein mass (after adjustment for body weight) of 0.62 kg and 0.43 kg, respectively. The corresponding figures for total body lipid mass are 2.59 kg and 1.67 kg, respectively.
In those of our replicates with more or less reasonable combinations of $\mu_{Pdep,max}$ and feeding level (i.e. on and some cells below the main diagonal in Table 3), the standard deviations of P_{dep} range from 0.31 to 0.53 kg, and those of L_{dep} from 1.23 to 2.15 kg. This variation in simulated final body protein and lipid mass is entirely dependent on the simulated variation in $P_{dep,max}$ and $R_{L/P,min}$, whereas the real-life conditions in which the above mentioned experimental data were produced would present a number of other sources of variation; hence such data would be expected to be somewhat (but not very much) more variable than our simulation output, as indeed they are.
In view of these results it seems that the presupposed coefficients of variation of $P_{dep,max}$ and $R_{L/P,min}$ (10 and 5 %, respectively) were of a realistic order of magnitude. But that does not guarantee that $P_{dep,max}$ and $R_{L/P,min}$, as they have been used in our approach, are sensible model parameters *per se*.

The model of Moughan and Smith (1984) assumes $P_{dep,max}$ to be constant over the 23 to 100 kg body weight range, whereas it has been shown (see Whittemore *et al.*, 1988, for experimental results and further references) to be curvilinear against body weight with a maximum between 70 and 100 kg.

$R_{L/P,min}$ was originally introduced as a model parameter by Whittemore and Fawcett (1976) to force a more or less artificial balance upon their metabolic equations which were based on a constant $P_{dep,max}$, especially at low energy intake. This concept has been falsified in pigs between 12 and 36 kg body weight fed at a very low energy intake level by Kyriazakis and Emmans (1992) who report that "some animals [...] lost small amounts of lipid but continued to deposit protein at appreciable rates. Thus there was no minimum lipid:protein ratio in the gain; rather it can be suggested that [...] protein can still be gained at the expense of energy drawn from the lipid reserves". De Greef and Verstegen (1995) deal with this matter in more detail.
De Greef *et al.* (1994) measured protein and lipid deposition in pigs between 25 and 105 kg body weight. These pigs were fed at a level low enough to make them grow "below their maximal protein deposition capacity", which should ensure $R_{L/P,min}$ to be operational. It was found that "within this weight range, each kilogram increment of live weight increases the ratio between protein and lipid deposition rate by 0.0031 [to] 0.0066 units".
All this suggests that the unjustified use of the $R_{L/P,min}$ model parameter should have its most

pronounced effects at low body weights.

Whittemore (1995) summarizes the above matter by suggesting to abandon the approach of constant $P_{dep,max}$ and $R_{L/P,min}$ that is under debate here, and to replace it with a dynamic $P_{dep,max}$ without any $R_{L/P,min}$ restrictions. $P_{dep,max}$ could be described by a Gompertz function of total body protein mass following Whittemore *et al.* (1988). Whittemore (1995; figure 1) shows that both approaches produce very similar results in terms of the predicted daily rate of protein retention as a function of total body protein mass. Hence the debate seems to be largely an academic one, with practical consequences (in terms of our simulation output being unrealistic) mainly for simulated pigs at low body weights and limited energy intake.

Apart from the above considerations, there is evidence that $P_{dep,max}$, as a trait that can be measured on the population level, is environment-dependent; Moughan (1995) refers to the level that can be observed in growing pigs on-the-farm as "operational" $P_{dep,max}$, which may be lower than "true $P_{dep,max}$ due to the effects of sub-clinical disease, management etc.", and mentions that the measured level "may be influenced by an animal's growth history". The latter point refers to studies such as by De Greef *et al.* (1992), where considerably higher P_{dep} levels were measured in pigs that were fed ad libitum after protein restriction than in pigs of the same genotype that had been adequately fed all the time.

We choose to place the discussion on $P_{dep,max}$ and $R_{L/P,min}$ as realistic model parameters outside the scope of this paper. Our goal was to create systematic variation in body composition of our simulated pigs because we are interested in the covariation in maintenance requirement; introducing stochasticity on parameters like $P_{dep,max}$ and $R_{L/P,min}$ turns out to create this variation very effectively. In other words, the model is considered good enough for the limited purposes of the present study.

Variation in maintenance requirement

Judging the ME_{maint} standard deviations in Table 4 against their corresponding mean values of 470 to 541 $kJ.kg^{-0.75}.d^{-1}$ suggests that, averaged over all feeding levels and $\mu_{Pdep,max}$ subclasses, total maintenance energy requirements show a protein turnover-related between-animal coefficient of variation of 1.4 to 2.0 %.

Although this does not sound very impressive, it may lead to considerable genetic changes as a result of selection. This variation is to be interpreted as animal-intrinsic (genetic) variation within a population (populations being equivalent with $\mu_{Pdep,max}$ classes). Directional genetic selection for increased levels of $\mu_{Pdep,max}$ (by whatever means) would cause such a population to shift gradually through the continuum from 100 to 250 $g.d^{-1}$ that has been considered here, all the time retaining its 1.4 to 2.0 % turnover-related variation in ME_{maint}. As a consequence of this gradual 150 % increase in $\mu_{Pdep,max}$ (and the literature cited in Chapter 1 shows that this is feasible), the ME requirements for turnover of present and newly deposited protein would increase from 139 ± 7.0 (mean ± standard deviation) to 162 ± 7.4 and from 42 ± 4.0 to 91 ± 5.9 $kJ.kg^{-0.75}.d^{-1}$, respectively, provided the feeding level is high enough to allow the underlying genotype to be expressed (bottom rows in Table 4). As a result, total protein turnover-related maintenance requirements would increase by 15 % from 470 ± 9.2 to 541 ± 10.3 $kJ.kg^{-0.75}.d^{-1}$.

Of course, the bottom rows in Table 4 represent a feeding level "high enough to allow the un-

derlying genotype to be expressed" but the lower $\mu_{Pdep,max}$ classes would not be fed at such a high level in real life. In an earlier paragraph we restricted the "reasonable combinations of $\mu_{Pdep,max}$ and feeding level" to "on and some cells below the main diagonal" of the table. Under this constraint $ME_{turn,pres}$ would increase from, say, 156 ± 7.5 to 162 ± 7.4 $kJ.kg^{-0.75}.d^{-1}$, and $ME_{turn,dep}$ would increase from 40 ± 3.3 to 91 ± 5.9 $kJ.kg^{-0.75}.d^{-1}$. The increase in ME_{maint} would then be 11 %, from 486 ± 9.0 to 541 ± 10.3 $kJ.kg^{-0.75}.d^{-1}$.
Expressing ME_{maint} as a linear function of $\mu_{Pdep,max}$ resulted in a residual standard deviation of 7.39 $kJ.kg^{-0.75}.d^{-1}$.

Verification

When a poorly understood system such as protein turnover and its energetic aspects is under study, and one is moreover interested in the variation of the system rather than in its mean levels, the real-life experiment that is required to provide meaningful statistics can be expected to be elaborate and costly. Hence its design should be supported by as much information as can be generated from the literature. The results from this simulation study should be interpreted as just that: they show what may be expected of variation in maintenance requirement and its protein turnover-dependent relation to body composition, based on the published body of knowledge of the dynamics of protein metabolism in growing pigs. Although our rearrangement of literature results does reveal insights that hitherto were "hidden" in the data, it does not make any original addition to this body of knowledge. Hidden information is revealed, but no new data have been produced. In that sense, simulation studies such as the present one can be regarded as a highly structured type of literature survey.
The purpose of (simple and cheap) simulation studies must be to provide figures that are needed for the proper statistical design of the (elaborate and costly) real-life experiment that is supposed to provide a true scientific advance. To design the trial that could verify or falsify our simulation results we need *a priori* information on the (co-)variances of the characteristics to be measured, and the simulation provides estimates of these. We will consider briefly, and not exhaustively, how they can be used. A more thorough treatment of the issue will have to follow at a later stage.

The above made inferences about the relation of body composition traits with total maintenance requirement (e.g. the protein turnover-related variation in ME_{maint} and its positive relation with $P_{dep,max}$) can hardly be verified by directly measuring the associated energy costs (e.g. in an energy / nitrogen balance and respiration trial). For such a trial to be conclusive, the measurable differences in ME_{maint} should be entirely due to differences in protein turnover; it will be very difficult to arrange an experimental set-up that ensures this. The influence on ME_{maint} of processes such as thermal regulation, immune response and coping with other stress factors can be minimized by proper environmental conditions (thermoneutral, pathogen-free, welfare-friendly, etc.) and physical activity can be either standardized or measured in order to adjust for its energy costs. But a process such as ion transport over cell membranes which has been shown (again by simulation) to cause at least as large a proportion of total body energy expenditure as protein turnover (Gill *et al.*, 1989) cannot be controlled experimentally, and it will be very difficult to measure the related energy costs *in vivo* in order to adjust them out.

Therefore our experiment should concentrate on $ME_{turn,pres}$ and $ME_{turn,dep}$. These will probably have to be determined together as ME_{turn}, by measuring the extent of whole body protein catabolism as discussed by Simon (1989) and deriving its energy costs using bio-energetic theory (Klein and Hoffmann, 1989; simplified in equations (4) and (5) of Chapter 1). Basically, the experiment should provide the data that are required to verify the relations predicted in Figures 1 and, especially, 2. Such data are to be collected on a group of pigs growing from 23 to 100 kg body weight to produce a wide range of realized P_{dep} values, which can be ensured by feeding the animals at a high level and varying their genetic predisposition for protein deposition (equivalent to $P_{dep,max}$). The latter could be approximated in terms of the estimated breeding values for body composition and growth-related traits such as they are routinely produced in practical pig breeding programmes.

ME_{turn} is expressed in $kJ.kg^{-0.75}.d^{-1}$, so the trait will have to be measured on each individual repeatedly to properly cover the course of metabolic body weight from 10.5 to 31.6 $kg^{0.75}$. A reasonable mid-point would be at 20 $kg^{0.75}$, i.e. at a 54 kg body weight. P_{dep} as it has been defined here assumes the measurement of P_{pres} at 23 kg and at 100 kg body weight. This means either some sort of serial slaughter trial, or the use of techniques like D_2O dilution or computerized tomography (see Allen, 1990, for a review); the latter method could also deliver data on $P_{dep,mus}$.

From these data we need to be able to produce good estimates of the means and among-animal variances of (i) ME_{turn} and (ii) total P_{dep} and $P_{dep,i}$ for muscle and connective tissue protein, and of the covariances between (i) and (ii).

The estimation of the covariances is statistically most demanding; hence the minimum required numbers of experimental units should be derived from the distribution parameters of these. It is operationally more appealing to think in terms of the regression coefficients of (i) on (ii) above.

The measurement of $P_{dep,mus}$ will be more complicated than P_{dep}, and the regression is statistically more demanding. In the simulated data in the seven $\mu_{Pdep,max}$ "populations" at the 1.2 feeding level, the linear regression of ME_{turn} on $P_{dep,mus}$ is $Y = 72.40 + 21.24 \times X$ with a correlation $r_{xy} = 0.90$; the standard deviations of X and Y are 1.12 kg and 26.27 $kJ.kg^{-0.75}.d^{-1}$, respectively. The estimate of the regression coefficient b_{yx} (21.24 $kJ.kg^{-0.75}.d^{-1}$ per kg) has a standard error of 0.17 $kJ.kg^{-0.75}.d^{-1}$, calculated as $se_{b_{yx}} = \sqrt{(X'X)^{-1}_{22} \times MSE}$ (SAS, 1990b; the first term is the second diagonal element of the inverse of the design matrix, the second term is the regression model's mean square for error) which in this case is equivalent to $se_{b_{yx}} = \sqrt{\frac{1 - r_{xy}^2}{(n-2) \times r_{xy}^2}} \times b_{yx}$ with n = 3500 replicates. This equation can be rearranged to find n_{req}, the number of observations required to estimate b_{yx} with a certain standard error, as follows:

$$n_{req} = 2 + \frac{b_{yx}^2 \times (1 - r_{xy}^2)}{se_{b_{yx}}^2 \times r_{xy}^2} \qquad (2)$$

Substituting $b_{yx} = 21.24$ and $r_{xy} = 0.90$, as above, into (2) gives $n_{req} = 2 + (100.2 / se_{b_{yx}}^2)$.

Hence for a standard error of, say, 4 $kJ.kg^{-0.75}.d^{-1}$ (about 20 % of the expected estimate) we would need nine observations. This assumes that these pigs can be arranged to have a standard deviation in $P_{dep,mus}$ of about 1.1 kg; the within-"population" standard deviations in Table 3 range from 0.40 to 0.45, so the experimental group will have to be obtained by deliberate sampling.

So far we have assumed that ME_{turn} and especially $P_{dep,mus}$ can be measured without error, and this assumption is clearly violated in practice. Erroneous data on the Y variable (ME_{turn}) just increase the error variance, but erroneous data on the X variable ($P_{dep,mus}$) cause it to become correlated with the error terms of the regression model, and the resulting regression coefficient estimates are biased (Neter *et al.*, 1985); this is a statistical problem in itself that goes far beyond the scope of this paper.

In terms of the above, the correlation coefficient of 0.90 reflects the relation between error-free simulated data; it would be reduced if the X and/or Y variables contained errors, as they would under experimental conditions.

Susenbeth (1984) derived that, with an end weight four times as large as the start weight (corresponding to our simulated growth trajectory from 23 to 100 kg), P_{dep} can be measured with a 5 and 10 % coefficient of variation due to measurement errors in serial slaughter trials and with D_2O dilution techniques, respectively. Based on the figures summarized by Allen (1990; table 17), computerised tomography would fall somewhere in between.

Table 5 Regression statistics of ME_{turn} on $P_{dep,mus}$ and required numbers of observations, for various error levels in the variables

error (% of mean)		standard deviation				
$P_{dep,mus}$	ME_{turn}	$P_{dep,mus}$	ME_{turn}	b_{yx}	r_{xy}	n_{req} †
0	0	1.12	26.27	21.24	0.90	9
	5		28.49	21.27	0.83	15
	10		34.33	21.30	0.69	33
	20		51.47	21.35	0.46	107
5	0	1.17	26.27	19.36	0.86	11
	5		28.49	19.41	0.80	16
	10		34.33	19.46	0.67	33
	20		51.47	19.56	0.45	99
10	0	1.32	26.27	15.33	0.77	13
	5		28.49	15.39	0.71	17
	10		34.33	15.45	0.60	30
	20		51.47	15.58	0.40	83
20	0	1.79	26.27	8.41	0.57	12
	5		28.49	8.47	0.53	14
	10		34.33	8.52	0.45	21
	20		51.47	8.63	0.30	50

† Required number of observations for a standard error of b_{yx} at 4 kJ ME $kg^{-0.75}$ d^{-1}, according to equation (2)

To get some idea of the problem's order of magnitude, error terms were added to each of the above 3500 ME_{turn} and $P_{dep,mus}$ data, drawn from two independent sets of random Normal $N(0,\sigma^2)$ deviates (SAS, 1990a). The standard deviations were set to σ = 0, 0.351, 0.702 and 1.404 kg for $P_{dep,mus}$ (X) and σ = 0, 22.14, 44.28 and 88.56 $kJ.kg^{-0.75}.d^{-1}$ for ME_{turn} (Y) to reflect measurement errors of zero and 5, 10 and 20 % of the mean X and Y values. As indicated above, real-life X errors may be expected to range from 5 to 10 %; Y errors will be

higher, possibly between 10 and 20 %. The statistics from the resulting data combinations are in Table 5.

The direct consequence of the imposed X and Y error terms is an increase in the standard deviation of the corresponding variable ($P_{dep,mus}$, ME_{turn}). As a result of this, the scatter plot grows wider which is reflected in reduced r_{xy} values and through these in increased n_{req} values. Y error terms do not affect the b_{yx} values, but in line with Neter *et al.* (1985; see above) X error terms cause a negative bias on b_{yx} and as a consequence on n_{req}. Hence the values for n_{req} in Table 5 are only meaningful for zero errors in $P_{dep,mus}$.

In terms of the number of observations required for our experiment the conclusion from Table 5 is quite clear, although not very optimistic: at least 33 and probably about 100 observations will be needed, dependent on the accuracy of ME_{turn} measurement. But more serious, the negative bias on the observed regression coefficient that results from measurement errors in $P_{dep,mus}$ leads to a paradox in the interpretation of the experimental results, as follows.

The aim of the experiment is to obtain an estimate for b_{yx} that is (or is not) significantly different from our simulated value of 21.24 $kJ.kg^{-0.75}.d^{-1}$ per kg. With a 10 % error in $P_{dep,mus}$, the estimated regression coefficient of (on average) 15.44 would be 5.80 units lower than the value to be verified, which is entirely due to bias on the estimate; this difference is 1.45 times as large as the requested $se_{b_{yx}}$ value, and this would approach statistical significance in a one-tailed t-test ($P = 0.079$ for $df = 30$, $P = 0.075$ for $df = 100$). It follows that a coefficient of variation due to measurement errors in $P_{dep,mus}$ of more than 10 % would make it effectively impossible to experimentally verify our simulation results: as a consequence of the X errors, the observed regression coefficient becomes so much deflated that it will appear to be significantly lower than the value to be verified.

In summary, the experiment required to verify or falsify the present simulation results should comprise a group of pigs sampled on the basis of their genetic predisposition for protein deposition. This sampling should aim at a standard deviation in the experimental group of about 1.1 kg $P_{dep,mus}$ over the growth trajectory from 23 to 100 kg body weight, when fed ad libitum and in the absence of any appetite-reducing factors. (Muscle) protein mass should be measured at the start and end weights to determine $P_{dep,mus}$ with a coefficient of variation due to measurement error of less than 10 %; this will likely involve a serial slaughter procedure (which doubles the required number of pigs) or, alternatively, computerized tomography (CT). The energy costs for whole body protein turnover should be measured at the start and end weights, and around 54 kg body weight, to determine ME_{turn}. The required number of observations (i.e. pigs in a CT trial, pairs of pigs in a serial slaughter trial) depends on the accuracy of ME_{turn} determination; a 10 % coefficient of variation due to measurement error requires 33 observations, a 20 % coefficient requires 100 observations. These numbers should be sufficient to obtain meaningful estimates of all other parameters of interest.

Acknowledgements

Valuable contributions were made by Martin Verstegen, Paul Moughan, John Woolliams, Ella Luiting, Gerry Emmans, and especially by an anonymous referee. Henry Jørgensen, Andreas Susenbeth, and Helmuth Pfeiffer kindly made available the data used in the Appendix.

Appendix. Partitioning of body protein of growing pigs over protein pools.

In Chapter 1 we re-analyzed data reported on by Tullis (1981), by Metz *et al.* (1980), by Jørgensen *et al.* (1985), by Susenbeth and Keitel (1988), and by Pfeiffer *et al.* (1990). The fractions of body protein in four pools (muscle, connective tissue, entrails, blood) were regressed on the natural logarithm of body protein mass (table 3 of Chapter 1); residual standard deviations around the estimated regression lines are in Table 6.

Table 6 Residual standard deviations (%) around the regression of percentage of body protein in various protein pools on the natural logarithm of total body protein mass

	source[†]				
Protein pool	Metz	Tullis	Jørgensen	Susenbeth	Pfeiffer
Muscle	1.85	2.28	2.36	1.83	1.93
Connective tissue	1.59	2.39	1.79	1.37	1.03
Entrails[‡]	0.67	1.67	0.86		1.30
Blood		0.70	0.63		0.66
Liver					0.28

[†] Metz *et al.* (1980); Tullis (1981); Jørgensen *et al.* (1985); Susenbeth and Keitel (1988); Pfeiffer *et al.* (1990)

[‡] Including liver in all entries

In table 4 of Chapter 1, we combined the data from these sources together and performed an overall regression analysis. The residual standard deviations around the resulting regression lines are in Table 7.

These residual terms show a distinct correlation pattern: muscle protein grows proportionally at the expense of the other three pools, which leads to negative correlations, especially with connective tissue protein. The calculated correlation coefficients among the main pools are in Table 8.

Table 7 Overall residual standard deviations (%) around the regression of percentage of body protein in various protein pools on the natural logarithm of total body protein mass

Protein pool	residual standard deviation
Muscle	2.44
Connective tissue	1.90
Entrails	1.06
Blood	0.66
Liver	0.28

The sceptical reader might argue that the among-animal variation in protein distribution over pools (Table 7) is an artefact due to experimental measurement errors. Solid evidence against such a statement would involve, for example, the establishment of significantly positive repeatabilities or heritabilities, and the data required for such an exercise would be difficult to collect. Repeatabilities would require repeated measurements of the protein content of the various pools *within animals*, which in turn requires non-destructive measurement techniques. In principle use could be made of computerized tomography, but it would be extremely difficult to separate the protein pools, especially blood plasma, the gastro-intestinal tract and a part of connective tissue. Heritabilities would require the determination of protein

content of the various pools in animals in a group with a known and well-structured system of genetic relationships.

Table 8 Correlation coefficients among the residual terms from Table 7

Protein pool	connective tissue	entrails	blood
Muscle	–0.838	–0.492	–0.523
Connective tissue		+0.052	+0.290
Entrails			+0.095

Repeatabilities and heritabilities are demanding statistics in terms of information to be collected to obtain a statistically significant estimate: to estimate a heritability of 0.25 at a significance level of 5 % would require data on 350 to 600 animals (e.g. 25 groups of 14 half sibs each, or 150 groups of four).

On the other hand, there is no clear reason why this body composition characteristic should *not* vary among animals just as other body composition traits are well-documented to do; its 5 to 10 % coefficient of variation in a 100 kg pig is of the same order of magnitude as those of other body composition traits. Susenbeth (1984) writes (our translation from German): "The accuracy of determination of gain of body fractions by serial slaughter trials is primarily dependent on the variation of body composition within the experimental group, which probably results mainly from genetic differences between animals. The accuracy of the chemical analysis has hardly any influence, because its resulting variation in [observed] body composition is far lower than the variation caused by animal differences".

The genotypes covered by these references vary widely with respect to fattening and slaughter characteristics; correspondingly, the differences between the regression parameters are sometimes quite large. It may be critical to determine these parameters specifically for the pig population to be simulated. The values derived here should be seen as generalizations, serving solely for parameterization in this general study.

In practice, few models are entirely confirmed by observational data, and few are entirely refuted. Typically, some data do agree with predictions and some do not. Furthermore, [the terms] *verify* and *validate* encourage the modeler to claim a positive result; we have never seen a paper in which the authors wrote "the empirical data invalidate this model".

N. Oreskes *et al.* (1994) *Science* **263**:641-646. Condensed.

Chapter 3 is based on

P.W. Knap (1999) Simulation of growth in pigs: evaluation of a model to relate thermoregulation to body protein and lipid content and deposition. *Animal Science* **68**:655-679

Chapter 3

Evaluation of a thermoregulation model

A dynamic model for simulation of growth in pigs was extended by a module to assess maximum and minimum heat loss (HL_{hot}, HL_{cold}) for a given pig, to compare these figures to heat production (HP), and to take thermoregulatory action when $HP < HL_{cold}$ (cold conditions) or $HP > HL_{hot}$ (hot conditions).

HL_{cold} and HL_{hot} were largely determined according to algorithms obtained from the literature, but HL_{cold} was made dependent on body fat depth through tissue insulation. Data to establish the relation ($Y = 0.05 + 0.002 \times X$) between cold tissue insulation (Y in $°C.m^2.W^{-1}$) and backfat depth (X in mm) independent of body weight were obtained from the literature. The same data showed that HL_{hot} is not related to backfat depth in pigs.

Cold thermoregulatory action included an increase of *ad libitum* feed intake. Hot thermoregulatory action included reduction of physical activity, increase of body temperature, wetting of a proportion of the skin, and reduction of *ad libitum* feed intake.

A sensitivity analysis showed that the model's output in terms of *ad libitum* feed intake, HP, protein deposition (P_{dep}) and lipid deposition (L_{dep}) is strongly sensitive to the characterisation of the genotype being simulated.

The model was used to simulate trials from the literature. Although the model does not explicitly calculate lower and upper critical temperatures, these could be adequately predicted from its output. Comparison of model output with experimental data revealed an adequate prediction of *ad libitum* feed intake and of the partitioning of ad libitum ingested ME into HP, P_{dep} and L_{dep} in cold, thermoneutral and hot conditions. At restricted ME intake, and especially in cold conditions, the model tends to overestimate HP and underestimate L_{dep}, probably because it does not take account of long-term acclimatisation.

Introduction

As was noticed in Chapter 1, although maintenance energy requirements constitute a significant source of genetic variation in production efficiency in meat animals, their underlying physiological processes are poorly documented and their relation with production traits is poorly understood. That there *is* some physiological relation between maintenance requirements and production traits in growing animals is likely, simply because body protein is more metabolically active than lipid and because its deposition takes place at higher energetic costs. But the actual relation is not immediately obvious. In hot environmental conditions, intrinsically "lean" growing animals are forced to reduce their metabolism (and the associated heat production) to a larger extent than fatter animals (*e.g.* Ferguson *et al.*, 1997). In cold conditions, lean growing animals profit from their larger heat increment of protein synthesis, but fatter animals have a better thermal insulation (*e.g.* Currie, 1988).

The study of such phenomena may benefit considerably from the incorporation of the underlying processes in dynamic simulation models, such as that pioneered by Whittemore and Fawcett (1976) and further developed by Moughan and Smith (1984) and Black *et al.* (1986), among others. Those models describe total maintenance energy requirements as a simple function of body weight or body protein mass. It is our purpose to produce a more elaborate quantification, separating "maintenance" into its most important component processes. The idea is that when these processes can be modeled separately, the interrelationships between maintenance energy requirements and deposition-related traits can be described in more detail. For this purpose we quantify the energy requirements of body protein turnover and thermoregulation in Chapters 1 to 4 of this thesis. The energy requirements of protein turnover, and the associated relations between body composition and maintenance requirements, were dealt with in Chapters 1 and 2, respectively. Thermoregulation is dealt with here.

As its first aim, this chapter describes the extension of an existing dynamic simulation model with algorithms from the literature that quantify thermoregulatory energy metabolism, where appropriate as a function of body protein and lipid content or deposition. These model extensions are used in Chapter 4 to quantify the thermoregulation-dependent covariance between body composition and maintenance requirements. Hence this chapter provides the tools for the study carried out in the next one.

The second aim of this chapter is to compare simulation output of the resulting thermoregulation model with real-life data to obtain an impression of the model's adequacy. Since pig genotypes differ considerably in terms of body leanness, we expressly allow for variation among genotypes when doing so.

Thermoregulation

Mammalian thermoregulatory processes have previously been described by Bligh (1973), Mount (1979), McArthur (1981b, 1987), and Blaxter (1989). Attempts to quantify porcine thermoregulation were made by Bruce and Clark (1979), Black *et al.* (1986, 1998), and Turnpenny *et al.* (2000).

Within the framework of the model used in this study, when a pig has the nutrients available to sustain its maintenance energy requirements (ME_{maint}, in $MJ.d^{-1}$) and its animal-intrinsic drive for protein and lipid deposition (P_{dep} and L_{dep}, in $kg.d^{-1}$), it will require a daily ME intake (ME_{req}, in $MJ.d^{-1}$) of

$$ME_{req} = ME_{maint} + ME_{Pdep} \times P_{dep} + ME_{Ldep} \times L_{dep} \quad (1)$$

From this intake a thermoneutral heat production (HP, in $MJ.d^{-1}$) will follow equal to

$$HP = ME_{req} - NE_P \times P_{dep} - NE_L \times L_{dep} \quad (2)$$

or, equivalently,

$$HP = ME_{maint} + HI_{Pdep} \times P_{dep} + HI_{Ldep} \times L_{dep} \quad (3)$$

ME_{Pdep}, ME_{Ldep}, NE_P, NE_L, HI_{Pdep}, and HI_{Ldep} represent the gross ME requirement for protein and lipid deposition (*e.g.*, $ME_{Pdep} = ME_{Ldep} = 53.0$ $MJ.kg^{-1}$; Moughan and Verstegen, 1988), the net (combustion) energy content of protein and lipid ($NE_P = 23.8$; $NE_L = 39.6$ $MJ.kg^{-1}$), and the heat increment of protein and lipid deposition ($HI_{Pdep} = 53 - 23.8 = 29.2$; $HI_{Ldep} = 53 - 39.6 = 13.4$ $MJ.kg^{-1}$).

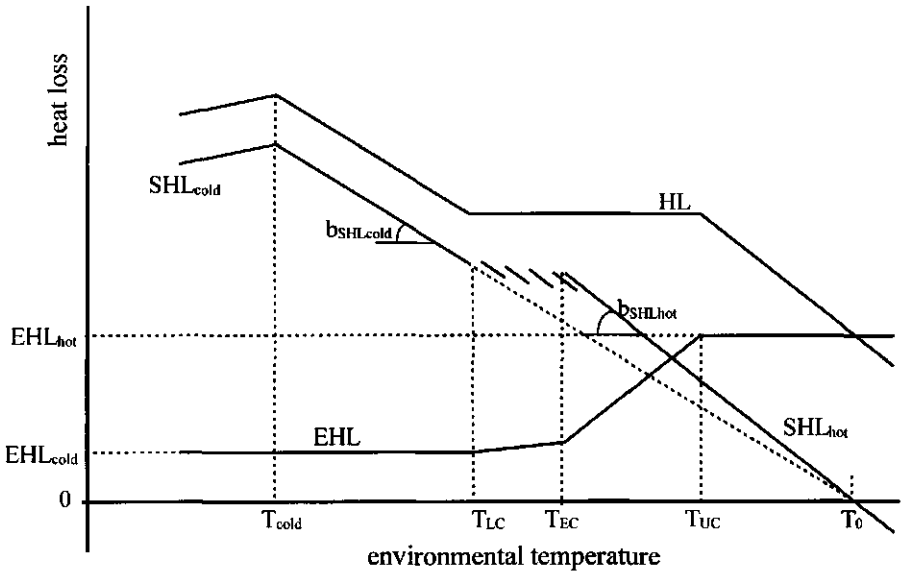

Figure 1 Schematic representation of the heat production and heat loss entities used in this study, as a function of environmental temperature. Modified after Mount (1974) and Blaxter (1989).

Except for short-term action of the body as a heat buffer, HP has to be released in the form of heat loss (HL), which occurs through radiation, conduction and convection when the environmental temperature T_{env} is lower than body temperature T_{body}. This sensible heat loss (SHL) declines to zero when T_{env} increases to T_{body}, see Figure 1. Heat loss occurs also as a consequence of evaporation of water from the pig's skin and respiratory tract (evaporative heat loss, EHL). In this thesis we use the term "heat loss" as a synonym of "environmental heat demand": the amount of heat that must be dissipated by the animal due to temperature and vapour-pressure gradients across its interface with the environment (Mount, 1979). HL then represents the interaction between animal and environment. In contrast, "heat production" is largely a property of the animal and its current nutritional status.

To conform with equation (3), HP must be quantified in terms of ME_{maint}, P_{dep} and L_{dep}. HL

must be quantified in terms of body protein (P) and lipid mass (L). HP and HL can then be compared, and appropriate action taken on the result. We model these processes in time steps of one day. Diurnal varation of T_{env} and/or T_{body}, and the action of the body's tissue mass as a heat buffer, are assumed to have no consequence for our simulation results. The same holds for long-term adaptation to cold or hot conditions.

Thermoneutral environment

The thermoneutral zone is bounded by the lower and upper critical temperatures (T_{LC}, T_{UC}). Between these T_{env} levels T_{body} is maintained by physical means without special metabolic action. In its lower part up to the evaporative critical temperature T_{EC}, thermoregulation is dealt with by vasomotor action: peripheral vasoconstriction is relaxed with increasing T_{env}. This reduces body thermal insulation capacity so that heat can be released easier and the curve of SHL against T_{env} becomes gradually steeper (from $b_{SHLcold}$ to b_{SHLhot} in Figure 1). Beyond T_{EC} body thermal insulation capacity is at its minimum, which allows the SHL curve to remain at its steepest decline (b_{SHLhot}) so that SHL_{hot} is operative. To compensate for the SHL decrease the pig has to increase its evaporation and EHL.

Hot environment

In a dry environment EHL has a practical maximum level (EHL_{hot}). When this has been reached (or even before that, see the end of this section) and T_{env} still increases, HP cannot be completely released by physical heat exchange and the pig has to reduce its metabolism to avoid a hyperthermic rise in T_{body}. In such conditions the environment is "hot". The components of equation (3) must then be changed to reduce HP far enough to be matched by the pig's maximum possible HL. For practical modeling purposes, consider a pig that attempts to maximise its heat dissipation capacity, which brings up SHL and EHL to this pig's theoretical maximum values SHL_{hot} and EHL_{hot} so that HL_{hot} (this pig's theoretical maximum total HL value) is operative. It follows that a pig has to deal with a "hot" environment if HP from equation (3) is larger than this pig's HL_{hot}. The required reduction of HP is then

$$RHP = HP - HL_{hot} \quad (4)$$

Considering equation (3), this HP reduction can be brought about in two ways: by reducing ME_{maint} (by minimising physical activity), and/or by reducing P_{dep} and L_{dep} (which is where the connection of hot thermoregulation with protein and lipid deposition lies). Both actions would reduce ME_{req}, FI, and HP.

Reduction of ME_{maint} by minimising physical activity might "save" about 20 % of thermoneutral HP according to Susenbeth and Mencke (1991). The energy costs of various types of behaviour in pigs that were put together by Curtis (1983), combined with the activity budget information of growing pigs reported by Curtis (1993), suggest that about 14 % of HP of growing pigs in a thermoneutral environment is due to activities such as sitting, standing, eating, moving, exploring, interacting and fighting. Hence pigs in hot conditions would be able to reduce their HP by 7 to 10 % by halving their physical activity.

Ferguson *et al.* (1994) present some reasoning on P_{dep} and L_{dep} reductions in relation to the first limiting nutrient in the feed.

Another option would be to *not* release some of the produced heat. Black *et al.* (1998) connect

the hot decline in ME_{req} to a rise in T_{body}, associating a 40 % reduction in feed intake with an increase in T_{body} by one degree. In the model described by Black *et al.* (1986), T_{body} "is assumed to be 39 °C, but as $[T_{env}]$ approaches $[T_{UC}]$, the pig is allowed to store sufficient heat [...] to permit its $[T_{body}]$ to rise to a maximum of 40.5 °C". Giles and Black (1991) show that this rise in T_{body} and the associated decline of feed intake already commence at T_{EC}. Hence thermoregulatory reduction of metabolism is initiated long before the pig's options for increasing physical heat exchange (through EHL) are exhausted.

Failing the above actions, the pig reaches its hyperthermic point T_{hot} (not in Figure 1) and loses homeothermy.

Cold environment

When $T_{env} < T_{LC}$ the environment is "cold": thermoneutral HP is not sufficient to meet the environmental heat demand, and extra heat must be produced for cold thermogenesis. The associated energy supply requires an extra, positive, term in equation (1).
For practical modeling purposes, consider a pig that attempts to minimise the increased environmental heat demand, which brings SHL and EHL down to this pig's theoretical minimum values SHL_{cold} and EHL_{cold} so that HL_{cold} (this pig's theoretical minimum total HL value) is operative. It follows that a pig has to deal with a "cold" environment if HP from equation (3) is less than this pig's HL_{cold}. The required additional term in equation (1), the "extra thermoregulatory heat", is then

$$ETH = HL_{cold} - HP \quad (5)$$

The connection to body composition is in the thermal insulation capacity of vasoconstricted subcutaneous fatty tissue. The higher insulation value of a deeper fat cover would lower T_{LC} and it would make the slope of the SHL_{cold} curve less steep, which in turn would reduce HL_{cold} and ETH.

Failing the above actions, the pig reaches its hypothermic point T_{cold} and loses homeothermy.

Model

When we studied the protein turnover-related consequences of variable body composition on maintenance requirements in Chapters 1 and 2, the framework of Moughan and Smith's (1984) nutrient partitioning model provided sufficient flexibility for the purpose of simulating pig populations with variable body protein and lipid content. Feed intake was dealt with by relating it to body weight according to consecutive feeding levels. This approach is sufficient for a deeply animal-intrinsic process such as body protein turnover, but the model should be able to predict feed intake as an output when studying an environment-sensitive process such as thermoregulation. To deal with this requirement, the version of Moughan and Smith's (1984) model that was extended in Chapter 1 was adapted to suit the potential growth rate and feed intake rules by Emmans (1988, 1994, 1997), Emmans and Kyriazakis (1995) and Kyriazakis *et al.* (1994).
Details of a simplified version of this adapted model, as far as these are essential for the understanding of the present text, are in Appendix I. It derives the animal's intrinsically "desired" protein and lipid deposition from a Gompertz function that scales current body protein mass to its mature value P_∞, and from an allometric relation between body lipid and protein mass. The latter leads to an animal-intrinsic ratio between mature body lipid and protein

mass, $R_{L\infty/P\infty}$. The desired deposition values are then combined with ME_{maint} (including protein turnover costs according to Chapter 1) to derive the required feed intake from equation (1), which may be restricted by a feeding scheme and/or by a volume constraint on voluntary feed intake. Ferguson *et al.* (1994) simulate such a constraint based on the feed's organic matter digestibility and the animal's body protein mass. Kyriazakis and Emmans (1995) describe a constraint based on the feed's water holding capacity.

So far, the simulated pig has been assumed to be thermoneutral. In order to retain thermal equilibrium outside the thermoneutral zone, the algorithm in Appendix I was extended by two routines. One of these deals with hot thermoregulation, imposing RHP from equation (4) as an additional constraint on voluntary feed intake following Ferguson *et al.* (1994). The other deals with cold thermoregulation, increasing ME_{maint} with ETH from equation (5) following Verstegen *et al.* (1995). Both routines are further detailed in the remainder of this section. We can then consider a pig that has a HP according to equation (3), assess the current climate, and evaluate equations (4) and (5) to take care of hot and cold conditions. Whether the pig *is* hot or cold can be determined at the same time: it is hot when RHP > 0 (HP > HL_{hot}), and cold when ETH > 0 (HP < HL_{cold}). Otherwise ($HL_{cold} \leq HP \leq HL_{hot}$) it is thermoneutral. Hence HL_{cold} and HL_{hot} must be determined for this pig, with its current equation (3) entities, in this climate. For the purposes of this study there is no need to determine the critical temperatures, nor to model the thermoneutral vasomotor processes.

The principles of the algorithm were taken from Black *et al.* (1986). An important exception is that T_{LC}, T_{EC}, and T_{UC} are not determined here *per se*, as these parameters do not play an explicit role in the calculations. The reasoning of Black *et al.* (1986) on thermoregulation goes largely back to Bruce and Clark (1979) and McArthur (1981a), and leads to the following:

$$HL_{cold} = EHL_{cold} + SHL_{cold} = EHL_{cold} + A_{eff,cold} \times b_{SHLcold} \times (T_{env} - T_0) \quad (6)$$

$$ETH = HL_{cold} - HP \quad (5)$$

$$HL_{hot} = EHL_{hot} + SHL_{hot} = EHL_{hot} + A_{eff,hot} \times b_{SHLhot} \times (T_{env} - T_0) \quad (7)$$

$$RHP = HP - HL_{hot} \quad (4)$$

where $A_{eff,cold}$ and $A_{eff,hot}$ denote the pig's effective body surface area in cold and hot conditions (subject to differences in posture and huddling), $b_{SHLcold}$ and b_{SHLhot} denote the slopes of SHL_{cold} and SHL_{hot} against T_{env}, and T_0 is the value of T_{env} at which $SHL_{cold} = SHL_{hot} = 0$ (Figure 1).

The seven parameters T_0, $b_{SHLcold}$, b_{SHLhot}, $A_{eff,cold}$, $A_{eff,hot}$, EHL_{cold}, and EHL_{hot} in equations (6) and (7) are described in Appendix II, where appropriate as a function of body composition.

After values for ME_{maint}, P_{dep}, L_{dep}, and thermoneutral HP have been predicted, fat depth is assessed (Appendix IV), tissue insulation values $I_{tissue,cold}$ and $I_{tissue,hot}$ are determined (Appendix III), and $b_{SHLcold}$ and b_{SHLhot} are calculated from equations (A2-2) and (A2-4). Following Black *et al.* (1986), the insulation of the air layer surrounding the animal (I_{air}) is initially calculated from equation (A2-3) with tentative skin temperature values of T_{skin} = 32 and 39 °C to deal with conditions below and above T_{LC}, respectively. After deriving the A_{eff} and EHL values from equations (A2-6) to (A2-9), HL_{cold}, HL_{hot}, RHP and ETH can be calculated from equations (4) to (7). This determines whether the environment is cold, hot or thermoneutral. When cold or hot,

T_{skin} is re-determined as $T_{skin} = T_0 - b_{SHL} \times HP$, using $b_{SHLcold}$ or b_{SHLhot} as appropriate and with HP in $W.m^{-2}$. Equations (A2-3), (A2-6) to (A2-9), and (4) to (7) can then be re-evaluated. When the environment is consequently thermoneutral, no additional action is taken.

When the environment is found to be hot, ME_{maint} of group-housed pigs is reduced by 7.5 % (or less, if that is sufficient to cover RHP) to reflect a decrease of physical activity by up to 50 %. The model's constrained feed intake parameter ConstrFI is recalculated by reducing the initially calculated feed intake by the thermoregulatorily required HP reduction as a proportion of the feed ME content (ConstrFI = FI – RHP / ME). The sections labelled "*do* DesiredFI" and "*do* Deposition" in Appendix I are then re-evaluated iteratively, until HP is reduced far enough to match HL_{hot} so that thermal inequilibrium (RHP > 0) is terminated.

When the environment is found to be cold, ETH is added to ME_{maint}. The sections labelled "*do* DesiredFI" and "*do* Deposition" in Appendix I are then re-evaluated iteratively, until HP is increased far enough to match HL_{cold} so that thermal inequilibrium (ETH > 0) is terminated, or until a feed intake volume constraint is reached (and thermal equilibrium is not attained).

Model behaviour

The model's output is likely to depend on three types of input variables: (i) the four parameters describing the animal's genotype: thermoneutral ME_{maint} (in terms of α in $ME_{maint} = \alpha \times BW^{0.75}$), mature body protein mass (P_∞, in kg), the ratio of mature body lipid to body protein mass ($R_{L\infty/P\infty}$, in $kg.kg^{-1}$), and the rate parameter of the Gompertz curve that describes "desired" body protein mass growth (B_{Gomp}, in $kg.d^{-1}.kg^{-1}$); (ii) nutritional parameters such as feeding level or volume intake capacity constraints, and diet composition (energy and protein content and digestibility, amino acid composition); (iii) climatic parameters (see Appendix II).

Sensitivity analysis

The extended model was evaluated repeatedly over a cumulative BW range from 23 to 100 kg, while varying T_{env} over [0, 1, ..., 39, 40] °C. Records were output to the analysis data set at BW values of [25, 35, 45, 60, 75, 100] kg. The genotype parameters were varied over [0.6, 1.0, 1.4] times the reference values of P_∞= 35.5 kg, $R_{L\infty/P\infty}$ = 2.90 $kg.kg^{-1}$ and B_{Gomp} = 0.0126 $kg.d^{-1}.kg^{-1}$ reported by Ferguson *et al.* (1994) for what seems to be an average Landrace/Large White genotype. The ME_{maint} regression coefficient α ($ME_{maint} = \alpha \times BW^{0.75}$) was varied over [0.7, 1.0, 1.3] times (495 – 0.75 × BW) $kJ.kg^{-0.75}.d^{-1}$ (from Chapter 1). The protein to energy ratio of the diet was varied by keeping DE content fixed at 13.26 $MJ.kg^{-1}$ feed while varying CP content over [0.15, 0.175, 0.20] $kg.kg^{-1}$. Air velocity (see Appendix II) was set to 0.15 $m.s^{-1}$, air water content was derived from relative ambient humidity (set to 0.7) according to Wilhelm (1976). Standardised thermal resistance of the floor was set to $I_{floor.45}$ = 0.07 $°C.m^2.W^{-1}$ to represent concrete slats, the maximum proportion of wetted skin in hot conditions to 0.15, and group size to one pig. Simulated feed intake was constrained by volume capacity with a constraint according to Ferguson *et al.* (1994) that put a maximum to *ad libitum* ME intake in cold conditions of up to 4.2 times thermoneutral ME_{maint}. All other model parameters were kept constant at values taken from Moughan and Verstegen (1988), Ferguson *et al.* (1994), and Chapter 1. The analysis data set thus held $41 \times 6 \times 3^5 = 59778$ possible [T_{env} × BW × genotype × CP] records. About 20 % of these lost so much lipid (mainly at the lower

and higher body weights in cold and hot conditions, respectively) that their simulation had to be terminated, either because of negative L or BF, or because the replicate needed more than 300 days to reach end weight. Hence only 47897 records were realised. These 20 % "lost" records do not result from a model anomaly, they just suggest that certain pigs in those conditions will grow very slowly, or die because they reach T_{cold} or T_{hot}.

The data set was split into three parts with records for cold, thermoneutral, or hot conditions, respectively, based on the outcomes of equations (4) and (5). HP, P_{dep}, L_{dep} and FI in the "cold" and "hot" subsets were expressed as proportions of their "desired" thermoneutral levels (nHP, dP_{dep}, dL_{dep} and dFI). The regression coefficients of HP/nHP, P_{dep}/dP_{dep}, L_{dep}/dL_{dep} and FI/dFI on T_{env} were estimated within each [BW × genotype × CP] subclass, describing the rate at which each trait changes with increasing T_{env} in cold or hot conditions. The minimum and maximum T_{env} value within each subclass of the "thermoneutral" subset were taken to represent T_{LC} and T_{UC}, which were thus implicitly rounded to whole °C. These approximated T_{LC} and T_{UC}, and the regression coefficients of "cold" and "hot" HP/nHP, P_{dep}/dP_{dep}, L_{dep}/dL_{dep} and FI/dFI on T_{env} were subjected to analysis of variance to quantify their dependence on BW, ME_{maint}, P_{∞}, $R_{L\infty/P\infty}$, B_{Gomp}, and feed CP. The ANOVA model for each of the ten dependent traits comprised these six factors as main effects plus the five two-way interactions of BW with the other factors. Eighty of the 60 main effects and 50 interactions with BW were significant ($0.0001 \leq P < 0.1$). The outstanding case is hot L_{dep}/dL_{dep} which was not significantly affected ($0.16 \leq P \leq 1.0$) by any of the 11 factors tested. The other 19 non-significant effects did not show any obvious pattern. Hence the simulated variation in genotype parameters and feed CP resulted in significant changes in simulated T_{LC} and T_{UC} and in the rate at which simulated cold and hot HP/nHP, P_{dep}/dP_{dep}, L_{dep}/dL_{dep} and FI/dFI change with increasing T_{env}. Most of these changes were significantly different at different BW levels.

Comparison with external data

The pronounced sensitivity of the thermoregulatory part of the model for genotype characteristics makes it practically impossible to simulate a given real-life situation without an exhaustive description (in terms of *ad libitum* feed intake, thermoneutral ME_{maint}, and protein and lipid deposition traits) of the genotype under consideration. Because most reports on thermoregulatory processes in pigs give a poor description of the genotypes used, the model's behaviour can hardly be compared with experimental results from the literature in a meaningful deterministic way. Hence a number of published trials were simulated with the same range of simulated genotype characteristics as in the previous section, *i.e.* varying P_{∞}, $R_{L\infty/P\infty}$, B_{Gomp} over [0.6, 1.0, 1.4] and (where appropriate) the ME_{maint} regression coefficient over [0.7, 1.0, 1.3] times their reference values. This range is assumed to cover the pig genotypes available in the western world during the last few decades. When the model performs well, the results of a narrow range of its simulated genotypes should coincide with the findings of the associated trial.

Comparison with external data: voluntary feed intake

The most important feature of Emmans's (1997) model, for the purposes of this study, is the prediction of *ad libitum* feed intake as an output. The influence of T_{env} on this trait in growing pigs was quantified by Straub *et al.* (1976; further referred to as "Straub"), by Nienaber *et al.*

(1987; "Nienaber") and by Hyun *et al.* (1998; "Hyun"). These trials are difficult to simulate because their conditions were poorly documented and because they started their measurements at relatively high BW without any relevant information on the pre-trial phase or on body composition at start weight. The known trial characteristics are in Table 1. Straub worked with German Landrace males, Nienaber with American four-way crossbred females, Hyun with American Duroc and [Yorkshire × Hampshire] castrates and females.

The same range of genotypes as for the above sensitivity analysis was simulated while imposing the respective environmental characteristics of these studies (Table 1). The simulation results have been combined with the experimental findings in Figure 2, which shows *ad libitum* FI in relation to T_{env} for one of the three simulated thermoneutral ME_{maint} levels. The three ME_{maint} clusters show much overlap within each trial, and the single plots are easier to interpret.

Table 1 Trial characteristics of the experiments used for comparison with the model's prediction of ad libitum feed intake

	source[†]		
	Straub	Nienaber	Hyun
BW range (kg)	70 to 110	44 to 87	35 to 58
feed DE, CP content ($MJ.kg^{-1}$, $kg.kg^{-1}$)	[?, 0.14] (DE set at 13.1)	[13.1, 0.16]	[14.4, 0.17]
CP digestibility	? (set at 0.77)	? (set at 0.77)	? (set at 0.77)
group size	1	2	8
T_{env} (°C)	15, 35	5, 11.5, 15, 20, 26, 31	24, 28 to 34
air humidity	0.60	0.70 (5 °C) to 0.37 (31 °C)	? (set at 0.4)
air velocity ($m.s^{-1}$)	? (set at 0.12)	0.12	? (set at 0.12)
pen floor type	straw litter	wire mesh	"partly slotted"
pen size (m^2 per pig)	?	1.0	0.56

[†] Straub *et al.* (1976); Nienaber *et al.* (1987); Hyun *et al.* (1998)

The simulated FI patterns in Figure 2 generally follow the trend of HL with increasing T_{env} that was postulated in Figure 1, but the $3^3 = 27$ genotypes in each of the three trial simulation clusters show a considerable variation due to the simulated variation in the genotype parameters, especially in cold and thermoneutral conditions. Nienaber's and Straub's higher-ranking genotypes could not increase their thermoneutral FI in cold conditions as a result of the imposed volume constraint (see the previous section).

The model fits Straub's two data points well which is almost trivial at 15 °C, considering the wide variation of simulated thermoneutral FI. But hot FI is reduced so drastically that hardly any variation remains at the highest T_{env} levels, and the 35 °C data point is predicted remarkably accurately. Nienaber's and Hyun's experimental results coincide with the FI values for a

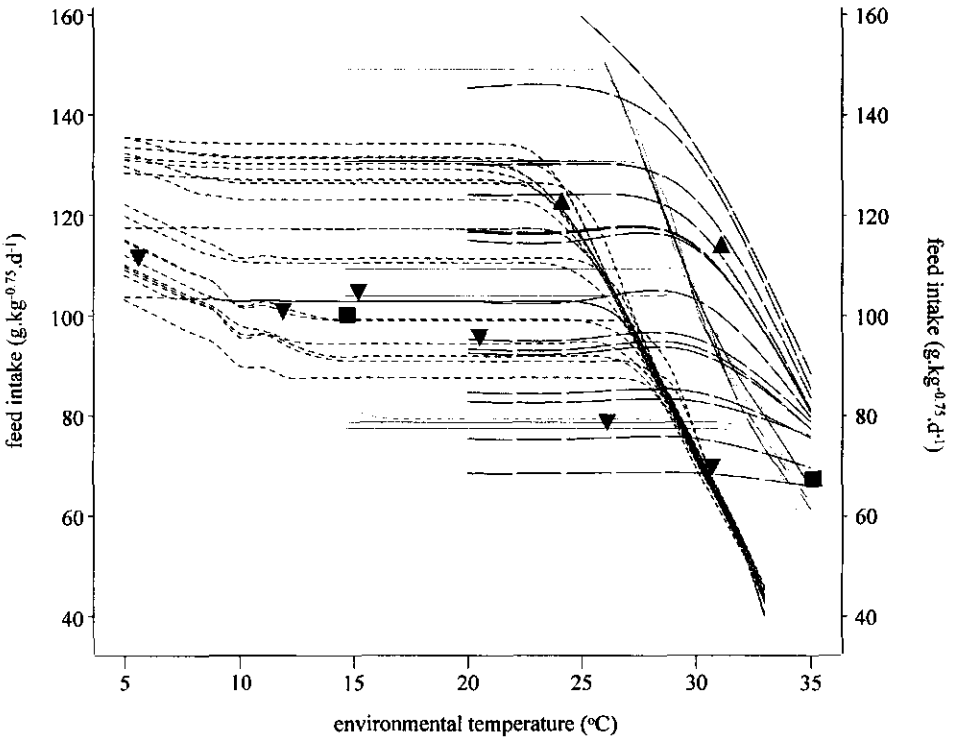

Figure 2 Daily *ad libitum* feed intake in relation to environmental temperature. Scatterplots: experimental results. Line plots: results of simulations of the trials that produced those results: Straub *et al.* (1976; ■ ——); Nienaber *et al.* (1987; ▼ -----); Hyun *et al.* (1998; ▲ — —). The variation between the lines within plots is due to simulated variation in the model's genotype parameters (see text for details).

reasonably narrow range of simulated genotypes. This is somewhat of a surprise for Hyun's data, considering that these derive from two sexes and two very different genotypes, and that his higher T_{env} level cycled between 28 and 34 °C. Bearing in mind the uncertainties with which these trials could be simulated, the model predicts *ad libitum* FI reasonably well over the range of temperatures considered.

Comparison with external data: critical temperatures

Critical temperatures provide a summary of much of the simulated thermoregulatory processes. T_{LC} and T_{UC} values published since 1970 were reviewed by Holmes and Close (1977). Their findings have been combined with simulated T_{LC} and T_{UC} from the above sensitivity analysis in Figure 3. This Figure shows these data for three BW levels in relation to *ad libitum* ME intake as a multiple of thermoneutral ME_{maint}. The latter parameter was set to 420 kJ.kg$^{-0.75}$.d^{-1} in Holmes and Close's (1977) review.

The simulation results in Figure 3 fall apart in three clusters, associated with the three levels of simulated thermoneutral ME_{maint}. The higher ME_{maint} levels coincide with the lower T_{LC} and T_{UC} values ($P < 0.0001$). The remaining dispersion is due to differences in the other genotype parameters and feed CP, which cause variation in heat production and in heat loss. The former is

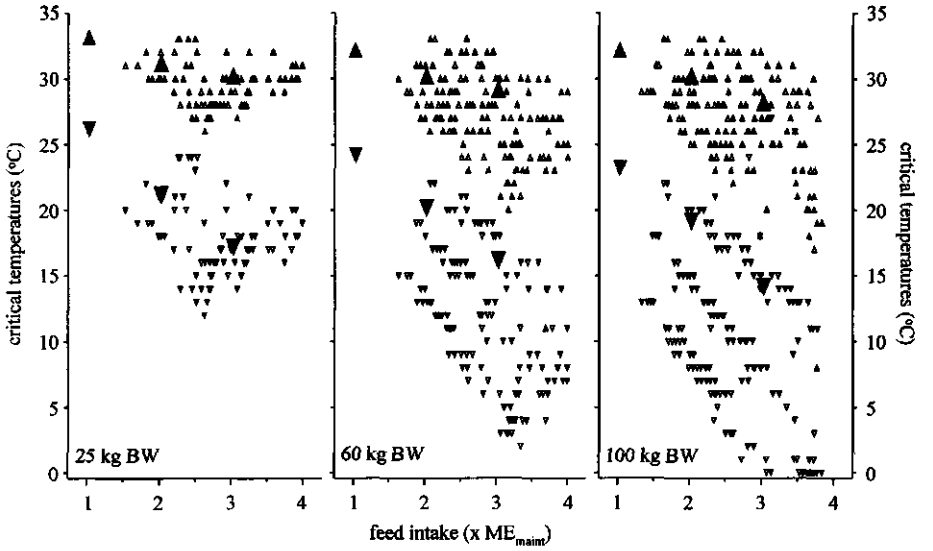

Figure 3 Lower (▼) and upper (▲) critical temperatures in relation to *ad libitum* ME intake as a multiple of thermoneutral ME_{maint}. Large solid symbols: experimental data as reviewed by Holmes and Close (1977). Small open symbols: simulation results. The three graphs represent body weight at 25 kg (20 kg for the experimental data), 60 and 100 kg. The three clusters in each plot are due to differences in simulated ME_{maint}; the dispersion within each cluster is due to simulated variation in the model's genotype parameters (*see text for details*).

a consequence of differences in the heat increment of maintenance and deposition processes (equation (3)), the latter of differences in (cold) tissue insulation through differences in body lipid content (Appendix III).

The overall variation within each ME_{maint} cluster (*e.g.* the 12 deg difference between the extreme T_{LC} values in the lowest cluster at 60 kg BW) is mainly due to the large variation in simulated *ad libitum* FI (see Figure 2). The much smaller variation within each FI level is due to genetic differences in ME partitioning into P_{dep} and L_{dep} and the associated heat increment (equation (3)). Considering the above described sensitivity of the critical temperatures for the genotype parameters, and the different way the horizontal axis scaling parameter (ME_{maint}) was obtained in the review *versus* the simulation, Figure 3 shows a satisfactory similarity between simulated T_{LC} and T_{UC} and experimental results.

The patterns in Figures 2 and 3 show that proper simulation of thermoregulatory processes requires proper characterisation of the genotype of interest, for this particular model in terms of thermoneutral ME_{maint}, B_{Gomp}, P_{∞}, and $R_{L\infty/P\infty}$, in that order of priority.

Comparison with external data: energy partitioning

Another useful summary of thermoregulatory processes can be found in the partitioning of consumed ME into HP, P_{dep} and L_{dep} in different thermal conditions. The influence of (controlled, and mostly strongly restricted) feeding level and T_{env} on this partitioning in young growing Landrace/Large White-type females and castrated males has been studied by Fuller and

Boyne (1971, 1972; further referred to as "Fuller"), by Holmes (1973; "Holmes"), by Verstegen *et al.* (1973; "Verstegen") and by Close and Mount (1978) and Close *et al.* (1978; "Close"). The same range of genotypes as for the above sensitivity analysis (with fixed thermoneutral ME_{maint} as in Table 2) was simulated while imposing the respective environmental characteristics of these studies (Table 2). The trial of Holmes is difficult to simulate because his measurements commenced five to seven weeks after the trial was started, and the pigs spent alternate periods in calorimeters and in pens with quite different thermal environments. As Holmes's published results are cumulative, intermediate conditions had to be simulated.

Table 2 Trial characteristics of the experiments used for comparison with the model's prediction of ME partitioning

	source[†]			
	Fuller	Holmes	Verstegen	Close
BW range (kg)	25 to 85	18-27 to 70	24–28 to 35–45	22–38 to 24–50
trial duration (d)	(to end weight)	(to end weight)	21	14
pre-trial acclimation period (d)	0	0	35	14
thermoneutral ME_{maint} ($kJ.kg^{-0.75}.d^{-1}$)	420 to 450	270 to 410	420	440
feed DE, CP content ($MJ.kg^{-1}$, $kg.kg^{-1}$)	[13.1, 0.173] → [12.9, 0.140][‡]	[13.7, 0.204]	[12.6, 0.180]	[12.4, 0.162]
CP digestibility	0.81	0.86	0.77	? (set at 0.77)
group size	1	1	4	1
T_{env} (°C)	5, 13, 23	25, 25 → 33[§]	8, 20	10, 15, 20, 25, 30
air humidity	?	0.3-0.4 (25 °C) to 0.5-0.8 (33 °C)	0.90 (8 °C) to 0.74 (20 °C)	0.81 (10 °C) to 0.47 (30 °C)
air velocity ($m.s^{-1}$)	0.45 (0.75 at 5 °C)	< 0.15	? (set at 0.12)	? (set at 0.12)
pen floor type	wire mesh	concrete slats, wire mesh	wooden slats	wooden slats
pen size (m^2 per pig)	0.16 to 0.56	1.2	1.38	1.25
ME intake (× ME_{maint})	2.1, 2.7, 3.2, 3.8, 4.4 (3 levels per T_{env})	2.1, 2.7	2.6, 3.0, 3.5 (2 levels per T_{env})	1, 2, 3, *ad libitum* (orthogonal with T_{env})

[†] Fuller and Boyne (1971, 1972); Holmes (1973); Verstegen *et al.* (1973); Close and Mount (1978), Close *et al.* (1978)

[‡] Diet change at 50 kg BW

[§] Temperature change at 38-50 days on trial

The simulation results have been combined with the experimental findings in Figure 4. This Figure shows HP and the energetic equivalents of P_{dep} and L_{dep} in relation to ME intake for thermoneutral (T_{env} from 20 to 23 °C) and hot (30 to 33 °C) conditions. Close's results at 15 and 25 °C follow similar courses as at 20 °C and are not shown here.

In the simulation runs it was attempted to realise ME intake levels similar to those reported for the trials. The close proximity of the $3^3 = 27$ simulated genotype-specific lines in each of the 12 + 6 = 18 [trial × trait × T_{env}]-specific line plots in Figure 4 shows that ME restriction allows for

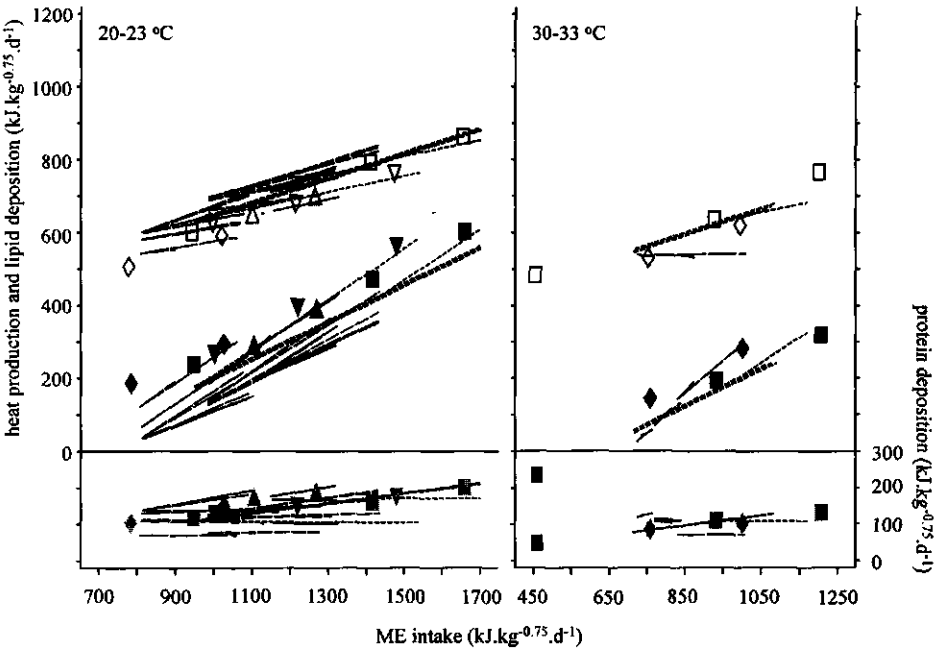

Figure 4 Partitioning of ME intake into heat production (open symbols), lipid deposition (L_{dep}; black solid symbols) and protein deposition (P_{dep}; grey solid symbols) in thermoneutral (20 to 23 °C) and hot (30 to 33 °C) conditions. Scatterplots: experimental results; line plots: results of simulations of the trials that produced those results: Fuller and Boyne (1971, 1972; ▼ – – –); Holmes (1973; ◆ — —); Verstegen *et al.* (1973; ▲ ——); Close and Mount (1978) and Close *et al.* (1978; ■ --------). The variation between the lines within each plot is due to simulated variation in the model's genotype parameters (see text for details). Note that the P_{dep} plots were lowered to avoid overlap with L_{dep} plots, and that the horizontal axes overlap.

little expression of the genetic variation in HP, L_{dep} and P_{dep} that is due to the simulated variation in P_∞, $R_{L\infty/P\infty}$ and B_{Gomp}, especially so in the most constraining (hot) conditions.

The model predicts thermoneutral and hot P_{dep} accurately. Close's and Verstegen's (hot and) thermoneutral L_{dep} and HP data are predicted well, too. Holmes's and especially Fuller's thermoneutral HP results are somewhat overestimated (in the sense that the data fall outside our simulated range of genotypes), consistently so over the whole range of evaluated ME intake levels. The associated L_{dep} results are underestimated (in the same sense as for HP).
To some extent, the opposite holds for Holmes's hot HP: this is predicted well at the lower ME intake level, but the model predicts HP to level off at the higher ME intake whereas Holmes's pigs were able to increase their HP. Holmes's hot L_{dep} is predicted well at both ME intake levels.

Figure 5 shows the same relations as Figure 4, for conditions around and below T_{LC} (T_{env} from 8 to 13 °C). This Figure does not show Fuller's results at 5 °C, which evaluates to an effective T_{env} (Curtis, 1983) of about –10 °C as a consequence of the high air velocity and the non-insulating floor material in that trial. It does not show Close's 10 °C results at the lowest ME intake level (maintenance feeding) either. Both were predicted very poorly, the model is clearly

not able to deal with subcritical thermal conditions of this severity. Figure 5 shows the same constraining effect of low ME intake on the expression of genetic variation as Figure 4, especially so in the most severely cold conditions. Apart from the above mentioned extreme subcritical cases, the model predicts cold P_{dep} accurately. At a given T_{env}, the course of HP with decreasing ME intake is expected to follow the thermoneutral declining trend until T_{LC} is reached. Below T_{LC} the trend should become horizontal because "increased food intake is not associated with an increase in heat loss [in cold conditions]; the energy dissipated in association with protein and fat retention spares some of the animal's heat production" (Verstegen *et al.*, 1973), and because of an increase in shivering thermogenesis.

Close's experimental results in Figure 5 show that pattern, as do the results of the simulation of his trial. But the model predicts a less steep thermoneutral HP decline at 10 °C (also less than its decline at 20 °C in Figure 4), so that about half of the simulated genotypes level off their HP plots in Figure 5 at a similar ME intake but a higher HP level than Close's pigs did. The other ones do so at a much higher ME intake. Hence the model again overestimates the absolute magnitude of subcritical HP, and underestimates that of L_{dep}, although the simulation follows a similar trend against ME as the experimental data. Verstegen's cold HP results suggest that at 8 °C his group-housed pigs reached their T_{LC} at an ME intake above 1.5 $MJ.kg^{-0.75}.d^{-1}$. The same holds for some of the simulated genotypes, which show a horizontal trend. Others are still on thermoneutral decline in Figure 5 and hence must reach T_{LC} at a lower ME intake level.

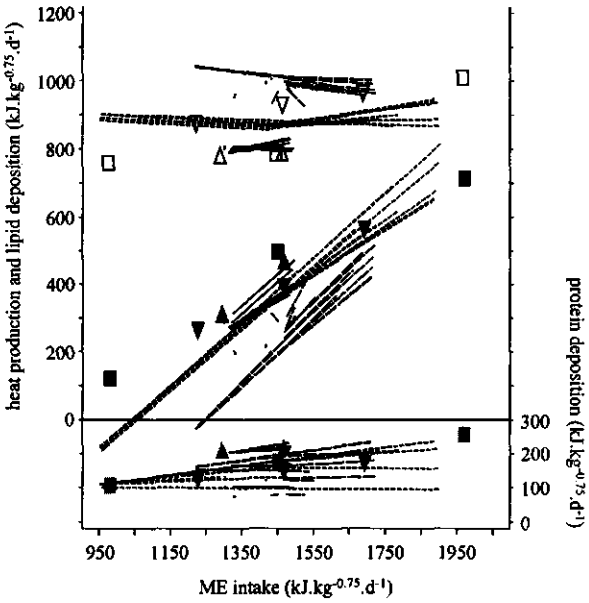

Figure 5 Partitioning of ME intake into heat production (open symbols), lipid deposition (L_{dep}, black solid symbols) and protein deposition (P_{dep}, grey solid symbols) in cold (8 to 13 °C) conditions. See Figure 4 for legend and other information.

The model predicts Fuller's pigs to have generally reached T_{LC} at an ME intake higher than 1.7 $MJ.kg^{-0.75}.d^{-1}$, as the line plots for HP are horizontal below that level. This is not supported by his experimental results, which still seem to be on thermoneutral HP decline well below that ME intake level. Fuller's trial lasted much longer than Verstegen's and Close's (see Table 2), so his pigs had more opportunity to acclimatise to the cold environment, for example by developing a coat of any significant depth. Coat insulation was ignored in the model (see Appendix II), and acclimatisation may explain why Fuller's data still suggest thermoneutrality at about 1.5 $MJ.kg^{-0.75}.d^{-1}$. Around that ME intake level, the simulation of

some genotypes had to be terminated because they had lost all their body lipid at about 40 kg BW. The remaining simulated genotypes slightly *increase* their HP with a further reduction of ME intake, in an attempt to meet the ever increasing environmental heat demand that results from the poorer insulation of these much leaner simulated pigs.

In summary, the model accurately predicts P_{dep}, and also the trends of HP and L_{dep} against T_{env} and ME intake, in all thermal conditions apart from extremely cold ones. But it tends to overestimate absolute HP and underestimate the associated L_{dep} levels, in the sense that the experimental data fall outside our simulated range of genotypes. The model predicts cold and thermoneutral HP and L_{dep} much better at higher ME intake levels, which may suggest that its poor performance in cold conditions is associated with a lack of accuracy in the assessment of T_{LC} at low ME intake. Figure 3 suggests that the main factor responsible for a poor T_{LC} assessment would be an improper estimate of the genotype's thermoneutral ME_{maint}. This may be a shortcoming of the simulation as well as of the experimental findings. Alternatively, the range of proportional genotype parameters used in the simulations may have been too narrow to include Fuller's pigs.

Discussion

In this study it was attempted to integrate current quantitative knowledge of thermoregulation (Appendix II) into a dynamic growth simulation model, describing the thermoregulatory processes as functions of body protein and lipid content or deposition where appropriate. This quantitative knowledge is far from complete, especially with regard to hot conditions, and the system involves a large number of parameters. This study focuses on the animal-specific parameters, which are treated as model variables. The "climatic" parameters have been given much more attention elsewhere, and have been treated largely as constants here.
Apart from the relation between body fatness and tissue insulation described in Appendix III, this study presents few novel principles. Most of the thermoregulatory relations in Appendix II were taken from the existing literature, although those describing evaporative heat loss were updated to some extent. However, the few models for thermoregulation in growing pigs that have been published up to now have not been extensively compared to real-life data, and this study presents such a comparison in Figures 2 to 5. The outcome of that comparison can be summarised as in the *Adequacy of the model* section below. Moreover, none of the comparisons of growth simulation results with real-life data that appear in the literature have taken into account that pig genotypes differ considerably in their elements of our equation (3), and hence should be properly characterised during simulation for the comparison to be meaningful. This study illustrates the possible magnitude of the variation between genotypes, and presents a framework to deal with this issue in a general way.

Adequacy of the model

The routine that generated the simulation results in Figures 2 to 5 comprises most of Emmans's (1988, 1994, 1997) thermoneutral potential growth rate and feed intake algorithm, the protein turnover rules derived by in Chapter 1, and the thermoregulation rules by Bruce and Clark (1979) and Black *et al.* (1986), all embedded in the framework of Moughan and Smith's (1984) model. Those thermoregulation rules were modified according to Appendix III in order to relate tissue insulation to body composition, but the impact of that modification on overall

thermoregulatory performance should be small, see the *Fat depth and tissue insulation* section below. A more elegant alternative might have been to use Black's model throughout, but that was not possible because it has been commercialised and is not freely accessible, and was incompletely documented by Black *et al.* (1986). Hence the performance of the resulting model in thermoneutral conditions (Figures 2 and 4) is indicative of the predictive quality of Emmans's algorithm in combination with our protein turnover equations. The comparison of its thermoneutral *versus* cold and hot predictive performance is indicative of the quality of the above mentioned thermoregulation rules.

Very generally, the model yields realistic predictions of *ad libitum* feed intake (Figure 2), and predicts the thermoneutral partitioning of *ad libitum* ingested ME into heat production and protein and lipid deposition reasonably accurately (Figure 4). Heat production is overestimated and lipid deposition is underestimated when ME intake becomes restricted (Figure 4). The model predicts the lower and upper critical temperatures adequately (Figure 3). Its prediction of *ad libitum* ingested ME partitioning in non-thermoneutral conditions is not markedly better or worse than at thermoneutrality (Figures 4 and 5) as long as those conditions do not get extremely cold.

We have chosen not to attempt to include long-term acclimatisation to cold or hot conditions into the model, mainly because there seems to be little quantitative information on this issue in the literature. This omission is the most likely reason for the model's poor prediction of the experimental results by Fuller and Boyne (1971, 1972) in Figure 5. Simulation models such as the one evaluated here would benefit considerably from the inclusion of a routine that could properly deal with these adaptative processes.

The UNICORN heat exchange model by Turnpenny *et al.* (2000) derives from Bruce and Clark's (1979) and McArthur's (1981a, 1987) rules in a way similar to the model evaluated here, although it was designed to provide answers to a different type of questions. When Turnpenny (1997) compared its predictions to experimental data, the model overestimated cold HP of group-housed pigs by about 20 %, similar to some of our findings in Figure 5. Turnpenny *et al.* (2000) attribute this discrepancy to UNICORN not taking into account the insulative effect of huddling (which is dealt with by our equation A2-5) and posture compaction in the cold. On the other hand it includes effects of shivering on tissue insulation, which our model does not. Figure 5 also suggests that the UNICORN model may have been inadequately parameterised to simulate the genotypes that its output was compared with.

The model's partitioning of ME into lipid deposition and heat production is largely regulated by the ME requirements for protein and lipid deposition (ME_{Pdep} and ME_{Ldep}), see equation (3) and Appendix I. Both parameters were set to 53 $MJ.kg^{-1}$ according to Moughan and Verstegen (1988), which corresponds to net efficiencies of protein and lipid deposition of k_P = 23.8 / 53 = 0.45 and k_L = 39.6 / 53 = 0.75, respectively. Tess *et al.* (1984) reviewed literature estimates of $31.4 \leq ME_{Pdep} \leq 66.8$ and $39.7 \leq ME_{Ldep} \leq 68.0$ for pigs, and report a strong dependence of the estimates of these parameters on ME_{maint}, which was either estimated simultaneously or assumed fixed.

The model's fit of the experimental ME partitioning data in Figures 4 and 5 was improved considerably, but to different extents for the various simulated trials, when ME_{Pdep} and ME_{Ldep} were set to 50 $MJ.kg^{-1}$ (*i.e.* k_P = 0.48 and k_L = 0.79). Within the framework of equa-

tion (3), this would lead to the conclusion that those two parameters should be treated as model variables rather than constants, which was already implied by Whittemore and Fawcett (1976). Generally, a large variation among estimates of a model parameter assumed to be a biological constant (such as the above ranges by Tess *et al.*, 1984) suggests that (i) this assumption was wrong and the parameter should be treated as a variable as proposed above, or (ii) the variation among the estimates is due to another parameter not (or not correctly) included in the model, especially when the estimates turn out to be strongly dependent on other parameters (*e.g.* on ME_{maint} in the data of Tess *et al.*, 1984). Equations (1) and (3) may be regarded as an oversimplification of ME partitioning. Following Emmans (1994), heat production can be expressed as an extension of equation (3):

$$HP = ME^*_{maint} + HI_{Pdep} \times P_{dep} + HI_{Ldep} \times L_{dep} + HI_{FI} \times (FI - FI_{maint}) \quad (8)$$

where HI_{FI} denotes the heat increment of feed intake (about 2.5 $MJ.kg^{-1}$ feed for a typical pig diet; G.C. Emmans, personal communication, 1998), FI denotes actual feed intake ($kg.d^{-1}$), and FI_{maint} denotes the feed consumed to satisfy maintenance requirements ($kg.d^{-1}$; obviously dependent on the feed ME content). ME^*_{maint} comprises fasting heat production plus the heat increments of endogenous urinary nitrogen and fecal organic matter at $P_{dep} = L_{dep} = 0$, which is considerably less than the original ME_{maint} parameter. HI_{Pdep} and HI_{Ldep} would be true constants now, with estimated values of 36.5 and 16.4 $MJ.kg^{-1}$ (Emmans, 1994). Because equation (3) does not specifically account for the heat increment of feed intake above maintenance (the last term in equation (8)), this term becomes included in the estimates of ME_{maint}, HI_{Pdep} and HI_{Ldep} when relating data to equations (1) or (3). Hence these parameters become diet-dependent and intercorrelated, and cannot be assumed constant.

The "desired" voluntary feed intake as predicted by Emmans's (1997) rules aims to satisfy the animal's requirements for both protein and energy, in contrast to the conventional point of view that animals eat to fulfill their energy requirements only, see Appendix I. Evidence in support is given by Kyriazakis *et al.* (1991) and Ferguson and Gous (1997), among others. This issue is discussed in more detail by Galef (1991).

Ambient temperature and vasomotor action

There is some disagreement in the literature concerning the nature of vasomotor action in the cold, especially with regard to the rate at which $b_{SHLcold}$ becomes operational below T_{LC}. This discussion is confused because studies have focused on animals of vastly different stage of development and allowed different acclimation periods, and also because vasomotor processes in the extremities appear to differ strongly from those in the central part of the body. The evidence on this matter is conflicting (Gonzalez-Jimenez and Blaxter, 1962; Fuller and Boyne, 1972; Bligh, 1973; Stombaugh *et al.*, 1973; Holmes and McLean, 1974; Stombaugh and Roller, 1977). In the approach outlined in equations (5) and (6), $b_{SHLcold}$ is supposed to be fully operational below T_{LC}. If in reality peripheral vasoconstriction is attained only gradually (first in the extremities and later on the central part of the body) with decreasing subcritical T_{env}, the model would overestimate $I_{tissue,cold}$ at T_{env} close to T_{LC}. This would lead to underestimation of SHL_{cold} and HP in such conditions, which is in contradiction with our previous findings in Figure 3, where cold HP is mostly *over*estimated.

Hot evaporation

The processes described in Appendix II quantify the thermal environment in terms of ambient temperature, air velocity, floor thermal resistance, group size, relative air humidity, and the pig's options for wetting its skin in hot conditions. Some of these parameters were used by Close (1981) and Curtis (1983) to assess the "effective environmental temperature" by rule-of-thumb, and our model was found to perform roughly according to those rules.

The parameter A_{wet} is crucial for quantification of EHL_{hot} in terms of physical descriptors such as equation (A2-10). This is illustrated in Figure 6 which shows, for a representative replicate of the above described sensitivity analysis, how T_{UC} and T_{hot} were influenced by the three types of metabolic action employed in the model to retain thermal equilibrium: (i) allowing T_{body} to increase to up to 40.5 °C, (ii) reducing HP by up to 7.5 % as a result of halved physical activity, and (iii) increasing EHL_{hot} by wetting of up to 15 % of the skin area. The model allows for these actions to be taken simultaneously, but in Figure 6 their cumulative effects are shown stepwise for illustrative purposes. The effect of a simple action like (iii) on T_{hot} and especially on T_{UC} is clearly much larger than the effects of metabolically serious actions as (i) or (ii), particularly so at higher BW levels. It follows that quantification of A_{wet} is critical for proper simulation of a real-life situation with hot conditions.

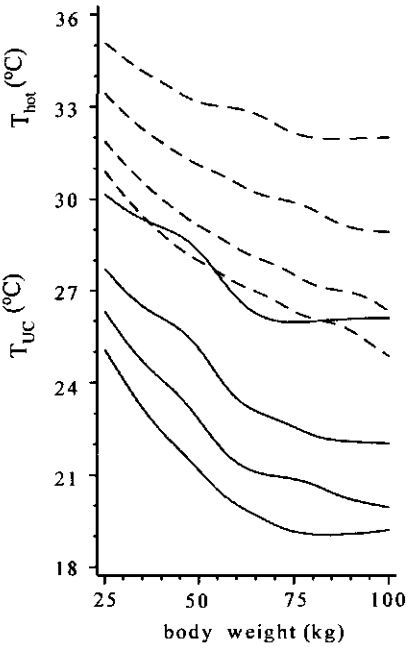

Figure 6 Simulated upper critical temperature (T_{UC}, ——) and hyperthermic point (T_{hot}, -----) in relation to body weight, dependent on cumulative thermoregulatory actions. From bottom to top: (i) thermoneutral metabolism; (ii): as (i), body temperature allowed to increase to 40.5 °C; (iii): as (ii), heat production decreased by 7.5 % through reduction of physical activity; (iv): as (iii), with additional evaporative heat loss from 15 % wetted skin area.

At the same time, however, A_{wet} is the most poorly documented parameter in the model. Pigs in hot and confined conditions have been reported to use much more water than they could be expected to drink (Mount *et al.*, 1971; Straub *et al.*, 1976; Giles *et al.*, 1988), and also to actively wet their pen with drinking water (Nienaber *et al.*, 1996) but A_{wet} seems to have never actually been quantified (Black *et al.*, 1998). Given the importance of this parameter, a model that aims at a proper prediction of HL_{hot} should be able to predict A_{wet} as an endogenous variable rather than request it as an input parameter as in equation (A2-10).

Fat depth and tissue insulation

The regression line for cold tissue insulation in Figure 7 (Appendix III) may seem to be disproportionately influenced by the data points of the very fat sows by Holmes and McLean (1974). Cook's D statistics of the data points above 15 mm BF range from 0.0037, with the highest

values at 0.022, 0.047, and 0.056 for BF = 43, 31, and 38 mm, respectively. Neter *et al.* (1985) suggest to relate Cook's D values to "the corresponding F distribution [...] and ascertain the percentile value. If [this] is less than about 10 or 20 percent, the [...] observation has little apparent influence on the fitted regression function. If [it] is near 50 percent [the] observation has a substantial influence". The above mentioned D values correspond to percentile values of 0.12, 0.17 and 0.19 of the appropriate F^{1}_{33} distribution, respectively. Omitting these three data points from the regression gives $I_{tissue} = 0.049 + 0.0024 \times BF$, indistinguishable from the relation established earlier. The short regression line in Figure 7 runs through the data points below 15 mm BF only: $I_{tissue} = 0.056 + 0.00162 \times BF$. It would have been useful to include data on pigs with 15 to 30 mm BF in this analysis, but these could not be found in the literature. Trials in which skin temperature, heat production, *and* fat depth were measured (*and* reported upon) seem very scarce.

An interesting statistic is the change in the T_{env}-related decline of SHL as a consequence of a change in average subcutaneous fat depth (FAT), $\frac{d(b_{SHL})}{d(FAT)}$. This can be rewritten as $\frac{d(b_{SHL})}{d(I_{tissue})} \times \frac{d(I_{tissue})}{d(FAT)}$, the first term of which equals $\frac{-1}{(I_{tissue} + I_{air})^2}$. Substituting $I_{t0} + \frac{d(I_{tissue})}{d(FAT)} \times FAT$ for I_{tissue} (assuming a hypothetical insulation I_{t0} at zero fat depth) gives

$$\frac{d(b_{SHL})}{d(FAT)} = \frac{-\frac{d(I_{tissue})}{d(FAT)}}{\left(I_{t0} + \frac{d(I_{tissue})}{d(FAT)} \times FAT + I_{air}\right)^2} \qquad (9)$$

When $I_{t0} = 0.05$ °C.m^2.W^{-1} and $\frac{d(I_{tissue})}{d(BF)} = 0.002$ °C.m^2.W^{-1} per mm P2 backfat depth (Appendix III), and $I_{air} = 0.13$ °C.m^2.W^{-1}, then $\frac{d(b_{SHL,cold})}{d(BF)} = \frac{-0.002}{(0.05 + 0.002 \times BF + 0.13)^2}$ W.m^{-2}.°C^{-1} per mm BF. An increase of P2 backfat depth by one millimeter from initial values of 10 and 20 mm (*i.e.* by 5 to 10 % of its average level) would lead to a reduction of $b_{SHLcold}$ by 0.049 and 0.041 W.m^{-2} per °C, respectively (*i.e.* about 1 % of its average level). Hence the reduction of fat depth that is the result of routine selection programs can be expected to have only a limited influence on cold thermoregulatory capacity.

Variation between animals

This study shows that *ad libitum* feed intake of growing pigs can be simulated with a considerable degree of variation between animals as a result of simulated variation in the model's genotype parameters (Figure 2), which include thermoneutral ME_{maint}. At a given level of feed intake and a given level of thermoneutral ME_{maint}, the lower and upper critical temperatures (Figure 3) and the ME partitioning into P_{dep}, L_{dep} and HP (Figures 4 and 5) show a clear genotype-specific variation between animals. This is relevant for model validation: model output has frequently been compared to real-life data without a proper characterisation of the geno-

type under concern, and our results show that this may accidentally lead to an almost perfect match and just as easily to complete predictive failure.
This variation is largely due to simulated differences in body composition, and the model could be used to quantify the relations between body composition and energy expenditure for thermoregulation (or "non-thermoneutral ME_{maint}"), which has not been done extensively before. That would involve stochastic simulation, and it will be reported upon separately.

Acknowledgements

Valuable contributions were made by Martin Verstegen, Gerry Emmans, Ella Luiting, John Black, and John Turnpenny. Henry Jørgensen and Andreas Susenbeth, and Prins van der Hel and Henk Brandsma, kindly made available their data used in Appendices II and III, respectively.

Appendix I. An outline of the simulation model used in this study.

The aspects of our computer algorithm that are necessary for the understanding of the present text have been coded below.

start
read feed composition (ME, CP, amino acids, digestibility coefficients)
read amino acid pattern of "ideal" protein
read ME requirement parameters for body maintenance (b_1, b_2)
read ME costs and heat increment of protein and lipid deposition (ME_{Pdep}, ME_{Ldep}, HI_{Pdep}, HI_{Ldep})
read ME yield of protein deamination (ME_{Pdeam})
read mature body protein mass, mature body lipid to protein ratio, Gompertz rate parameter (P_∞, $R_{L\infty/P\infty}$, B_{Gomp})
read body weight at start and end (BW_{start}, BW_{end})
determine chemical score of feed CP, ME in feed CP
determine efficiency of protein deposition (eff_{Pdep})
determine amounts of balanced and deaminated protein, and non-protein ME per g feed
set start values for body protein, lipid, and inorganic matter (P, L, ash)
$BW = BW_{start}$
days = 0
while $BW < BW_{end}$ *do*
  days = days + 1
  determine desired protein and lipid deposition (dP_{dep}, dL_{dep})
  determine ME and protein requirements for body maintenance (ME_{maint}, P_{maint})
  determine volume feed intake constraint (ConstrFI)
  do DesiredFI
    $P_{req} = P_{maint} + dP_{dep} / eff_{Pdep}$
    $ME_{req} = ME_{maint} + ME_{Pdep} \times dP_{dep} + ME_{Ldep} \times dL_{dep}$
    $dFI_{ME} = ME_{req} / ME$
    $dFI_P = P_{req} /$ balanced protein
    $dFI = \max(dFI_{ME}, dFI_P)$
  end do DesiredFI
  do Deposition
    $FI = \min(ConstrFI, dFI)$
    determine intake of "balanced" and deaminated protein
    determine non-protein ME intake
    P_{prod} = balanced protein $- P_{maint}$
    $P_{dep} = min(eff_{Pdep} \times P_{prod}, dP_{dep})$
    ME_{+deam} = non-protein ME + deaminated protein $\times ME_{Pdeam}$
    $ME_{prod} = ME_{+deam} - ME_{maint} + 23.6 \times P_{dep} + ME_{Pdeam} \times (P_{prod} - P_{dep})$
    if $ME_{prod} - P_{dep} \times ME_{Pdep} \geq 0$

$$L_{dep} = \frac{ME_{prod} - P_{dep} \times ME_{P_{dep}}}{ME_{L_{dep}}}$$

$$HP = ME_{maint} + HI_{Pdep} \times P_{dep} + HI_{Ldep} \times L_{dep}$$

else

$$L_{dep} = \frac{ME_{prod} - P_{dep} \times ME_{P_{dep}}}{NE_L}$$

$$HP = ME_{maint} + HI_{Pdep} \times P_{dep} - 39.8 \times L_{dep}$$

end do Deposition

determine ash deposition (ash_{dep})

$P = P + P_{dep}$

$L = L + L_{dep}$

$ash = ash + ash_{dep}$

determine water mass

$BW = P + water + ash + L$

end do

end

Appendix II. Thermoregulatory parameters.

The literature on thermoregulation has been followed here, and entities are expressed in Watt per m^2 body surface area rather than in $kJ.day^{-1}$ per $kg^{0.75}$ metabolic body weight. Watts can be converted to $kJ.day^{-1}$ by multiplying by 86.4. The conversion from body weight (BW, in kg) to body surface area (A, in m^2) is accomplished with the Meeh formula $A = a \times BW^b$. Its parameters have been reported as a = 0.097, b = 0.633 (Brody *et al.*, 1928; cited by Kelley *et al.*, 1973); a = 0.087, b = 0.67 (Lusk, 1928; cited by Mount, 1979); and a = 0.073, b = 0.656 (Kelley *et al.*, 1973). The latter set gives A = 0.57 m^2 for 23 kg BW and A = 1.50 m^2 for 100 kg BW. The former two give 19 to 23 % larger values in that weight range. The appropriate parameters for modern pigs might lead to even smaller values. Although Brody's and Lusk's estimates are grossly outdated, they have been used by practically all the sources quoted in this chapter. To be able to compare results with the literature we have used Brody's values.

T_0

"Extrapolation of the line for non-evaporative heat loss [...] cuts the temperature axis just below the deep-body temperature" (Mount, 1979), which means that T_0, the point where the SHL curves reach their zero value, should be around 39 °C in pigs. The significant aspect of this is that, in pigs, T_{hot} lies at 29 to 32 °C well below this point (Bianca, 1969; Mount, 1979; Figure 6 of this chapter). Because EHL_{hot} is reached when SHL_{hot} is still above zero, SHL_{hot} must be taken into account when establishing HL_{hot}. In species with better possibilities for evaporation there would be no more options for sensible heat loss in the trajectory where EHL_{hot} becomes operative. To allow for a rise in body temperature in hot conditions, T_0 is set to 40.5 °C (Black *et al.*, 1998) when calculating SHL_{hot} in equation (7).

$b_{SHLcold}$

The slope of the SHL_{cold} curve can be measured experimentally because it must equal the slope of HP below T_{LC}. The relation to body composition should work through the thermal insulation of the fully vasoconstricted subcutaneous fatty tissue, $I_{tissue,cold}$.

Tissue insulation accounts for less than half the overall body insulation in livestock. The remainder is made up by insulation of the coat and the air boundary layer surrounding the animal (I_{air}). Although coat insulation can be substantial in cattle and sheep we ignore it here. Blaxter (1989) shows that it comprises only < 5 % of coat plus air insulation in pigs with up to 2 cm deep coats. Alexander (1974) approximated heat loss below T_{LC} (in $W.m^{-2}$) as

$$HL_{cold} = \frac{(T_0 - T_{env}) + I_{air} \times EHL_{cold}}{I_{tissue,cold} + I_{air}} \qquad \text{(A2-1)}$$

Differentiating with respect to T_{env} gives

$$b_{SHLcold} = \frac{-1}{I_{tissue,cold} + I_{air}} \qquad \text{(A2-2)}$$

Alexander (1974) and Hovell *et al.* (1977) report I_{air} values in (clipped) sheep and sows between 0.13 and 0.19 $°C.m^2.W^{-1}$. Black *et al.* (1986) approximate I_{air} in their model in terms of conductance associated with radiation and with free and forced convection as

$$I_{air} = \frac{1}{C_{rad} + C_{free} + C_{forced}} \quad \text{(A2-3)}$$

with $C_{rad} = 4.037 \times 10^{-8} \times (273 + [T_{skin} + T_{env}] / 2)^3$, $C_{free} = 2.64 \times (T_{skin} - T_{env})^{0.25} \times BW^{-0.082}$, and $C_{forced} = 13.4 \times V^{0.6} \times BW^{-0.13}$. The constant 4.037×10^{-8} is the product of the Stefan-Boltzmann constant with a parameter that quantifies emissivity of pig skin and building materials, the constant 273 converts °C to °K, and the other constants are empirical values reported by Bruce and Clark (1979). V denotes air velocity in $m.s^{-1}$.

Appendix III relates $I_{tissue,cold}$ to backfat depth (BF, in mm) as $I_{tissue,cold} = 0.05 + 0.002 \times BF$. During simulation, BF can be derived from body lipid mass as described in Appendix IV.

b_{SHLhot}

The slope of the SHL_{hot} curve involves the thermal insulation value of fully vasodilated subcutaneous fatty tissue. The equivalent of equation (A2-2) is:

$$b_{SHLhot} = \frac{-1}{I_{tissue,hot} + I_{air}} \quad \text{(A2-4)}$$

$I_{tissue,hot}$ is quantified in Appendix III as 0.038 °C.m^2.W^{-1}, independent from fat depth.

$A_{eff,cold}$

The effective body surface area A_{eff} was put together by Bruce and Clark (1979) from the overall area A, minus the area that is in contact with other pigs ($A_{contact}$), plus a proportion of the area that is in contact with the floor (A_{floor}). This assumes that no net heat exchange takes place over $A_{contact}$, and that heat exchange with the floor is dependent on the floor's thermal resistance value, I_{floor}. When $I_{floor} < I_{air}$, the body dissipates more heat per m^2 through floor contact than through the air, so $I_{air} - I_{floor} > 0$ in equation (A2-5): the effective body area is increased; and *vice versa*. This leads to the effective body surface area according to

$$A_{eff} = A - A_{contact} + \frac{I_{air} - I_{floor}}{I_{tissue} + I_{floor}} \times A_{floor} \quad \text{(A2-5)}$$

$A_{contact}$ was approximated by Bruce and Clark (1979) as a function of group size (n_{group}): $A_{contact} = 0.075 \times A \times \frac{2 \times (n_{group} - 1)}{n_{group}}$. This would deal with the effect of huddling in cold conditions, *i.e.* it represents $A_{contact,cold}$ whereas $A_{contact,hot}$ is zero.

Bruce and Clark (1979) set $A_{floor} = 0.2 \times A$, and Black *et al.* (1986) adopt this relation for conditions above T_{LC} ($A_{floor,hot} = 0.2 \times A$ in equation (A2-7)), whereas they halve it for cold conditions: $A_{floor,cold} = 0.1 \times A$ in equation (A2-6).

Bruce and Clark (1979) give a "partly empirical, partly intuitive expression for the floor thermal resistance" as $I_{floor} = I_{floor,45} \times \left(\frac{BW}{45}\right)^{0.33} \times \frac{A_{floor}}{0.2 \times A} \times \sqrt{n_{group}}$, where $I_{floor,45}$ denotes the thermal resistance of the floor material as determined in 45 kg pigs. Black *et al.* (1986) quote values of $0.0 \leq I_{floor,45} \leq 0.5$ °C.m^2.W^{-1} for floor types ranging from steel mesh to straw bedding.

All this leads to the following prediction for effective body surface area in cold conditions:

$$A_{eff,cold} = A \times \left[1 + 0.1 \times \frac{I_{air} - (BW/45)^{0.33} \times 0.5 \times \sqrt{n_{group}} \times I_{floor,45}}{I_{tissue,cold} + (BW/45)^{0.33} \times 0.5 \times \sqrt{n_{group}} \times I_{floor,45}} + -0.075 \times \frac{2 \times (n_{group} - 1)}{n_{group}} \right] \quad \text{(A2-6)}$$

$A_{eff,hot}$

Effective body surface area in thermoneutral and hot conditions follows from equation (A2-5) in a similar way as equation (A2-6), with $A_{floor,hot} = 0.2 \times A$ and $A_{contact,hot} = 0$:

$$A_{eff,hot} = A \times \left[1 + 0.2 \times \frac{I_{air} - (BW/45)^{0.33} \times \sqrt{n_{group}} \times I_{floor,45}}{I_{tissue,hot} + (BW/45)^{0.33} \times \sqrt{n_{group}} \times I_{floor,45}} \right] \quad \text{(A2-7)}$$

EHL_{cold}

Ingram (1964) measured local water loss through the skin of young pigs and concluded that between –5 °C and 10 °C, "heat loss by evaporation from the skin of the trunk" ranges from 4.9 to 7.0 W per m^2 trunk skin area. These figures can be adjusted to a whole-body value by making use of proportional surface areas of trunk and extremities in piglets (Stombaugh and Roller, 1977); the resulting values are 3.7 and 5.3 $W.m^{-2}$. Black *et al.* (1986) model $EHL_{resp,cold}$ values (due to inevitable evaporation through the respiratory tract) of the same magnitude as $EHL_{skin,cold}$ which would suggest a total EHL_{cold} level of twice the above range, *i.e.* from 7 to 11 $W.m^{-2}$.

Bruce and Clark (1979) used results from three other sources to derive a linear regression equation of EHL_{cold} on BW. Including Ingram's values this relation becomes

$$EHL_{cold} = A \times (7.4 + 0.089 \times BW) \quad \text{(A2-8)}$$

(in W), with an $R^2 = 0.96$.

EHL_{hot}

The operational value of EHL_{hot} is set by the fact that, in pigs, T_{hot} is attained before the SHL curves attain their zero value (see the above section on *T_0*). In dry conditions, hyperthermia may occur even when there is still reasonable scope for sensible heat loss. This creates a practical upper limit to EHL: EHL_{hot} is equivalent to the EHL value at T_{UC}.

In the above cited experiment of Ingram (1964), heat loss by evaporation at 30 °C with additional infra-red radiation heating was around 23.4 W per m^2 trunk skin area. The adjusted whole-body value (see the above section on *EHL_{cold}*) is 17.8 $W.m^{-2}$. Black *et al.* (1986) model $EHL_{resp,hot}$ values (due to panting) twice as large as $EHL_{skin,hot}$ which would suggest an EHL_{hot} level of three times the above value, *i.e.* 53 $W.m^{-2}$.

Bruce (1981) compiled results from five other sources and concluded that total EHL_{hot} per unit of body surface area is BW-independent in a range from 2 to 91 kg. But some of the animals in those studies were clearly not at their T_{UC} level, so their actual EHL must have

been lower than EHL_{hot}. When we include Ingram's values and omit the records below T_{UC} we get $EHL_{hot} = 57.3 - 0.238 \times BW$ (in $W.m^{-2}$); the standard error of the slope is 0.019. Black *et al.* (1986) fitted a non-linear model to Bruce's data; refitting that model to the updated records gave a similar fit ($R^2 = 0.98$) as the above linear regression. Thus, to stay with Black's approach,

$$EHL_{hot} = A \times (12.2 + 110.8 \times BW^{-0.33}) \qquad \text{(A2-9)}$$

So far, the pig has been assumed to be completely dry. When pigs have the option to keep themselves wet by wallowing in water, mud or manure, the evaporation of water from the skin can generate a substantial temporary heat loss (Bligh, 1973; Ingram, 1974). Black *et al.* (1986) put together information from Bruce (1981) to calculate the rate of heat loss (in $W.m^{-2}$) through evaporation from wet skin, to be added to EHL_{hot} from equation (A2-9), as

$$EHL_{wet} = A_{wet} \times 45.4 \times V^{0.6} \times BW^{-0.13} \times (0.0461 - W_{air}) \qquad \text{(A2-10)}$$

The term $[45.4 \times V^{0.6} \times BW^{-0.13}]$ derives from the forced convection term in equation (A2-3), the constant 0.0461 $kg.kg^{-1}$ denotes the air water vapour content at 39 °C and 100 % relative humidity, and W_{air} denotes the air water content (in $kg.kg^{-1}$) at the prevailing temperature and relative humidity at the skin surface. Black's model uses a fixed value of 90 % for the latter parameter. The authors state that "in commercial practice, it is rare to find a completely dry pig in a hot environment. Even pigs kept on slatted floors are usually able to wet some skin with drinking water or urine". Mount *et al.* (1971), Straub *et al.*(1976), Giles *et al.* (1988) and Nienaber *et al.* (1996) provide support for this statement, but do not present any quantification. Although Black *et al.* (1998) mention that "the proportion of skin that becomes wet under different management conditions has not been defined accurately", the model by Black *et al.* (1986) assumes "that the pig is able to [voluntarily] wet up to 15 % of its skin by these means", *i.e.* $A_{wet} \leq 0.15 \times A$, or alternatively that the whole body surface area not in contact with the floor is wet by spray-cooling, *i.e.* $A_{wet} = A - A_{floor}$ in terms of equation (A2-5).

Appendix III. Tissue insulation.

Equation (9) leaves the term $\frac{d(I_{tissue})}{d(FAT)}$ to be described. To do so, fat depth and tissue insulation must be measured. The latter can be estimated by measuring T_{body}, T_{skin} and heat loss (usually in terms of HP) and combining these as

$$I_{tissue} = \frac{T_{body} - T_{skin}}{HP} \qquad \text{(A3-1)}$$

Tissue insulation values determined by equation (A3-1) could be related to subcutaneous backfat depth (BF) and body weight by making use of experimental results published by Holmes and McLean (1974), Stombaugh and Roller (1977; BF of these 6 to 12 kg piglets assumed to be about 3 mm based on data by Tullis, 1981), Verstegen and Curtis (1988; BF data made available by M.W.A. Verstegen, personal communication, 1996), Henken *et al.* (1991), and Van der Hel *et al.* (1997; BF data made available by W. van der Hel and H.A. Brandsma, personal communication, 1996).

Tissue insulation can be expected to be higher in larger animals, simply because these have more peripheral tissue available to act as a body shell. Bruce and Clark (1979) described cold whole-body tissue insulation of pigs as a function of body weight raised to the power 1/3 (approaching a linear dimension): $I_{tissue,cold} = 0.02 \times BW^{0.33}$. However, we aim at relating tissue insulation to body composition, more specifically to subcutaneous fat depth (equation (9)). Therefore the data from the above sources were analysed with the regression model

$$I_{tissue} = b_0 + b_1 \times BF + b_2 \times BW^{0.33} \qquad \text{(A3-2)}$$

I_{tissue} levels were either adopted directly as reported, or calculated from the reported T_{body}, T_{skin} and HP values according to equation (A3-1). HP of the sows that Verstegen and Curtis (1988) fed close to maintenance was estimated (by them) from the assumed maintenance requirements and the observed ME intake. HP was determined from respiratory exchange in all other studies.

We have adjusted BF for the site of measurement making use of the diagrams by Kaufmann and StClair (1965), and performed a stepwise regression with weighting according to the numbers of animals in each experiment (as given in Figure 7). The results showed a non-significant ($P \approx 0.94$) fit of model (A3-2) for

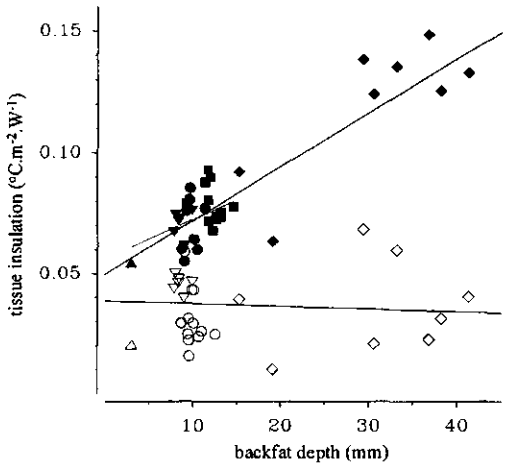

Figure 7 Tissue insulation in relation to backfat depth in cold (solid symbols) and hot (open symbols) conditions. Data from Stombaugh and Roller (1977; 8 piglets: ▲); Henken *et al.* (1991; 6 groups of 16 growing pigs: ▼); Van der Hel *et al.* (1997; 2 groups of 8 growing pigs measured several times: ●); Verstegen and Curtis (1988; 12 groups of 2 to 10 sows: ■); Holmes and McLean (1974; 4 sows each measured twice: ◆). See the **Discussion** section for the short trendline through the leftmost cold data points.

data obtained at T_{env} well above the T_{LC} values reported in these studies (*i.e.*, where vasodilation may be assumed). Hence, neither $BW^{0.33}$ nor BF influence $I_{tissue,hot}$ to any significant extent. For data from $T_{env} < T_{LC}$ (*i.e.*, where complete vasoconstriction may be assumed), the stepwise procedure reported a partial $R^2 = 0.60$ ($P < 0.0001$) for BF (entered as the first regressor) and a partial $R^2 = 0.008$ ($P = 0.43$) for $BW^{0.33}$ as the second regressor when estimating $I_{tissue,cold}$. The correlations of the b_1 and b_2 estimates with the b_0 estimate were +0.28 and –0.90, respectively. An analysis with the non-linear regression model

$$I_{tissue} = b_0 + b_1 \times BF + b_2 \times BW^{b3} \qquad \text{(A3-3)}$$

did not converge for values of $0 \leq b_3 \leq 1$, and produced similar b_1 estimates as model (A3-2). It may be concluded from the above that $BW^{0.33}$ can safely be omitted from the regression model, which leaves us to describe $I_{tissue,cold}$ in terms of BF only.

Figure 7 shows two scatter plots, relating $I_{tissue,cold}$ and $I_{tissue,hot}$ (in $°C.m^2.W^{-1}$) to BF (in mm). As above, $I_{tissue,hot}$ does not depend on BF: the regression is $I_{tissue,hot} = 0.038 - 0.0001 \times BF$, with standard errors of 0.0056 (for the intercept) and 0.0005 (for the slope). In contrast, $I_{tissue,cold}$ is increased by 0.002 $°C.m^2.W^{-1}$ per mm increase in BF: $I_{tissue,cold} = 0.050 + 0.0022 \times BF$, with standard errors of 0.0036 (intercept) and 0.00031 (slope).
As would be expected, the regression intercepts (the insulation values of the peripheral tissue at zero BF) are close together, although they differ significantly from each other ($P \approx 0.03$).

Appendix IV. Partitioning of body lipid over lipid depots, and derivation of (sub)cutaneous fat depth.

Body lipid partitioning

Tullis (1981) lists in the appendices of her PhD thesis 19 individual observations on the size of the various lipid depots measured in pigs with a total body lipid mass up to 37 kg, the more or less arbitrary range that is considered here. The individual data behind the results published by Jørgensen *et al.* (1985) and by Susenbeth and Keitel (1988) were made available by H.H. Jørgensen (personal communication, 1991; n = 207) and A.J.J. Susenbeth (personal communication, 1992; n = 35). The percentages of body lipid mass in the (sub)cutaneous, muscle (intra- and intermuscular lipid), entrails, and bone depots were regressed on the natural logarithm of total body lipid mass. Likewise, the percentage of body protein mass in the (sub)cutaneous pool was regressed on the natural logarithm of total body protein mass. This pool had been included in the "connective tissue protein" pool in Chapter 1. The estimated regression coefficients are in Table 3.

Table 3 Regression coefficients (% per ln[kg]) of percentage of body lipid in various lipid depots on log total body lipid mass, and of percentage of body protein in the (sub)cutaneous pool on log total body protein mass

	source[†]		
Lipid depot	Tullis	Jørgensen	Susenbeth
(Sub)cutaneous	+6.47	+20.31	+10.10
Muscle	–5.52	–12.35	–4.60
Entrails	+0.83	–0.76	–0.24
Bone	–1.88	–7.17	–5.25
(Sub)cutaneous protein	+0.97	+0.039	

[†] Tullis (1981); Jørgensen *et al.*(1985); Susenbeth and Keitel (1988)

The proportion of body lipid that is present in the entrails shows little change when total body lipid mass increases. It is the only pool for which both negative and positive regression coefficients are reported, and these are all close to zero. The (sub)cutaneous depot seems to complement the muscle and bone depots, although the exact course with increasing total body lipid varies greatly.

We have pooled the data from these sources together and performed an overall regression analysis similar to the one in Chapter 1. The results are in Table 4.

The genotypes covered by these references vary widely with respect to growth and body composition traits. Correspondingly, the differences between the regression parameters are sometimes quite large. It may be critical to determine these parameters specifically for the pig population to be simulated. The values in Table 4 should be seen as generalisations, serving solely for parameterisation of this general study.

Table 4 Overall regression coefficients (% per ln[kg]) of percentage of body lipid in various lipid depots on log total body lipid mass, and of percentage of body protein in the (sub)-cutaneous pool on log total body protein mass

Lipid depot	intercept	regression
(Sub)cutaneous	22.60	+13.10
Muscle	49.69	–7.49
Entrails	8.98	–0.25
Bone	18.94	–5.37
(Sub)cutaneous protein	12.85	+0.286

Fat depth

Average whole-body (sub)cutaneous fat depth (FAT, in mm) can be derived from body weight (BW, in kg) and from the (sub)cutaneous tissue's (SCT) weight (SCTW, in kg) and its volume density (ρ, in $kg.dm^{-3}$). This requires transformation of SCTW into SCT volume by division through ρ, and division of this volume through body surface area ($A = 0.097 \times BW^{0.633}$).

The density of the (sub)cutaneous tissue can be calculated according to Chato (1985) as

$$\rho\ (kg.dm^{-3}) = [m_{water} + 0.649 \times m_{protein} + 1.227 \times m_{lipid}]^{-1} \qquad \text{(A4-1)}$$

The parameters $m_{...}$ represent the water, lipid and protein mass fractions of the SCT, in $kg.kg^{-1}$: $m_{protein} = \frac{\text{SCT protein mass}}{\text{SCTW}}$ etc. The numerator terms [SCT protein mass] and [SCT lipid mass] equal $p_{P,SCT} \times P$ and $p_{L,SCT} \times L$, respectively, with $p_{P,SCT}$ and $p_{L,SCT}$ denoting the proportions of body protein mass (P) and of body lipid mass (L) present in the SCT. These proportions follow from the regression coefficients in Table 4. Hence we can write $m_{protein} = \frac{p_{P,SCT} \times P}{SCTW}$, and $m_{lipid} = \frac{p_{L,SCT} \times L}{SCTW}$. SCT water mass can be expressed as a multiple of SCT protein mass (water mass = 2.727 × protein mass in the SCT data of Jørgensen *et al.*, 1985; $R^2 = 0.62$), so that $m_{water} = \frac{2.727 \times p_{P,SCT} \times P}{SCTW}$. Substituting into equation (A4-1) gives:

$$\rho = \frac{SCTW}{3.376 \times p_{P,SCT} \times P + 1.227 \times p_{L,SCT} \times L} \qquad \text{(A4-2)}$$

When deriving the SCT volume from SCTW / ρ, the numerator term in (A4-2) cancels out, so that whole-body (sub)cutaneous tissue depth is finally estimated as

$$FAT = \frac{SCTW}{A \times \rho} = \frac{3.376 \times p_{P,SCT} \times P + 1.227 \times p_{L,SCT} \times L}{0.097 \times BW^{0.633}} \qquad \text{(A4-3)}$$

with $p_{P,SCT} = 0.1285 + 0.00286 \times \ln(P)$, and $p_{L,SCT} = 0.2260 + 0.1310 \times \ln(L)$.

To obtain a model predictor for tissue insulation that can actually be parameterised we have to deal, at this stage, with an anomaly. Of course, whole-body tissue insulation depends on whole-body fat cover. But parameterisation of equation (9) is only possible on the basis of backfat depth (BF) values, as these appear to be the only subcutaneous fat depth readings that have been measured (and published) in connection with I_{tissue} up to now. So we need to make a detour, and derive BF from FAT.

Regressing the 42 P2 backfat depth readings by Tullis (1981) on the corresponding FAT values (estimated according to equation (A4-3)) resulted in a power function according to $BF = 0.82 \times (FAT)^{1.212}$, with an R^2=0.93. This equation, when combined with the above one for FAT (A4-3), yields P2 backfat depth values increasing from 4 to 18 mm in a pig that grows at a normal pattern from 25 to 100 kg BW; this is about the same range as described by Tullis's (1981) relation of P2 with L according to $BF = (-0.56 + 3.89 \times L)^{0.676}$. Black *et al.* (1986) relate P2 to the body lipid mass fraction as $BF = -0.95 + 75 \times L / BW$, increasing from 5 to 16 mm over the same BW range.

Appendix V. Dimensions and meaning of parameter abbreviations used in Chapter 3.

parameter	dimension	meaning	parameter	dimension	meaning
α	$kJ.kg^{-0.75}.d^{-1}$	metabolic rate parameter	L	kg	body lipid mass
ρ	$kg.dm^{-1}$	subcutaneous tissue density	L_{dep}	$kg.d^{-1}$	lipid deposition rate
A	m^2	body surface area	ME_{Ldep}	$MJ.kg^{-1}$	lipid deposition ME requirement
$A_{contact}$	m^2	A in contact with other pigs	ME_{maint}	$MJ.d^{-1}$ / $kJ.kg^{-0.75}.d^{-1}$	maintenance ME requirement
A_{eff}	m^2	effective body surface area			
A_{floor}	m^2	*A in contact with the floor*	ME_{Pdep}	$MJ.kg^{-1}$	*protein deposition ME requirement*
A_{wet}	m^2	wet body surface area	ME_{req}	$MJ.d^{-1}$	required ME intake
BF	mm	backfat depth	m_{lipid}	$kg.kg^{-1}$	lipid mass fraction of SCT
B_{Gomp}	$kg.d^{-1}.kg^{-1}$	rate parameter of protein growth	$m_{protein}$	$kg.kg^{-1}$	protein mass fraction of SCT
b_{SHL}	$W.m^{-2}.°C^{-1}$	regression of SHL on T_{env}	m_{water}	$kg.kg^{-1}$	water mass fraction of SCT
BW	kg	body weight	NE_L	$MJ.kg^{-1}$	lipid NE content
ConstrFI	$kg.d^{-1}$	constrained feed intake	NE_P	$MJ.kg^{-1}$	protein NE content
CP	$kg.kg^{-1}$	feed CP content	n_{group}	1	group size
DE	$MJ.kg^{-1}$	feed DE content	nHP	same as HP	thermoneutral HP
dFI	$kg.d^{-1}$	desired FI	P	kg	body protein mass
dL_{dep}	$kg.d^{-1}$	desired L_{dep}	P_∞	kg	mature body protein mass
dP_{dep}	$kg.d^{-1}$	desired P_{dep}	P_{dep}	$kg.d^{-1}$	protein deposition rate
EHL	same as HL	evaporative heat loss	$p_{L,SCT}$	$kg.kg^{-1}$	proportion of L in SCT
ETH	same as HP	required "cold" increase in HP	$p_{P,SCT}$	$kg.kg^{-1}$	proportion of P in SCT
FAT	mm	average subcutaneous fat depth	RHP	same as HP	required "hot" reduction in HP
FI	$kg.d^{-1}$	feed intake	$R_{L\infty/P\infty}$	$kg.kg^{-1}$	ratio of mature L to P
HI_{Ldep}	$MJ.kg^{-1}$	lipid deposition heat increment	SCT	–	subcutaneous tissue
HI_{Pdep}	$MJ.kg^{-1}$	protein deposition heat increment	SCTW	kg	subcutaneous tissue weight
			SHL	same as HL	sensible heat loss
HL	$MJ.d^{-1}$ / $kJ.kg^{-0.75}.d^{-1}$ / $W.m^{-2}$	heat loss	T_0	°C	T_{env} level where SHL=0
			T_{body}	°C	body temperature
			T_{EC}	°C	evaporative critical temperature
HP	$MJ.d^{-1}$ / $kJ.kg^{-0.75}.d^{-1}$ / $W.m^{-2}$	heat production	T_{env}	°C	environmental temperature
			T_{LC}	°C	lower critical temperature
I_{air}	$°C.m^2.W^{-1}$	air boundary layer insulation	T_{skin}	°C	skin temperature
I_{tissue}	$°C.m^2.W^{-1}$	tissue insulation	T_{UC}	°C	upper critical temperature
k_L	$MJ.MJ^{-1}$	lipid deposition net efficiency	V	$m.s^{-1}$	air velocity
k_P	$MJ.MJ^{-1}$	protein deposition net efficiency	W_{air}	$kg.kg^{-1}$	air water content

The comparative approach [...] should confirm [...] the old adage that all animals are equal – once one has accounted for all the differences.

A.J.F. Webster (1988) in *Comparative nutrition* (Eds. K. Blaxter & I. MacDonald). Libbey, London; p. 51.

Chapter 4 is based on

P.W. Knap (2000) Stochastic simulation of growth in pigs: relations between body composition and maintenance requirements as mediated through protein turnover and thermoregulation. *Animal Science* **71**(1):000-000

Chapter 4

Protein turnover- and thermoregulation-dependent relations with body composition

A model for simulation of growth in pigs, extended to describe thermoregulatory processes, was made stochastic to simulate groups of pigs with between-animal variation in mature body protein (P_∞) and lipid mass (L_∞), in the potential rate at which mature mass is attained (B*), and in the distribution of body protein and lipid over pools and depots. The resulting variation in body composition leads to variation in energy requirements for protein turnover and thermoregulation, causing between-animal variation in maintenance requirements (ME_{maint}).

Simulated population means for P_∞, L_∞/P_∞ and B* were varied in 3 steps each. Excluding unrealistic parameter combinations this led to $3^3 - 6 = 21$ simulated genotypes. Simulated within-population coefficients of variation (CV) were 7, 15 and 3 %. Random replicates of each genotype were simulated five times, in climatic conditions that were subsequently severely cold, mildly cold (about 5 and 1 °C below lower critical temperature), thermoneutral, mildly hot and severely hot (about 1 and 5 °C above upper critical temperature), during the entire growth period of 23 to 100 kg liveweight. Simulated feed intake was ad libitum.

Simulated thermoneutral within-population standard deviations of body protein and lipid content were 0.21–0.46 kg and 0.78–2.14 kg at 100 kg body weight. On average, the corresponding values in cold and hot conditions were slightly higher.

ME_{maint} showed a protein-turnover-related within-population CV of 1.5 % at thermoneutrality. Thermoregulatory action contributed about 4 % extra variance in cold and hot conditions, but CV values were not affected. A genetic increase in the maximum protein deposition rate from 100 to 250 $g.d^{-1}$ would increase ME_{maint} as related to protein turnover and thermoregulation by 11 % at thermoneutrality, and by 6–11 % in cold or hot conditions.

Two relevant groups of genotypes could be distinguished based on the within-population regression coefficients of ME_{maint} on daily or cumulative protein deposition ($b_{dailyPdep}$, $b_{cumPdep}$). These ranged from 0.250 to 0.428 $kJ.kg^{-0.75}.d^{-1}$ per $g.d^{-1}$, and from 2.77 to 5.45 $kJ.kg^{-0.75}.d^{-1}$ per kg, respectively, in 12 "conventional" genotypes at thermoneutrality. On average, $b_{dailyPdep}$ was increased by 48 %, 20 %, –11 % and –36 % in the other climatic conditions mentioned above, respectively. The corresponding increase of $b_{cumPdep}$ was 32 %, 14 %, 8 % and 48 %. Three fast-growing lean genotypes showed similar $b_{dailyPdep}$ and $b_{cumPdep}$ at thermoneutrality, but much more pronounced increases in cold and hot conditions: 137 %, 49 %, –12 % and +88 % for $b_{dailyPdep}$ and 248 %, 108 %, 17 % and 196 % for $b_{cumPdep}$.

It is concluded that differences in body composition traits between pig genotypes do not cause important between-genotype differences in thermoregulatory ME_{maint}, and that thermoregulatory processes contribute little body-composition-related variation to hot or cold ME_{maint} within most genotypes.

The inferences to be made from this with regard to experimental design are discussed. The verification of the above predictions will require a very elaborate and large-scaled experiment.

Introduction

The algorithm described by Emmans (1988, 1997) for simulation of the protein and energy metabolism and feed intake of growing pigs was extended in Chapter 3 to make explicit the ME requirements for thermoregulation. These requirements depend on body composition-related traits in two ways. First, faster rates of protein and/or lipid deposition lead to a higher heat production (HP). Second, tissue thermal insulation in cold conditions depends on subcutaneous fat depth, and hence on total body lipid mass and the proportion of this present in the subcutaneous depot. All these traits show variation among animals of equal body weight. Therefore, the ME requirements for thermoregulation will vary among animals simply as a result of variation in body composition-related traits. When these ME requirements are regarded as part of the energy requirements for body maintenance (ME_{maint}), it follows that ME_{maint} will show thermoregulation-related variation among animals as a result of variation in body composition.

This variation in cold or hot conditions comes in addition to the protein-turnover-related variation in ME_{maint} that was shown in Chapter 2 to be the result of variation in protein deposition traits. The simulation model extended in Chapter 3 was used in the present study for stochastic simulation of growing pigs with variable body composition and hence variable ME_{maint}, and to quantify the variation in ME_{maint}. Parameter abbreviations are in Appendix II.

Simulation methods

Model

The model described in Chapter 3 was based on Emmans's (1988) potential growth rules, characterising the "growing pig" genotype with three parameters: mature body protein and lipid mass (P_∞ and L_∞, both in kg) and a rate parameter (B_{Gomp}, in $kg.d^{-1}.kg^{-1}$) that regulates the growth of both portions. Body protein and body lipid mass potential are supposed to increase according to Gompertz functions of age with different asymptotes (P_∞, L_∞) but with the same rate parameter (B_{Gomp}), which assumes full allometry between the two portions. This pattern leads to a potential rate of protein deposition that varies with the stage of development, starting at a low value and increasing to a maximum level that is roughly attained between 40 and 90 kg body weight, with a subsequent gradual decline towards zero when maturity is attained. Environmental factors (*e.g.* inadequate nutrition) may cause the simulated phenotype to deviate from the genotype's potential.

Because "larger animals will have a lower growth rate relative to body size" (Ferguson *et al.*, 1997), B_{Gomp} and P_∞ are negatively correlated. This makes it difficult for a stochastic study to impose variation on each of them, which can be solved (Emmans, 1988; p. 164) by scaling B_{Gomp} by the 0.27 power of P_∞ according to Taylor (1985). The "scaled rate parameter" $B^* = B_{Gomp} \times P_\infty^{0.27}$ is theoretically uncorrelated to P_∞ (Emmans and Fisher, 1986, who make use of the genetic size scaling rules of Taylor, 1985). For convenience, L_∞ is expressed as its ratio to P_∞ ($R_{L\infty/P\infty}$, in $kg.kg^{-1}$), which is assumed to be uncorrelated to both P_∞ and B^*.

Ad libitum feed intake is predicted in this model as the intake required to satisfy both the protein and energy needs of the potential growth thus described plus the maintenance requirements (Emmans, 1997), subject to capacity constraints to feed intake volume (Ferguson *et al.*, 1994). In Chapter 3 we embedded these growth and feed intake rules into the version of the nutrient

partitioning model by Moughan and Smith (1984) that was extended in Chapter 1, where the growth rules replace Whittemore and Fawcett's (1974) linear-plateau model of protein deposition, and the feed intake rules allow for an evaluation of the system without constraints of scale feeding or suchlike. In Chapter 3 we also added a module to predict metabolic changes due to cold or hot conditions. In this module, cold thermoregulatory action includes an increase of *ad libitum* feed intake. Hot thermoregulatory action includes reduction of physical activity, increase of body temperature, wetting of a proportion of the skin, and reduction of *ad libitum* feed intake.

Simulation runs

This model was evaluated in two settings for two different purposes, as detailed below. In each of these runs, the three genotype parameters were varied over a proportional range of their reference values, as follows: P_∞ = [0.8, 1.0, 1.2] × 32.5 kg, $R_{L\infty/P\infty}$ = [0.4, 1.0, 1.6] × 3.2 $kg.kg^{-1}$ and B* = [0.6, 1.0, 1.4] × 0.0323 $kg.d^{-1}.kg^{-0.73}$. The B* value 0.0323 is 0.0126 × $(32.5)^{0.27}$. The reference values 32.5, 3.20 and 0.0126, and the associated proportional ranges, follow from Chapter 6. Protein and lipid growth data of five pig genotypes from between 1969 and 1993 were analysed there, which lead to estimates of P_∞, $R_{L\infty/P\infty}$ and B_{Gomp} that roughly encompassed the above ranges. We adopt these figures here to ensure that our simulation results are appropriate for the western pig genotypes represented in the contemporary literature. This would generate $3^3 = 27$ simulated genotypes, each represented by a [P_∞ × $R_{L\infty/P\infty}$ × B*] combination. However, based on the findings of Chapter 6, the combination of high population means of $R_{L\infty/P\infty}$ and B*, and *vice versa*, is unlikely to be found in real life. Hence the associated six genotypes were not generated, leading to 21 simulated genotypes.

The diet was set to contain 13.26 MJ DE and 0.198 kg CP per kg feed up to 50 kg body weight (BW). From that point onwards the CP content was set to 0.178 $kg.kg^{-1}$. These CP levels had been found to be adequate to support the growth potential of the simulated genotypes in preliminary runs; it had been decided at an earlier stage to abandon the idea of a continuously optimised diet composition for each combination of genotype, environment and BW in order to keep the simulation manageable. The amino acid pattern of the feed protein was according to the "high quality" protein in table 2 of Chapter 2 (0.0143 g lysine per g feed, etc.). *Ad libitum* feed intake (as specified in the *Model* section above: the amount of feed that satisfies both the energy and the protein requirements of potential growth plus maintenance) was constrained to about 4.2 times thermoneutral maintenance requirements. Group size was set to six pigs, which in this model is relevant for cold thermoregulation only. All other model parameters were set to the levels used in the sensitivity analysis of Chapter 3; the simulated growth trajectory was from 23 to 100 kg BW, air velocity was 0.15 $m.s^{-1}$, relative humidity 0.7, floor thermal resistance 0.07 $°C.m^2.W^{-1}$ to represent concrete slats, and the maintenance ME requirements independent from protein turnover were set at 0.65 × (495 − 0.75 × BW) $kJ.kg^{-0.75}.d^{-1}$ (see below).

Simulation runs: thermoneutral variation

The model was run with the thermoregulatory routines deactivated, evaluating each genotype in thermoneutral conditions during its entire growth trajectory. For each of the 21 genotypes, 500 random replicates (i.e. individual pigs) were generated. The model had been made stochas-

tic by generating variation in three sets of model parameters, as follows. All the stochastic parameters used here have been summarised in Table 1.

First, variation in P_∞, $R_{L\infty/P\infty}$, and B*, following the procedure described in Chapter 2: random deviates for each parameter were obtained for each replicate as $dev_{P\infty} = \mu_{P\infty} + rannor \times \sigma_{P\infty}$ and analogously for the other parameters; rannor denotes a random drawing from the standard Normal distribution (SAS, 1990a). The coefficients of variation were initially taken from Ferguson *et al.* (1997), who proposed likely values of $\sigma_{P\infty} = 0.05 \times \mu_{P\infty}$, $\sigma_{RL\infty/P\infty} = 0.10 \times \mu_{RL\infty/P\infty}$, and $\sigma_{B^*} = 0.01 \times \mu_{B^*}$ ($\mu_{P\infty}$ etc. denote the genotype means, and $\sigma_{P\infty}$ etc. denote the within-genotype standard deviations). These coefficients of variation were subsequently adjusted by iterative re-evaluation of the model as described in Appendix I. Random B_{Gomp} deviates were derived from the B* and P_∞ deviates as $dev_{BGomp} = dev_{B^*} / (dev_{P\infty})^{0.27}$.

Table 1 Parameters describing the stochastic processes in the simulations

	trait†	coefficient of variation	standard deviation (%)	r_{45}‡
1	P_∞	0.07		
2	$R_{L\infty/P\infty}$	0.15		
3	B*	0.03		
4	P_{mus}		2.44	–0.838
5	P_{con}		1.90	
6	L_{sub}		3.78	

† P_{mus}, P_{con}: percentage of body protein mass present in the muscle and connective tissue protein pools.
L_{sub}: percentage of body lipid mass present in the (sub)cutaneous lipid depot.
‡ correlation between traits 4 and 5.

Second, variation in the proportions of body protein present in the muscle and connective tissue pools, which is relevant for the simulation of protein turnover. The model distinguishes various body protein pools with different turnover rates, the partitioning of which varies between animals (see Chapter 5). The base levels of these two proportions ($FrcP_1$, $FrcP_2$) were derived as in Chapter 2 from the prevailing body protein mass (P) as $FrcP_i = a_i + b_i \times \ln(P)$. Deviates for $FrcP_1$ and $FrcP_2$ were then created (and correlated to each other) by adding the terms $rannor_1 \times \sigma_1$ and $(rannor_1 \times r_{12} + rannor_2 \times \sqrt{1 - r_{12}^2}) \times \sigma_2$, respectively.

Third, variation in the proportion of body lipid present in the (sub)cutaneous depot, which is relevant for the simulation of thermal insulation, calculated as above for protein proportion $FrcP_1$.

The relevant output of this simulation consists of the genotype means and within-genotype standard deviations of model output traits like cumulative protein deposition from 23 to 100 kg BW ($cumP_{dep}$, in kg), average daily gain in body weight (ADG, in $g.d^{-1}$) and average daily feed intake (DFI, in $g.d^{-1}$). The results from these simulations would be expected to coincide to a large extent with the results of the earlier simulations in Chapter 2 with the model of Moughan and Smith (1984) which was based on the nutrient partitioning rules by Whittemore and Fawcett (1974).

Simulation runs: cold, thermoneutral and hot variation

The simulation described in the previous section was repeated, evaluating 500 random replicates of each genotype at each of five T_{env} levels: about 5 and 1 degrees Celsius below T_{LC}, thermoneutral conditions (as above), and about 1 and 5 deg above T_{UC}. Hence each replicate was evaluated five times, in conditions that were subsequently severely and mildly cold, thermoneutral, and mildly and severely hot during its entire growth trajectory.
The relevant output of this simulation consists of the genotype means and within-genotype standard deviations of predicted traits like ME intake, daily and cumulative protein and lipid deposition, and the maintenance requirement and its protein turnover- and thermoregulation-related components.

This output is highly multi-dimensional, and its full presentation would lead to a very confusing series of tables and graphs. Preliminary graphical analyses of the simulation results (Chambers *et al.*, 1983) revealed that the relation of predicted ME_{maint} with daily and cumulative protein deposition captures most of the variation of interest. Hence these relations were further statistically analysed within each combination of the 21 genotypes and five climatic conditions with the regression model

$$\begin{aligned} ME_{maint} &= \mu + \Sigma dailyP_{dep} + b_{cumPdep} \times cumP_{dep} \\ ME_{maint} &= \mu + \Sigma cumP_{dep} + b_{dailyPdep} \times dailyP_{dep} \end{aligned} \qquad (1)$$

where μ denotes the overall mean, and $b_{cumPdep}$ and $b_{dailyPdep}$ are linear regression coefficients. The variable $\Sigma cumP_{dep}$ (with 100 classes) was created by collapsing the sorted $cumP_{dep}$ variable into 100 groups of five adjacent realisations, and calculating the group averages; $\Sigma dailyP_{dep}$ was created similarly. In a conventional bivariate regression analysis with two continuous independent variables (such as $ME_{maint} = \mu + b_{dailyPdep} \times dailyP_{dep} + b_{cumPdep} \times cumP_{dep}$), the estimates of the regression coefficients (*i.e.* $b_{cumPdep}$ and $b_{dailyPdep}$) are usually prone to sampling covariance, which in this case is further augmented by the functional covariance between the two independent variables. This is no problem when the analysis is only used to predict ME_{maint} from cumulative and daily protein deposition, but it makes the interpretation of the regression coefficients independent from each other rather meaningless because the estimates are confounded. And it is this separate interpretation that must be accomplished here: we are interested in the linear relation of ME_{maint} with either cumulative or daily protein deposition, independent from the possible effects of the other variable.
The mixed (continuous–discrete) analysis in model (1) allows for the quantification of this relation of ME_{maint} with either of these variables while controlling for the effects of the other one without making any, possibly confounding, assumptions on the nature (linear or otherwise) of its relation to ME_{maint}. A similar approach was followed by Luiting and Urff (1991; p. 328).

The adequacy of the model used in this study, in terms of its realistic prediction of (i) thermoregulatory action and (ii) within-genotype variability of output traits, can be judged by (i) the simulation results described in the section on *Variation between genotypes* below, and (ii) the comparison of the predicted within-genotype variation of growth rate, daily feed intake and cumulative protein deposition with real-life data as in Appendix I. The genotype parameter

means derived from Chapter 6 should ensure that the model adequately predicts the metabolic behaviour of the range of pig genotypes currently available in the western world. Having accepted all that, it is relevant to draw inference from the model output that cannot be compared to real-life data. This is dealt with in the section on *Variation within genotypes* below.

Simulation results

The results of these simulations are complicated to describe. The express intention of the study is to quantify variation in output variables, and the more interesting results among these are relations between variables (co-variation). Hence the ultimate subject of interest is within-genotype regression coefficients, which call for a high level of abstraction.

The two simulated genotypes with the highest proportional values for both $\mu_{P\infty}$ and $\mu_{RL\infty/P\infty}$ failed to complete any replicates in the more severely cold and hot conditions. These simulated pigs lost so much lipid (mainly at the lower and higher body weights in cold and hot conditions, respectively) that their simulation had to be terminated, either because of negative body lipid mass or fat depth, or because the replicate needed more than 300 days to reach end weight. Hence the simulated data set holds 21 genotypes, but only 19 of these provide meaningful data at 5 °C below T_{LC} or above T_{UC}; we will further refer to "the 21 simulated genotypes" in this sense. These "lost" records do not result from a model anomaly, they just suggest that those genotypes will grow very slowly in such conditions, or die because they reach their hypo- or hyperthermal points.

Thermoneutral variation

The simulated values of B* and P_∞ result in genotype averages for B_{Gomp} that range from 0.0072 to 0.0188 $kg.d^{-1}.kg^{-1}$, close to the intended values (see Chapter 6). All its associated coefficients of variation are around 0.034 (again, as intended). This range for B_{Gomp}, combined with the simulated P_∞ extremes, results in genotype means of the maximum potential rate of protein deposition ($P_{dep,max} = B_{Gomp} \times P_\infty / e$, according to Ferguson and Gous, 1993a) ranging from 77 to 241 $g.d^{-1}$.

The 21 simulated combinations of $\mu_{P\infty}$, $\mu_{RL\infty/P\infty}$ and μ_{B^*} result in between-genotype variation in the average predicted values for $cumP_{dep}$, $cumL_{dep}$ (cumulative lipid deposition) and ME_{maint}. These values can be compared with the simulation results of Chapter 2, which were due to combinations of seven levels (100, 125, ..., 225, 250 $g.d^{-1}$) of the genotype parameter $\mu_{Pdep,max}$ with seven scaled feeding levels, the latter according to [0.6, 0.7, ..., 1.1, 1.2] times an empirical function that related *ad libitum* feed intake of a specific pig genotype to BW. When the genotype means of DFI from the current simulations were related to that same function they evaluated to levels between 0.4 and 1.1. The current ranges of cumulative protein and lipid deposition and of ME_{maint} are similar to the associated ranges up to the 1.1 feeding level in tables 3 and 4 of Chapter 2. This agrees with the range for $\mu_{Pdep,max}$ derived in the previous paragraph. Hence the current 21 simulated genotypes encompass a slightly wider range of genotypes than the earlier 49 $P_{dep,max}$-based ones.

The simulated $\sigma_{P\infty}$, $\sigma_{RL\infty/P\infty}$ and σ_{B^*} result in within-genotype variation in cumulative protein and lipid deposition and in ME_{maint}. Again, the associated predicted within-genotype

standard deviations are similar to the corresponding values from Chapter 2.

Cold, thermoneutral and hot variation between genotypes

It is useful to separate the simulated maintenance ME requirements (ME_{maint}) into (i) the ME requirements for body protein turnover (ME_{turn}) and (ii) the part of ME_{maint} independent of protein turnover ($ME_{maint,indep}$). In the current model, the latter is affected by thermoregulatory action. ME_{turn} and $ME_{maint,indep}$ derive from equations (4) to (6) of Chapter 1. ME_{turn} represents the ME requirements for turnover of present body protein, assuming different turnover rates for different protein pools, plus repeated synthesis-and-breakdown of newly deposited protein. $ME_{maint,indep}$ is calculated as 0.65 times ME_{maint} as a function of metabolic body weight ($BW^{0.75}$); the factor 0.65 was found in Chapter 1 to fit best to the comparison of the [ME_{turn}, $ME_{maint,indep}$] complex with the conventional representation of ME_{maint}.

Table 2 Ranges of genotype averages of simulated traits in cold, thermoneutral and hot conditions

environmental temperature (°C)	$T_{LC} - 5$ †	$T_{LC} - 1$ †	thermoneutral	$T_{UC} + 1$ †	$T_{UC} + 5$ †
ME intake ($kJ.kg^{-0.75}.d^{-1}$)	1295 – 2172	1183 – 2059	1143 – 2026	1141 – 2026	1065 – 1913
daily P_{dep} ($g.d^{-1}$)	72 – 216	72 – 215	72 – 215	72 – 215	72 – 203
daily L_{dep} ($g.d^{-1}$)	136 – 403	136 – 404	136 – 404	136 – 404	125 – 400
cumulative P_{dep} (kg)	9.81 – 13.8	9.81 – 13.7	9.81 – 13.7	9.81 – 13.7	9.83 – 13.8
cumulative L_{dep} (kg)	12.6 – 31.7	12.6 – 31.7	12.6 – 31.7	12.6 – 31.7	12.4 – 31.6
ME_{maint} ($kJ.kg^{-0.75}.d^{-1}$)	588 – 680	498 – 575	470 – 537	433 – 497	412 – 456
$ME_{maint,indep}$ ($kJ.kg^{-0.75}.d^{-1}$)	409 – 430	318 – 327	288 – 291	248 – 255	212 – 235
ME_{turn} ($kJ.kg^{-0.75}.d^{-1}$)	179 – 251	178 – 250	178 – 249	178 – 249	179 – 238
HP ($kJ.kg^{-0.75}.d^{-1}$)	782 – 1104	691 – 1007	663 – 975	626 – 936	607 – 881

† "$T_{LC} - 5$" etc. refer to 5.27 and 1.27 degree below T_{LC}, and 1.43 and 5.36 degrees above T_{UC}. See the text for details.

Table 2 gives the ranges of the genotype means of simulated ME intake, protein and lipid deposition traits, heat production, and maintenance-related traits for the various simulated climatic conditions. As expected, average $ME_{maint,indep}$ shows hardly any variation among genotypes in thermoneutral conditions. Average thermoneutral ME_{turn} shows a considerable variation, functionally dependent on the genotype level of daily protein deposition. This trait shows much thermoneutral variation as well, as does daily lipid deposition. Neither the simulated protein and lipid deposition processes nor ME_{turn} are much affected by the change from thermoneutral to cold or hot conditions. Daily protein and lipid deposition are reduced by about 4 % at 5 deg above T_{UC}. In contrast, $ME_{maint,indep}$ is strongly affected, as follows.

The change from thermoneutral to mildly cold conditions (1.27 ± 0.002 degrees Celsius below T_{LC} ± the between-genotypes standard deviation; further referred to as "$T_{LC} - 1$ °C") increases

the average $ME_{maint,indep}$ (calculated as above) by 22.1 to 28.9 $kJ.kg^{-0.75}.d^{-1}$ per deg below T_{LC}. The associated extra thermoregulatory heat production (ETH; calculated as the actual heat production of each replicate at T_{LC} – 1 °C, minus the thermoneutral heat production of the same replicate, and analogously for the other climatic conditions) ranges from 22.0 to 26.2 $kJ.kg^{-0.75}.d^{-1}$ per deg, functionally related to cold tissue insulation (I_{tissue}, with simulated genotype means from 0.08 to 0.10 $°C.m^2.W^{-1}$ at 100 kg BW) as $ETH = 32.5 - 96.4 \times I_{tissue}$ ($r = 0.79$). The model aims at increasing ME intake through an increase in ME_{maint} (more specifically, $ME_{maint,indep}$) by the difference between minimum heat loss and thermoneutral heat production (see Chapter 3), which turns out, on average, to be sufficient to realise 23.8 / 24.7 = 0.97 of the required increase in heat production. Among genotypes, this ratio ranges from 0.87 to unity, the lower values being due to feed intake volume constraints. These genotypes cannot sufficiently increase their ME intake to fulfill their increased $ME_{maint,indep}$, and compromise lipid deposition to retain thermal equilibrium and to retain their thermoneutral level of daily protein deposition.

In more severely cold conditions (5.27 ± 0.003 deg below T_{LC}; "T_{LC} – 5 °C"), average $ME_{maint,indep}$ is increased by 22.5 to 26.9 $kJ.kg^{-0.75}.d^{-1}$ per deg below T_{LC}. The associated ETH ranges from 22.5 to 25.8 $kJ.kg^{-0.75}.d^{-1}$ per deg, which is 0.94 to 1.00 of the required levels. Thus the cold thermoregulatory actions of the model turn out to be linear in relation to T_{env}, as expected.

The change from thermoneutral to mildly hot conditions (1.43 ± 0.04 deg above T_{UC}; "T_{UC} + 1 °C") reduces $ME_{maint,indep}$ by 26.0 to 27.3 $kJ.kg^{-0.75}.d^{-1}$ per deg above T_{UC}. The thermoregulatory reduction of heat production (RHP) has the same magnitude. Hence in these mildly hot conditions, thermal equilibrium can just be retained by the simulated reduction in ME_{maint} as a result of diminished physical activity, by increased evaporative heat loss and/or by some increase in body temperature (see Chapter 3).

In more severely hot conditions (5.36 ± 0.04 deg above T_{UC}; "T_{UC} + 5 °C"), the average required RHP is 53.0 to 138.8 $kJ.kg^{-0.75}.d^{-1}$. This is equivalent to 10.4 to 25.9 $kJ.kg^{-0.75}.d^{-1}$ per deg above T_{UC} (as compared to 26.0 to 27.3 at T_{UC} + 1 °C). Comparison to the corresponding figures for T_{UC} + 1 °C (26.0 to 27.3, see above) shows that the hot thermoregulatory pattern is distinctly nonlinear in relation to T_{env}, as expected, and shows a progressively increasing variation between genotypes. Simulated ME_{maint} could not be reduced by more than 50.9 to 79.9 $kJ.kg^{-0.75}.d^{-1}$ (equivalent to 10.3 to 14.4 $kJ.kg^{-0.75}.d^{-1}$ per deg). To cover the RHP deficit, the model is forced to reduce daily protein deposition by 0.01 to 16 %, lipid deposition by 1 to 16 %, and ME intake by 5 to 16 % of the associated thermoneutral values.

Table 2 shows that the genotype means of thermoneutral ME_{maint} differ by 14 % between the simulated extreme combinations of the genotype parameters, and that this proportional range changes little in cold or hot conditions. The relation between the ME_{maint} and $P_{dep,max}$ genotype means turned out to be linear, with essentially the same regression coefficients at T_{LC} – 1 °C and T_{UC} + 1 °C as at thermoneutrality. The regression coefficients at T_{LC} – 5 °C and T_{UC} + 5 °C are more strongly affected, see Table 3. The ME_{maint} levels predicted from these regression parameters for $P_{dep,max}$ = 250 $g.d^{-1}$ are 11, 10, 11, 11 and 6 % higher than for $P_{dep,max}$ =

Table 3 Regression coefficients (± standard errors) of simulated genotype means of ME_{maint} (in $kJ.kg^{-0.75}.d^{-1}$) on $P_{dep,max}$ (in $g.d^{-1}$): $ME_{maint} = b_0 + b_1 \times P_{dep,max}$

environmental temperature (°C)	$T_{LC} - 5$ †	$T_{LC} - 1$ †	thermoneutral	$T_{UC} + 1$ †	$T_{UC} + 5$ †
b_0	576.4 ± 5.3	482.1 ± 3.7	452.3 ± 4.5	417.8 ± 3.7	410.3 ± 5.3
b_1	0.439 ± 0.034	0.350 ± 0.024	0.340 ± 0.029	0.314 ± 0.024	0.181 ± 0.034

† See Table 2

100 $g.d^{-1}$ in the subsequent climatic conditions from T_{LC} – 5 °C to T_{UC} + 5 °C, respectively. The thermoneutral figure equals the corresponding result from Chapter 2.

It follows that the average maintenance requirements of the simulated genotypes vary considerably with their metabolic intensity, as triggered by the combined effect of their growth potential parameters. This raises the question as to what extent each of these parameters contributes to this effect. To quantify this, the ME_{maint}, ME_{turn} and $ME_{maint,indep}$ genotype means were analysed with a regression model including the effects of P_∞, $R_{L\infty/P\infty}$, and B*, nested within each of the five climatic conditions. This analysis produced R^2 values between 0.97 and 0.99. As expected, B* had the largest effect on each of the dependent variables (F^5_{109} = 292.1, 776.5 and 28.5, respectively), followed by P_∞ (F^5_{109} = 72.6, 141.4 and 3.8) and $R_{L\infty/P\infty}$ (F^5_{109} = 35.4, 66.9 and 5.0). When this analysis was restricted to the climatic conditions from T_{LC} – 1 °C to T_{UC} + 1 °C, the contribution of $R_{L\infty/P\infty}$ to the variation among the $ME_{maint,indep}$ means became non-significant (F^3_{69} = 0.68, P = 0.57).

Cold and hot versus thermoneutral variation within genotypes

Table 4 shows that the simulated within-genotype standard deviations of the protein and lipid deposition traits are mostly unaffected by the change from thermoneutral to mildly cold or hot conditions, similar to the between-genotype variation (Table 2). Only cumulative protein and lipid deposition show somewhat more variation within some of the genotypes in cold conditions. The within-genotype variation of daily protein and lipid deposition is reduced at T_{UC} + 5 °C.

Table 4 Ranges of the within-genotype standard deviation of simulated protein and lipid deposition traits in cold, thermoneutral and hot conditions

T_{env} (°C) †	daily P_{dep} ($g.d^{-1}$)	daily L_{dep} ($g.d^{-1}$)	cumP_{dep} (kg)	cumL_{dep} (kg)
$T_{LC} - 5$	4.14 – 13.2	14.1 – 43.5	0.297 – 0.463	1.32 – 2.13
$T_{LC} - 1$	4.09 – 13.2	14.1 – 44.0	0.239 – 0.464	1.00 – 2.14
thermoneutral	4.09 – 13.2	14.1 – 44.0	0.205 – 0.464	0.78 – 2.14
$T_{UC} + 1$	4.07 – 13.2	14.1 – 44.0	0.205 – 0.464	0.78 – 2.14
$T_{UC} + 5$	3.64 – 11.4	14.1 – 42.3	0.213 – 0.461	0.77 – 2.11

† See Table 2

Table 5 shows that the within-genotype standard deviations of ME_{turn} follow the patterns of the variation in daily and cumulative protein deposition (Table 4), but the effects of climatic change on its variation are very small. As expected, the change from thermoneutral to cold or hot conditions causes a progressive increase of the within-genotype variation of $ME_{maint,indep}$, to the extent that the variance due to thermoregulatory action comprises about 4 % of the variance in ME_{turn}, and in some genotypes much more so at T_{LC} – 5 °C. As a result of these climate-induced changes in the variation in ME_{turn} and $ME_{maint,indep}$, and as a result of changes in the covariance between these traits (not shown), the within-genotype standard deviation of ME_{maint} decreases with increasing environmental temperature.

Table 5 Ranges of the within-genotype standard deviation of simulated maintenance-related traits (all in $kJ.kg^{-0.75}.d^{-1}$) in cold, thermoneutral and hot conditions

T_{env} (°C) †	ME_{maint}	$ME_{maint,indep}$	ME_{turn}
T_{LC} – 5	7.45 – 12.1	1.36 – 8.29	6.90 – 8.51
T_{LC} – 1	7.09 – 9.21	0.75 – 2.40	6.84 – 8.48
thermoneutral	6.79 – 8.48	0.05 – 0.50	6.82 – 8.45
T_{UC} + 1	6.56 – 8.34	0.65 – 2.10	6.82 – 8.44
T_{UC} + 5	6.51 – 7.28	0.87 – 2.29	6.86 – 7.88

† See Table 2

Because the absolute levels of ME_{maint} also decrease with increasing environmental temperature (Table 2), the within-genotype coefficients of variation of ME_{maint} are hardly affected by climatic change. These values range around 1.5 % (similar to the values found in Chapter 2), with somewhat more variation between genotypes at T_{LC} – 5 °C.

The within-genotype variation in ME_{maint} results from the combined variation in $ME_{maint,indep}$ and in ME_{turn}, and it is of interest to what extent each component contributes to the total variation. To quantify this, a simple regression of ME_{maint} on ME_{turn} was performed within genotypes. The R^2 values of these analyses have been summarised in Table 6, and show that ME_{turn} determines practically the entire within-genotype variation in thermoneutral maintenance requirements, as expected. The contribution of $ME_{maint,indep}$ (the complement of the proportions in Table 6) in hot conditions is still very limited, at most 7 % of the ME_{maint} variance. This proportion is somewhat larger at T_{LC} – 1 °C, but it is only at T_{LC} – 5 °C that the variation in thermoregulatory energy expenditure of some genotypes causes a noticeable variation in ME_{maint}.

Table 6 Ranges of the proportions of within-genotype variance of ME_{maint} due to variation in ME_{turn} in cold, thermoneutral and hot conditions

T_{env} (°C) †	proportion
T_{LC} – 5	0.451 – 0.966
T_{LC} – 1	0.916 – 0.989
thermoneutral	0.995 – 0.998
T_{UC} + 1	0.940 – 0.991
T_{UC} + 5	0.927 – 0.990

† See Table 2

The relations between maintenance requirements and body composition traits in the various simulated climatic conditions are summarised in Figure 1. ME_{maint} of individual replicates is

shown here in relation to cumulative and daily protein deposition from 23 to 100 kg BW. To minimise overlap of genotype scatter plots and hence make the graphs more informative, only ten of the 21 simulated genotypes are shown. These are representative for the full range in terms of their average x- and y-coordinates and their ME_{maint} variation.

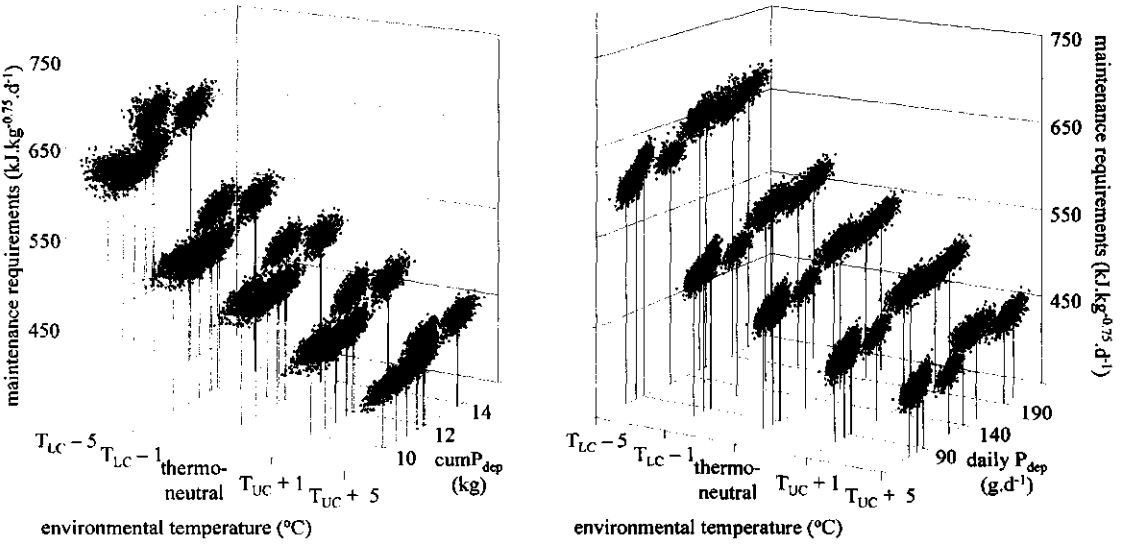

Figure 1 Simulated maintenance ME requirements in relation to simulated cumulative (left) and daily (right) body protein deposition (23 to 100 kg body weight), in five climatic conditions. The scatterplots show 500 random replicates for each of ten representative genotypes. The vertical anchor lines indicate the genotype means. See the text for further details.

The between- and within-genotype variation of the traits in Figure 1 reflect the results in Tables 4 to 6. At each level of T_{env} the ten scatterplots (*i.e.* genotypes) line up in terms of increasing protein deposition, with so much overlap between them that a more or less continuous distribution is created. Each of these aggregates shows an overall increase in ME_{maint}: the between-genotypes relation between ME_{maint} and protein deposition is clearly positive. A similar pattern can be detected *within* most of the scatterplots in Figure 1 (similar to figure 2 in Chapter 2), but the within-genotypes relation between ME_{maint} and protein deposition (*i.e.* the slope of the scatterplots) varies considerably. This is most evident in the left-hand plot at T_{LC} − 5 °C, and in the right-hand one at T_{UC} + 5 °C. In a quantitative sense, the most important feature here is the variation between genotypes in the extent to which ME_{maint} depends on each of the protein deposition traits.

This is summarised in Figure 2, which shows the estimates from model (1) of the within-genotype regression coefficients of ME_{maint} on cumulative and daily protein deposition. These have been plotted in relation to the genotypes' average ME_{maint}, in an attempt to show the metabolic load of the various simulated climatic conditions for each particular genotype. Hence the slope of each of the scatterplots in Figure 1 (plus the 11 × 5 other ones not shown there, see above) is shown on the vertical axis of Figure 2; on the horizontal axis is that

scatterplot's mid-value of ME_{maint}, which is its vertical aspect in Figure 1. The line plots in Figure 2 connect the five data points (five levels of T_{env}) for each genotype.

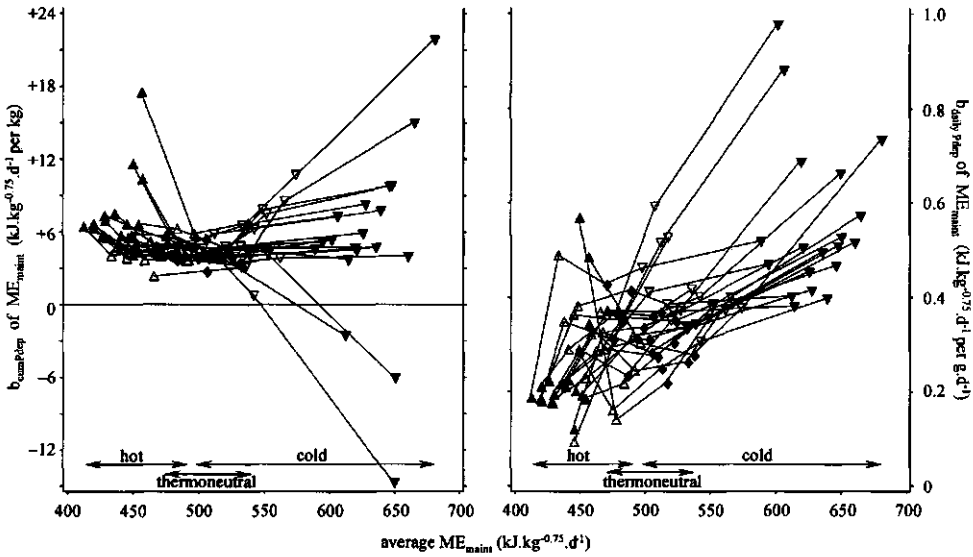

Figure 2 Within-genotype linear regression coefficients (statistical model (2)) of maintenance ME requirements (ME_{maint}) on cumulative ($cumP_{dep}$, left) and on daily protein deposition (daily P_{dep}, right), in relation to the genotype means of ME_{maint}, in five climatic conditions (▲: 5 degrees above T_{UC}; Δ: 1 deg above T_{UC}; ♦: thermoneutral; ∇: 1 deg below T_{LC}; ▼: 5 deg below T_{LC}). The line plots connect the data points of each genotype. Higher values of the y-variables correspond to steeper slopes of the associated scatterplots in Figure 1.

The 21 simulated genotypes fall apart into four groups, distinguished by the regression coefficients of ME_{maint} on cumulative protein deposition and by those on daily protein deposition ($b_{cumPdep}$ and $b_{dailyPdep}$ from model (1)), as follows.

(i) Three genotypes show negative $b_{cumPdep}$ estimates at T_{LC} – 5 °C. These genotypes grow and metabolise at high rates, but are at the same time very fat: they have the highest mean values and within-population variation of daily lipid deposition, and the highest mean daily ME intake, of all 21 evaluated genotypes. Moreover, they show high means of cumulative lipid deposition and daily protein deposition (and hence ADG), and have high means and variation of thermoneutral heat production. In fact, these genotypes belong to the same class as the six that were discarded for further analysis because of unrealistic combinations of genotype parameters (see the first paragraph of the **Simulation results** section).

(ii) Three other genotypes show strongly elevated $b_{dailyPdep}$ estimates at T_{LC} – 5 °C. By contrast with group (i), these genotypes grow and metabolise at low rates: they have the lowest means of daily ME intake, ADG and thermoneutral heat production of all 21 evaluated genotypes. Moreover, they show low means and variation of daily lipid deposition, and low means of daily protein deposition.

(iii) Three genotypes show strongly elevated $b_{cumPdep}$ estimates both at T_{UC} + 5 °C and at T_{LC} – 5 °C. At the same time, their $b_{dailyPdep}$ estimates are comparatively high at T_{LC} – 5 °C and strongly elevated at T_{UC} + 5 °C. These genotypes have the highest means and lowest variation of cumulative protein deposition, and the lowest means and variation of cumulative lipid deposition, of all 21 evaluated genotypes. Moreover, they show low means and variation of dailylipid deposition. These fast-growing lean genotypes represent the most advanced "meat-type pigs" of the current industry (see Chapter 6), combining the highest P_∞ and B_{Gomp} levels with the lowest $R_{L\infty/P\infty}$ levels.
(iv) The remaining twelve genotypes show $b_{cumPdep}$ estimates that are, on average, somewhat higher in cold (3.73 to 9.77 $kJ.kg^{-0.75}.d^{-1}$ per kg at T_{LC} – 5 °C) and hot conditions (4.99 to 7.59 at T_{UC} + 5 °C) than at thermoneutrality (2.77 to 5.45). Their $b_{dailyPdep}$ estimates decrease steadily with increasing T_{env} levels, from 0.382 to 0.519 $kJ.kg^{-0.75}.d^{-1}$ per $g.d^{-1}$ at T_{LC} – 5 °C, via 0.250 to 0.428 at thermoneutrality, to 0.124 to 0.287 at T_{UC} + 5 °C. These genotypes could be characterised as "conventional".

Disregarding the "odd" groups of genotypes (i, ii), the above can be generalised as follows.
Leaner pigs have higher maintenance requirements; this relation becomes stronger when conditions get either cold or hot. More formally, the regression of ME_{maint} on cumulative protein deposition is progressively higher in cold and hot conditions than at thermoneutrality. The average thermoneutral $b_{cumPdep}$ value of 4.19 $kJ.kg^{-0.75}.d^{-1}$ per kg in conventional genotypes is increased by 8 and 48 % in mildly and severely hot conditions, respectively. It is increased by 14 and 32 % in mildly and severely cold conditions.
Pigs with higher lean growth rates have higher maintenance requirements; this relation becomes stronger when conditions get colder, and weaker when conditions get warmer. More formally, the regression of ME_{maint} on daily protein deposition decreases with increasing environmental temperature. The average thermoneutral $b_{dailyPdep}$ value of 0.322 $kJ.kg^{-0.75}.d^{-1}$ per $g.d^{-1}$ in conventional genotypes is reduced by 11 and 36 % in mildly and severely hot conditions, respectively. It is increased by 20 and 48 % in mildly and severely cold conditions.
Both patterns are much more pronounced in the more advanced "meat-type pig" genotypes.

Discussion

In Chapter 3 it was attempted to integrate present quantitative knowledge of thermoregulatory processes into a dynamic growth simulation model. In the present study, this extended simulation model was made stochastic to generate replicates (animals) with variable body composition and, hence, variable energy requirements for thermoregulation. It must therefore be stressed that the between-animal variation of ME_{maint} in our present simulations is the result of variation in body composition-related traits only. Specifically, variation in behavioural characteristics is entirely ignored.

As mentioned earlier, apart from thermoregulatory functions, the model used here differs from the one used in Chapter 2 mainly in the use of the potential growth and feed intake rules of Emmans (1988, 1997) *versus* the nutrient partitioning rules of Whittemore and Fawcett (1974) and Moughan and Smith (1984). Hence a comparison of the present thermoneutral simulation results with those from Chapter 2 is largely equivalent to a comparison of those two sets of

metabolic rules. Although these sets seem strongly different at first sight, the results that follow from the simulation models in which they were incorporated are surprisingly similar, up to and including the within-genotype variation of protein and lipid deposition and maintenance requirements. The most important difference between these models is the prediction of *ad libitum* feed intake according to Emmans (1997). Moughan and Smith's (1984) parameters are discussed in Chapter 2, and those of Emmans (1988) in Chapter 6.

Adequacy of the model

As long as the *ad libitum* feed intake of our simulated genotypes is not constrained, simulated protein and lipid deposition are hardly affected by cold or hot conditions. This would be consistent with the remarks by Black *et al.* (1999, p. 79): "when [...] pigs exposed to cold are given free access to feed and can increase consumption sufficiently to compensate for the additional heat loss, there appear to be no effects of temperature on the rates of either fat or protein deposition" and "there is no evidence for a direct effect of temperature on the [chemical] body composition of pigs other than through a change in energy intake. This appears to be true even under conditions of extreme heat".

Averaged over genotypes and over the 23 to 100 kg BW growth trajectory, our simulations predict an increase of heat production in cold conditions of ETH = 23.8 $kJ.kg^{-0.75}.d^{-1}$ per degree Celsius. Holmes and Close (1977; table 4.7) reviewed the literature on growing pigs between 1966 and 1976 and report ETH values of 28.8, 25.0 and 23.8 $kJ.kg^{-0.75}.d^{-1}$ per deg for group-housed pigs of 20, 60 and 100 kg BW, respectively. Our simulation results seem to agree with the published ETH data.

The average prediction for the reduction of heat production in hot conditions of RHP = 26.6 $kJ.kg^{-0.75}.d^{-1}$ per deg is higher than the one for ETH (as it should be). Close (1978) and Close and Mount (1978) subjected *ad libitum*-fed pigs of about 40 kg BW to indirect calorimetry at $10 \leq T_{env} \leq 30$ °C. They estimated T_{LC} at 16.6 °C, and their reported change in ME_{maint} below 15 °C (subcritical) gives an estimate of ETH at 29.2 $kJ.kg^{-0.75}.d^{-1}$ per deg. The reported measurements of sensible heat loss give an estimate of RHP at 45.1 $kJ.kg^{-0.75}.d^{-1}$ per deg. Giles and Black (1989) measured O_2 consumption in pigs of about 90 kg BW at $23 \leq T_{env} \leq 31$ °C. The associated RHP value is around 23 $kJ.kg^{-0.75}.d^{-1}$ per deg when assuming a respiratory quotient of unity for these growing pigs, but it is difficult to decide upon the upper bound of the thermoneutral zone from the published results. Ferguson and Gous (1997) derived heat production from measurements of ME intake and retention in pigs between 13 and 30 kg BW at $18 \leq T_{env} \leq 30$ °C and on various dietary CP levels. Their results suggest an RHP value between 30 and 46 $kJ.kg^{-0.75}.d^{-1}$ per deg. Our simulation results may be at the lower range of the published RHP data, but it is difficult to make a meaningful comparison with these literature results, given the large differences in body weight and the uncertain assumptions that had to be made when interpreting the experimental data.

Although the cold and hot conditions simulated in this study are long-term, the simulation model does not provide for acclimatisation functions. As noticed in Chapter 3, this is one of its major shortcomings. The thermal load at $T_{LC} - 1$ °C or $T_{UC} + 1$ °C is limited, and hence the corresponding "real life" acclimatisation effects should be limited too. On the other hand, these mildly cold or hot conditions operate during the entire simulated growth period, which

is a more extreme situation than what most pigs ever encounter in real life. Acclimatisation functions are notoriously difficult to quantify (see Pohl, 1976; Brück, 1986; Young *et al.*, 1989; Derno *et al.*, 1995) and poorly documented, which is the main reason why they were not included into the model.
Any metabolic event in cold or hot conditions cannot be considered independent from previous events: the pig's energy metabolism and body composition is continuously influenced by the thermal environment. The simulation results cannot be interpreted out of that context; for example, the simulated events at 50 kg BW are not equivalent to a short cold or hot treatment of a 50 kg pig that was thermoneutral previously. One of the most difficult tasks in a study like the present one is to specify the pattern of environmental conditions to be simulated. The required balance is the one between obtaining a manageable and reproducible contrast (*e.g.* T_{LC} – 1 °C *versus* T_{UC} + 1 °C) on the one hand, and staying close to real-life conditions on the other hand. The purpose of the present study is to determine the effect of deviations from thermoneutrality on the between-animal variation in energy metabolism, and for that particular purpose it was "experimentally" convenient to simulate permanently cold or hot conditions. Such conditions would rarely be required in the simulation of any real-life system.

It should be stressed again that variation in ME_{maint} independent from body composition is ignored in this study. See also the end of Appendix 1.

Cold, thermoneutral and hot variation

We concentrate here on the three "advanced" (iii) and twelve "conventional" (iv) genotypes mentioned at the end of the **Simulation results** section; the plots of these two groups from Figure 2 have been averaged in Figure 3. Higher thermoneutral levels of daily and cumulative protein deposition are associated with higher levels of ME_{turn} (see Chapter 2) and hence with higher thermoneutral ME_{maint}. It is therefore consistent with expectations that the thermoneutral regression coefficients of ME_{maint} on daily and cumulative protein deposition in Figure 3 are positive. The changes in these regression coefficients with the change from thermoneutral to cold or hot conditions are much more interesting, and need some specific attention.

(i) Cold $b_{dailyPdep}$. The increase in the regression coefficients of ME_{maint} on daily protein deposition with the change to cold conditions is an "inverse dilution effect": $ME_{maint,indep}$ is progressively increased in the cold but, once $P_{dep,max}$ has been reached, daily protein deposition is not affected by this. The higher regression coefficient then just expresses a wider range of the y-variable at the same range of the x-variable.

(ii) Hot $b_{dailyPdep}$. Of course, the opposite effect takes place with the change from thermoneutral to hot conditions. In addition, the higher thermoneutral ME_{turn} (and ME_{maint}) associated with higher levels of thermoneutral daily protein deposition lead to a higher required reduction of heat production with increasing T_{env}, and thus to a higher RHP and a larger reduction of ME_{maint} from thermoneutral to hot conditions. So the decrease in the regression coefficients of ME_{maint} on daily protein deposition with the change of the conventional genotypes from thermoneutral to hot conditions in Figure 3 is consistent with expectations.
However, the *increase* in the fast-growing lean genotypes at T_{UC} + 5 °C is unexpected; further analyses showed a significant interaction between daily and cumulative protein deposition

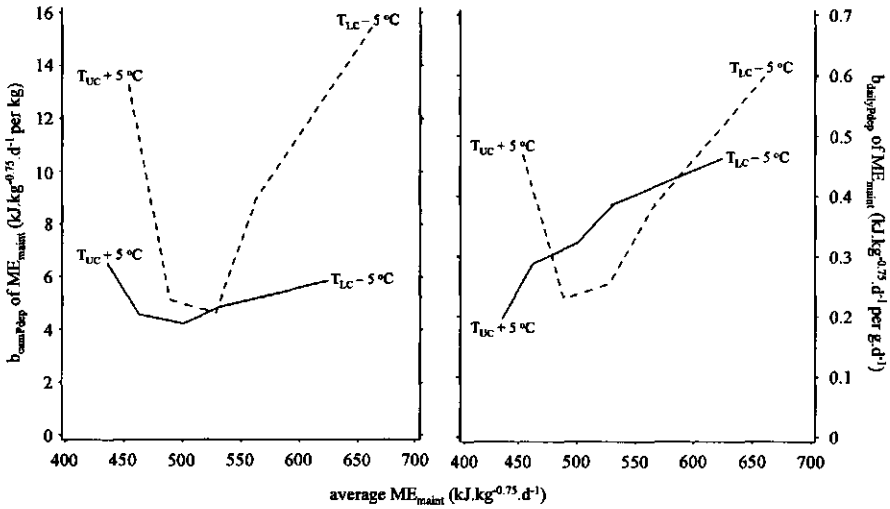

Figure 3 Linear regression coefficients of maintenance ME requirements (ME_{maint}) on cumulative ($cumP_{dep}$, left) and on daily protein deposition (daily P_{dep}, right), in relation to the genotype means of ME_{maint}, in five climatic conditions. Solid lines: averages of the estimates in Figure 2 of twelve conventional genotypes; broken lines: averages of the estimates in Figure 2 of three fast-growing lean genotypes.

(daily P_{dep} and $\Sigma cumP_{dep}$ in statistical model (1)): the "hot" $b_{dailyPdep}$ estimates for these genotypes are strongly elevated ($P<0.0001$) at the highest values of $\Sigma cumP_{dep}$, which increases the average estimate (Figure 3) more than twofold. The change to T_{UC} + 5 °C leads these genotypes to reduce their daily protein deposition from 188 to 163 g.d^{-1}, which reduces ME_{turn} from 240 to 230 kJ.kg$^{-0.75}$.d^{-1}, but the variation in ME_{turn} is not affected. By contrast, the standard deviation of daily protein deposition is reduced from 9.57 to 7.89 g.d^{-1}. As a consequence, the regression of ME_{turn} on daily protein deposition is increased. The same pattern occurs in the conventional genotypes, but much less pronounced. This can be seen in Figure 4, where the ME_{maint} patterns in Figure 3 are repeated for ME_{turn} and $ME_{main,indep}$.

(iii) Cold $b_{cumPdep}$. At a fixed end weight, higher levels of cumulative protein deposition are associated with lower levels of cumulative lipid deposition, which translate into lower subcutaneous fat depth, lower tissue insulation levels, and higher heat loss in cold conditions. This leads to a higher required increase of heat production to retain thermal equilibrium with decreasing T_{env}, and thus to a higher ETH and to a larger increase in $ME_{maint,indep}$ and ME_{maint} from thermoneutral to cold conditions. Hence the increase in the regression coefficients of ME_{maint} on cumulative protein deposition with the change from thermoneutral to cold conditions in Figure 3 is consistent with expectations. The more pronounced increase in the fast-growing lean genotypes is also consistent with this.

(iv) Hot $b_{cumPdep}$. The increase in the regression coefficients of ME_{maint} on cumulative protein deposition with the change from thermoneutral to hot conditions in Figure 3 is not obvious. The interaction between daily and cumulative protein deposition levels on ME_{maint} that was

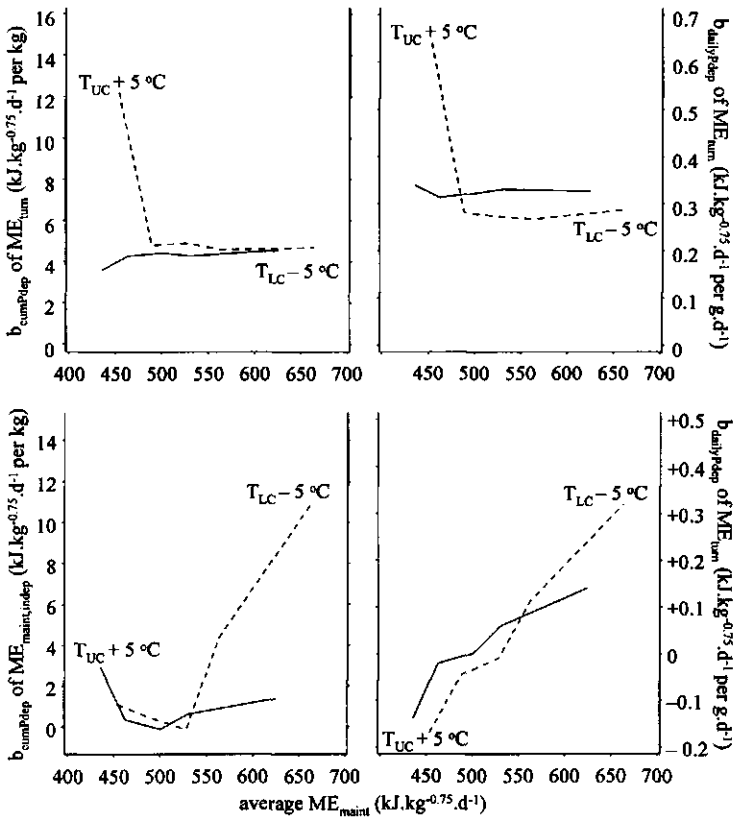

Figure 4 The same relationships as in Figure 3, for ME requirements for protein turnover (ME_{turn}; top) and for maintenance ME requirements independent of protein turnover ($ME_{maint,indep}$; bottom).

mentioned at point (ii) is non-significant ($0.07 \leq P \leq 0.98$) here. Figure 4 shows that the elevated ME_{maint} regression of the conventional genotypes is entirely due to (as yet unexplained) effects on $ME_{maint,indep}$, whereas the advanced genotypes show a strongly elevated regression coefficient of ME_{turn} on cumulative protein deposition, similar to the situation at point (ii) above. Part of the difference in the patterns of these two groups is due to the interrelations among the variables analysed in statistical model (1). The simple correlation between cumulative and daily protein deposition ($cumP_{dep}$ and $\Sigma dailyP_{dep}$ in model (1)) ranges up to 0.5 in the conventional genotypes, but is essentially zero in the advanced ones; this reduces the $b_{cumPdep}$ estimate for ME_{turn} in the conventional genotypes, but leaves the estimate in the advanced ones unaffected. But essentially, the hot $b_{cumPdep}$ pattern in Figure 3 is not properly understood.

For points (i) to (iii) above, the predictions of the simulation model are qualitatively obvious. Their added value is in the quantification of the described trends, most notably in the prediction of much more pronounced trends of cold and hot ME_{maint} in relation to cumulative

protein deposition in the fast-growing lean genotypes. This suggests an increase of environmental sensitivity in the most advanced modern pig breeds.
But for point (iv), the simulation challenges our present understanding of the system under study. This may be due to inadequacy of the model just as well as to an overly simplistic representation of reality, or of course to a "true" effect. It therefore calls for experimental verification.

Comparison to experimental results

The purpose of (simple and cheap) simulation studies must be to provide the information that is required for the proper statistical design of the (elaborate and costly) real-life experiment that is supposed to provide a true scientific advance. To design the trial that could verify our simulation results we need *a priori* information on the (co-)variances of the characteristics to be measured, and the simulation provides estimates of these. We will consider briefly, and not exhaustively, how they can be used.

Basically, our experiment should provide the data required to verify the relations predicted in Figure 1 and, especially, in Figure 2. Such data are to be collected on a group of pigs growing from 23 to 100 kg BW, sampled so as to realise a within-group variation in the independent variables (daily P_{dep} and $cumP_{dep}$) of a similar magnitude as in our simulation results (Table 4). This can be ensured by feeding the animals *ad libitum* with a balanced diet and varying their genetic predisposition for protein deposition. The latter could be approximated in terms of the estimated breeding values for growth and body composition-related traits such as they are routinely produced in practical pig breeding programmes.

An effective T_{env} that stays close to 5 deg below the regularly changing T_{LC} of a particular genotype during its whole growth period can be simulated quite easily. To physically create such an environment for the real-life experiment set up to evaluate that simulation is prohibitively difficult, mainly because the true critical temperature cannot be assessed without grossly disturbing the trial itself. This may be overcome by running the experiment at a T_{env} that is certain to be subcritical all the time (which means that it will have to be gradually reduced when the pigs grow heavier), and carry out a series of parallel trials with pigs of the relevant genotype, body weight, growth rate and fatness that are kept at a range of T_{env} levels to evaluate their HP profiles and establish their T_{LC}. The results can then be used to regularly estimate the difference between T_{env} and T_{LC} of the pigs in the main experiment. Since the cold thermoregulatory actions of the model were found to be linear in relation to T_{env} (see the **Simulation results** section), evaluation of the simulation results should be easily accomplished by extrapolation. The hot thermoregulatory pattern is non-linear, and would require more detailed simulation results (in terms of smaller T_{env} steps than in the present study) to allow for proper evaluation. In all cases, a proper characterisation of the genotype used in the experiments (in terms of its model parameters) would be necessary to allow for a meaningful simulation to be evaluated.

The aim of the experiment would be to obtain estimates for the regression coefficients of ME_{maint} on cumulative and daily protein deposition, and to test the differences between these estimates in various climatic conditions. The more interesting comparisons involving daily

P_{dep} are statistically more demanding than those involving $cumP_{dep}$. Hence we focus on $b_{dailyPdep}$ in the genotypes represented in Figure 3

For the group of conventional genotypes in that Figure, $b_{dailyPdep}$ ranges from 0.250 to 0.428 $kJ.kg^{-0.75}.d^{-1}$ per $g.d^{-1}$ in thermoneutral conditions, and from 0.306 to 0.527 at T_{LC} – 1 °C. The associated correlation coefficients (square roots of the R^2 of model (1)) are r = 0.54 to 0.65, and r = 0.56 to 0.73, respectively. These figures can be used to parameterise the equation for the standard error ($se_{b,i}$) of the estimate of a linear regression coefficient (b_i) that is based on n_i observations made in treatment i (see Chapter 2):

$$se_{b,i} = \sqrt{\frac{1-r_i^2}{(n_i-2)\times r_i^2}\times b_i^2} \qquad (2)$$

For a difference between the regression coefficients estimated in cold and thermoneutral conditions (b_C, b_N) to be found significant at the 5 % level in a one-sided t-test, we would require $(b_C - b_N) / t_{0.95} \leq se_b$, using a pooled standard error according to

$$se_b = \sqrt{\frac{n_C \times se_{b,C}^2 + n_N \times se_{b,N}^2}{n_C + n_N}} \qquad (3)$$

Assuming $n_C = n_N = n$, we can substitute $n_i \times se_{b,i}^2$ from equation (2) into equation (3) to obtain

$$\frac{(r_C^{-2}-1)\times b_C^2 + (r_N^{-2}-1)\times b_N^2}{2\times(n-2)} \leq \frac{(b_C - b_N)^2}{t_{0.95}^2}$$

which leads to

$$n_{req} \geq 2 + \tfrac{1}{2} t_{0.95}^2 \times \frac{(r_C^{-2}-1)\times b_C^2 + (r_N^{-2}-1)\times b_N^2}{(b_C - b_N)^2} \qquad (4)$$

for the required numbers of observations (n_{req}) in each of the two climatic conditions to be compared.

Notice that n_{req} approaches infinity for $b_C \to b_N$, and also for $r_C \to 0$ or $r_N \to 0$. For $r_C \to 1$ and $r_N \to 1$, n_{req} approaches the value of 2.

Substituting the above estimates for $b_{dailyPdep}$ and their correlation coefficients into equation (4) gives estimates of n_{req} ranging from 42 to 750 observations, mainly dependent on the actual difference between the regression coefficients per genotype. The median value is n_{req} = 153. This would then be the average required number of observations (per treatment) of individual daily P_{dep} and ME_{maint} to detect the simulated difference between their regression coefficients at T_{LC} – 1 °C *versus* in thermoneutral conditions, at a 5 % significance level. Calculated the same way, the n_{req} estimates for T_{LC} – 5 °C and T_{UC} + 5 °C (with much larger contrasts *versus* the thermoneutral values) are 27 (the median value of estimates ranging from 18 to 116) and 21 (12 to 422), respectively. The mostly very small contrasts of the estimates at T_{UC} + 1 °C *versus* the thermoneutral ones would require n_{req} = 297 (11 to 12022).

Calculated the same way for the three advanced genotypes in Figure 3, the n_{req} estimates are 8 (6 to 11) observations at T_{LC} – 5 °C, 32 (27 to 58) at T_{LC} – 1 °C, 865 (46 to 1032) at T_{UC} + 1 °C, and 28 (16 to 86) observations at T_{UC} + 5 °C. Again, these would be the required numbers

of observations per treatment. These numbers should also be sufficient to test the differences in $b_{dailyPdep}$ between the two genotypes groups at either of the five climatic conditions.

So far it has been assumed that individual daily P_{dep} and ME_{maint} can be measured without error, and this assumption is clearly violated in practice. Measurement errors in the y-variable in a regression model (*i.e.* ME_{maint}) do not affect the regression coefficient but reduce the correlation, which may drastically increase n_{req} from equation (4). Measurement errors in the x-variable (*i.e.* daily P_{dep}) cause a negative bias on the estimate of the regression coefficient itself. As shown and discussed in Chapter 2, this would make the experimental verification of our simulation results very difficult.

Conclusions

The 21 genotypes simulated in this study show realistic values of deposition and maintenance-related traits (Table 2). They differ considerably from each other, which can be summarised in terms of their mean $P_{dep,max}$ values which range from 77 to 241 $g.d^{-1}$. An increase in mean $P_{dep,max}$ from 100 to 250 $g.d^{-1}$ is predicted to cause an 11 % increase in the mean level of thermoneutral ME_{maint} which is, in this model, mediated by variation in protein turnover only. The corresponding increase in ME_{maint} in cold or hot conditions is very similar. Differences in body composition traits between pig genotypes do not cause important differences in thermoregulatory ME_{maint} between those genotypes.

The simulated variation among the genotype means of ME_{maint}, ME_{turn} and $ME_{maint,indep}$ can be attributed to variation in the model parameters B*, P_{∞}, and $R_{L\infty/P\infty}$, in that order of importance. $R_{L\infty/P\infty}$ makes a significant contribution to the variation among the $ME_{maint,indep}$ means only in more severely cold or hot conditions.

Within each of the 21 simulated genotypes, thermoneutral ME_{maint} shows a 1.5 % coefficient of variation due to variation in protein turnover, functionally dependent on protein deposition traits. In cold or hot conditions, both the mean level and the within-population variation of ME_{maint} are increased or reduced, respectively, so that this CV level is maintained. Thermoregulatory processes contribute little body composition-related variation to hot or cold ME_{maint} within most genotypes.

The regression coefficients of ME_{maint} on cumulative or daily protein deposition are affected by a change from thermoneutral to cold or hot conditions, but this change is limited in conventional genotypes as long as the deviation from thermoneutrality is not severe. However, these regression coefficients are strongly elevated in cold and severely hot conditions in the most extreme lean and fast-growing genotypes, suggesting an increase in environmental sensitivity in advanced genotypes.

It will require a very elaborate and large-scaled experiment to verify or falsify most of these predictions. Particularly, the predicted change in the regression coefficient of ME_{maint} on P_{dep} in cold or hot versus thermoneutral conditions is unlikely to be ever measured in real life.

P_{dep} are statistically more demanding than those involving $cumP_{dep}$. Hence we focus on $b_{dailyPdep}$ in the genotypes represented in Figure 3

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Acknowledgements

Valuable contributions were made by Martin Verstegen, Gerry Emmans, Ella Luiting, Bas Kemp, and an anonymous referee.

Appendix I. Approximation of the coefficients of variation of P_∞, $R_{L\infty/P\infty}$, and B_{Gomp}.

Estimated genetic standard deviations of ADG, DFI and carcass lean content (L%), established in growing pigs with *ad libitum* feeding, were obtained from sources quoted by Ducos (1994; figures 1a and 2b) and by Clutter and Brascamp (1998; table 15.1), and further from Fender *et al.* (1979), Johansson *et al.* (1986), Kalm (1986), Cameron *et al.* (1990), Knap (1990), Brandt and Götz (1993), Karras *et al.* (1993), Short *et al.* (1994), Stern *et al.* (1994), Von Felde *et al.* (1996), Knapp *et al.* (1997), Labroue *et al.* (1997), Gibson *et al.* (1998), Groeneveld *et al.* (1998), Hall *et al.* (1998), Hermesch *et al.* (1998) and Tholen *et al.* (1998). Because the simulation model in its present form does not predict L%, these standard deviations were transformed into standard deviations of cumulative protein deposition making use of the regression of body protein content on carcass lean content, which ranges from 0.19 to 0.31 $kg.kg^{-1}$ in the literature (Kielanowski, 1976b; Rook *et al.*, 1987; Siemens *et al.*, 1989). These are phenotypic values, we have assumed a value of 0.25 $kg.kg^{-1}$ for the associated genetic regression coefficients.

The genetic standard deviations from this review range from 17 to 91 $g.d^{-1}$ for ADG (n=67), from 61 to 214 $g.d^{-1}$ for DFI (n=28), and from 0.22 to 0.72 kg protein per 100 kg BW for cumulative protein deposition (n=45, transformed values), respectively. The frequency distributions of these estimates are represented by the broken line plots in Figure 5.

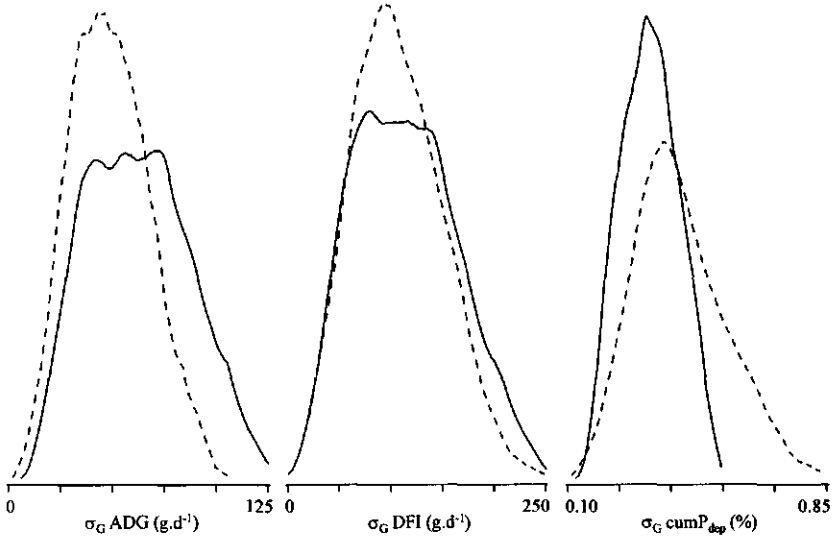


Figure 5 Frequency distributions of the estimated genetic standard deviations (σ_G) of average daily gain (ADG), average daily feed intake (DFI) and cumulative body protein deposition ($cumP_{dep}$) from the simulations carried out in the present study (——) and from a literature review (– – –). In the review, $cumP_{dep}$ was approximated as 0.25 × carcass lean content; its dimension is kg protein per 100 kg body weight. The scales of the vertical axes are arbitrary.

Reasonable values for the coefficients of variation of P_∞, $R_{L\infty/P\infty}$, and B* could then be derived by comparing the predicted within-genotype standard deviations of ADG, DFI and cumulative

protein deposition from our thermoneutral simulations to the above literature data, and adjusting the CV values until a satisfactory match was achieved. A similar procedure was followed by Ferguson *et al.* (1997).

The within-genotype standard deviations of ADG, DFI and cumulative protein deposition for the simulated genotypes that are represented by the solid line plots in Figure 5 were obtained with $\sigma_{P\infty} = 0.07 \times \mu_{P\infty}$, $\sigma_{RL\infty/P\infty} = 0.15 \times \mu_{RL\infty/P\infty}$, and $\sigma_{B^*} = 0.03 \times \mu_{B^*}$. This constitutes the final set of CV values used in the further simulations in this study.

On average, these CV values for P_∞ and $R_{L\infty/P\infty}$ can be shown to correspond to a CV for mature body weight of 0.077, which would be a genetic variation coefficient. The CV values for observed adult body weight or estimated mature body weight as reported in the literature for pigs (Kemp *et al.*, 1991 and B. Kemp, personal communication, 1999; Grandhi, 1992; Backus *et al.*, 1997), sheep (Stobart *et al.*, 1986; Näsholm and Danell, 1996), cattle (Jenkins *et al.*, 1991; Northcutt and Wilson, 1993; Bullock *et al.*, 1993; Oliveira *et al.*, 1994; Meyer, 1995; Koenen *et al.*, 1999), poultry (Hancock *et al.*, 1995) and mice (Kownacki and Keller, 1978; Parratt and Barker, 1982; Bünger and Schönfelder, 1984; Kachman *et al.*, 1988) range between 0.075 and 0.138. Combining these phenotypic values with the simulated "genetic" estimate suggests heritabilities for mature body weight of $0.4 < h^2 < 1$. This would agree with the h^2 estimates by Parratt and Barker (1982), DeNise and Brinks (1985), Stobart *et al.* (1986), Jenkins *et al.* (1991), Northcutt and Wilson (1993), Bullock *et al.* (1993), Oliveira *et al.* (1994), Meyer (1995), and Näsholm and Danell (1996), which range from 0.45 to 0.73.

The simulated range of standard deviations of $cumP_{dep}$ is clearly narrower than the range derived from the reviewed L% values. This may indicate that the above CV parameterisation is not yet optimal, but it is at least partly caused by the assumedly fixed value of 0.25 $kg.kg^{-1}$ for the genetic regression of body protein content on carcass lean content, which is likely to be more or less invalid for most of the genotypes in the review. In addition, carcass lean content was measured in a wide variety of ways in those studies (often documented poorly), and the more extreme genetic standard deviations of L% may well reflect another trait being measured. The simulated range of standard deviations of ADG is somewhat wider than the range derived from the literature, and it includes more high values. The growth rate of our simulated pigs is not restricted by environmental factors (possibly non-thermoneutral conditions, and impaired health conditions and other stressors) that would cause a reduction of ADG and of its variation in real life. The same would hold for DFI, but the simulation does not completely cover the real-life variation in maintenance requirements (activity and immune and stress responses are not included in the model in its present form) which seems to counterbalance the above effect on DFI variation.

Appendix II. Dimensions and meaning of parameter abbreviations used in Chapter 4.

parameter	dimension	meaning
μ_i		mean of parameter i
σ_i		standard deviation of parameter i
ΣcumPdep	same as $cumP_{dep}$	collapsed $cumP_{dep}$ variable (model (1))
ΣdailyPdep	same as $dailyP_{dep}$	collapsed daily P_{dep} variable (model (1))
ADG	$g.d^{-1}$	average daily gain
B*	same as B_{Gomp}	scaled B_{Gomp} parameter
$b_{cumPdep}$	$kJ.kg^{-0.75}.d^{-1}$ per kg	regression of required ME on $cumP_{dep}$
$b_{dailyPdep}$	$kJ.kg^{-0.75}.d^{-1}$ per $g.d^{-1}$	regression of required ME on daily P_{dep}
B_{Gomp}	$kg.d^{-1}.kg^{-1}$	rate parameter of protein and lipid growth
BW	kg	body weight
$cumP_{dep}$	kg	cumulative protein deposition
CV		coefficient of variation (= σ / μ)
$dailyP_{dep}$	$g.d^{-1}$	daily protein deposition
dev_i		random Normal deviate for trait i
DFI	$g.d^{-1}$	daily feed intake
ETH	same as HP	required "cold" increase in HP
HP	$kJ.kg^{-0.75}.d^{-1}$	heat production
I_{tissue}	$°C.m^2.W^{-1}$	tissue insulation
L%	%	carcass lean content
L_∞	kg	mature body lipid mass
ME_{maint}	$kJ.kg^{-0.75}.d^{-1}$	maintenance ME requirement
$ME_{maint,indep}$	same as ME_{maint}	ME_{maint} independent from protein turnover
ME_{turn}	same as ME_{maint}	protein turnover ME requirement
P_∞	kg	mature body protein mass
P_{dep}	$g.d^{-1}$	protein deposition rate
$P_{dep,max}$	$g.d^{-1}$	potential protein deposition rate
RHP	same as HP	required "hot" reduction in HP
$R_{L\infty/P\infty}$	$kg.kg^{-1}$	ratio of L_∞ to P_∞
T_{env}	°C	environmental temperature
T_{LC}	°C	lower critical temperature
T_{UC}	°C	upper critical temperature

Proper input variance components were determined using "best guess" methodology.

R.M. Enns (1995) *Simulation of across-breed comparisons for direct and maternal weaning weight in beef cattle.* PhD thesis, Colorado State University, Fort Collins; p. 54.

Chapter 5 is based on

P.W. Knap and H. Jørgensen (2000) Animal-intrinsic variation in the partitioning of body protein and lipid in growing pigs. *Animal Science* **70**:29-37

Chapter 5

Variation in protein and lipid partitioning

Body composition in the pig, and its variation, is mostly referred to in terms of body protein and lipid content of the whole body. This study was made to check for animal-intrinsic variation in the partitioning of body protein into protein pools and of body lipid into lipid depots. Results from serial slaughter trials on 316 Danish Landrace and 76 Danish Yorkshire pigs were used to estimate additive genetic and litter-associated variance components for several traits. These traits were total body protein and lipid mass (TOTPROT and TOTLIPD), the proportions of total body protein that are present in the muscles (PROTMUS) or in the (sub-)cutaneous tissue plus bones (connective tissue protein, PROTCON), and the proportions of total body lipid that are present in the (sub-)cutaneous tissue (LIPDSUB), in the muscles (inter- and intramuscular fat, LIPDMUS), or in the bones (LIPDBON). TOTPROT and TOTLIPD were adjusted by regression for body weight; PROTMUS and PROTCON were adjusted for TOTPROT; and LIPDSUB, LIPDMUS and LIPDBON were adjusted for TOTLIPD. The pooled estimates (± standard errors) of the degree of genetic determination (the sum of the additive genetic and litter-associated variance components, which approximates the repeatability) of these traits were 0.48 ± 0.19 for TOTPROT, 0.56 ± 0.20 for TOTLIPD, 0.56 ± 0.12 for PROTMUS, 0.57 ± 0.15 for PROTCON, 0.32 ± 0.10 for LIPDMUS, 0.33 ± 0.12 for LIPDSUB, and 0.22 ± 0.10 for LIPDBON. It is concluded that there is animal-intrinsic variation in partitioning of body protein and lipid.

Introduction

This study attempts to quantify between-animal variation in the partitioning of body protein and lipid mass. In Chapters 1 and 3, literature data were used to parameterise a model to simulate tissue growth in pigs. This was done in terms of the partitioning of body protein into pools such as muscle and connective tissue, and in terms of the partitioning of body lipid into depots such as (sub-)cutaneous tissue. These data were used to express the proportion of body protein and lipid that is present in those pools or depots as a function of total body protein or lipid mass, respectively. In order to do so, the proportions were regressed on the natural logarithm of protein or lipid mass; the observed residual variation around the regression lines was used to parameterise a stochastic simulation model in terms of the between-animal variation in the partitioning of protein into these pools (Chapter 2) and lipid over these depots (Chapter 4). Examples of the relations found are given in Figure 1. The trend lines shown are from the fit of logarithmic and cubic regression lines.

When the journal articles associated with Chapters 2 and 4 were being reviewed it was suggested that this residual variation could at least in part be regarded as the result of analytical errors of nitrogen and lipid measurement in the experiments that led to the data used, rather than as the result of true animal-intrinsic variation. The purpose of the present study is to check whether there does exist systematic variation between individual animals in the partitioning of their body protein and lipid mass into pools and depots.

The parameter of actual interest is then the repeatability of the deviations from the regression lines in Figure 1. A high repeatability would mean that animals with positive deviations early in life (at a low total body protein or lipid mass) tend to have positive deviations later in life as well, and *vice versa*. Repeatabilities of protein and lipid partitioning are difficult to obtain because their estimation requires repeated measurements of protein and lipid mass per animal, which would only be possible with expensive techniques like computerised tomography or nuclear magnetic resonance imaging (see Allen, 1990). In this study we estimate heritabilities and litter-associated variance components of protein and lipid partitioning traits from results of earlier performed dissection trials to approximate the repeatabilities of these traits.

Materials and methods

Data

The analysed data set was produced in a series of experiments carried out in 1982 and 1983 and previously reported upon by Jørgensen *et al.* (1985ab) and Just *et al.* (1985). These trials were carried out in order to "compare the anatomical and chemical composition of female pigs and barrows of Danish Landrace fed diets varying widely in chemical composition" and to determine "whether the current standards for essential nutrients per feed unit for pigs were sufficient for *ad libitum* fed [Landrace and Yorkshire] pigs", respectively. These trials are serial slaughter trials: each pig has been measured just once, after slaughter. This makes it impossible to estimate repeatabilities. But it was possible to document the distribution of the animals over full-sib groups (that is, it could be determined which animals were full sibs of each other), and partly retrace the ancestry of their parents. This enables the estimation of variance components due to genetic and litter environmental effects, which may serve as an approximation of the repeatability.

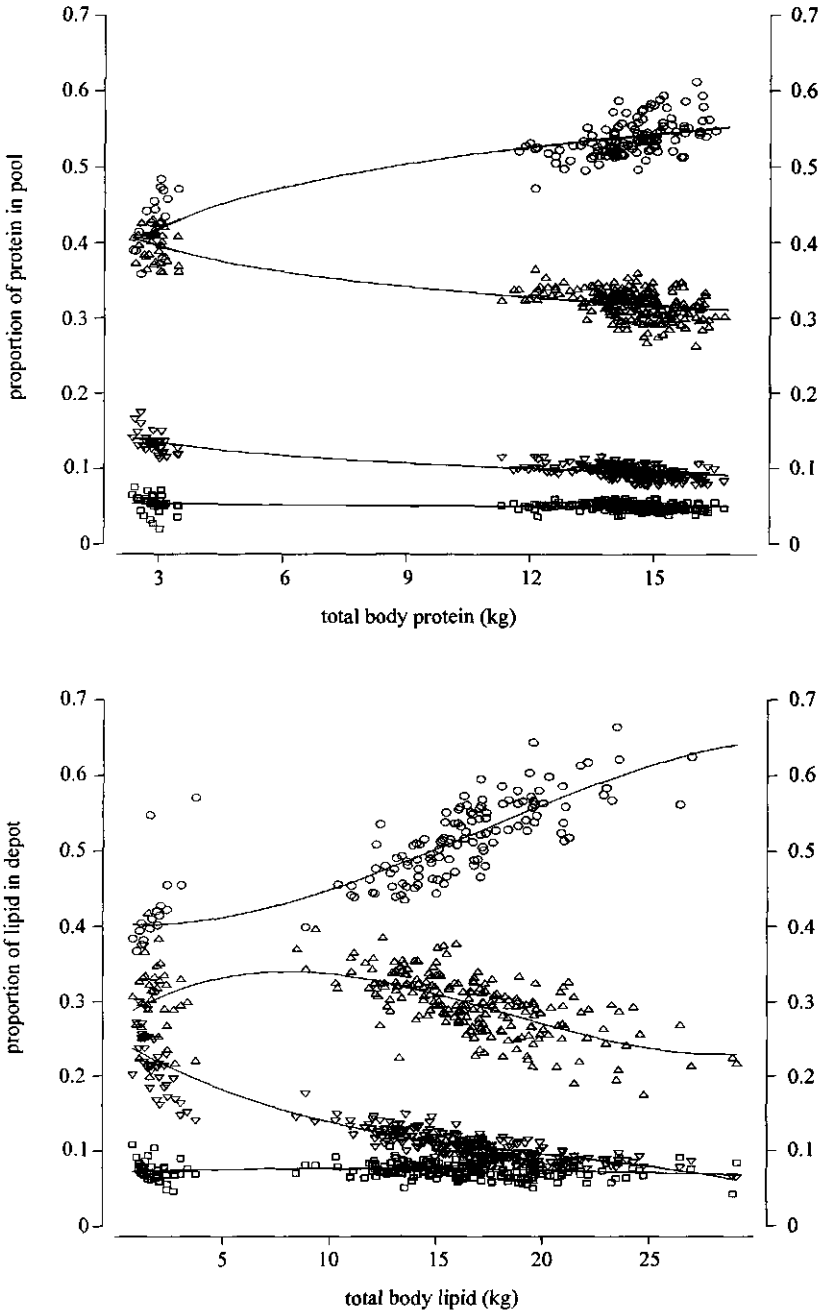


Figure 1 The course of the proportions of body protein (upper plot) present in muscle (○), connective tissue (Δ), viscera (▽) and blood (□), and of the proportions of body lipid (lower plot) present in (sub-)cutaneous tissue (○), muscle (Δ), bone (▽) and viscera (□) in growing pigs. Data from Jørgensen *et al.* (1985a). The trend lines are from the fit of logarithmic (upper plot) and cubic (lower plot) regressions.

The animals in the above mentioned trials were all treated according to the same protocol (see Jørgensen *et al.*, 1985ab and Just *et al.*, 1985). Pigs were recruited from Danish breeding farms at a liveweight between 14 and 18 kg, and housed in groups in the experimental facilities for a few days until entering the experiments at less than 20 kg liveweight. Subsequent housing was in individual crates with feeding to scale in order to obtain equal growth rates independent of dietary treatment (n=244; Jørgensen *et al.*, 1985a), or with feeding to appetite (n=148; Jørgensen *et al.*, 1985b; Just *et al.*, 1985). Animals were sacrificed either at the start of the experiment (around 20 kg liveweight; n=60) to determine initial parameters, or at about 90 kg liveweight (n=332). Slaughter was followed by the quantitative determination of, among other things, empty body weight, total body protein and lipid mass, and the protein and lipid mass of muscle, skin plus subcutaneous fatty tissue, and bones.

Animals were entire males (n=32), castrated males (n=145) or females (n=215), and had been recruited in full-sib (FS) groups of five to seven littermates. The Landrace data comprise 316 animals in 48 such FS groups, the ancestry of six of which could be traced back by the Danish National committee for pig breeding, health and production (S. Andersen, personal communication, 1996). The Yorkshire data comprise 76 animals in twelve FS groups, ten of which had retrievable ancestry information. The pedigree structure of these six Landrace and ten Yorkshire FS groups is illustrated in Figure 2.

The only pedigree-related information that could be made available for the remaining animals (originating from farms outside the Danish nucleus breeding system) was their FS group identification. Given the way the animals were sampled it is not possible that any two of these FS groups were produced by the same dam (A. Just, personal communication, 1999), and hence each FS group was assigned a unique dummy dam ID code. But considering the highly interconnected pedigrees in Figure 2, several of those FS groups probably descend from common sires and/or from some of the same sires as the FS families with known ancestry. We have dealt with the missing sire ID codes in two extreme ways: (i) by pooling these FS groups into single "Landrace" and "Yorkshire" sire groups, assigning the base (unknown) ancestor code for each breed as a dummy sire ID code, and (ii) by assigning a unique dummy sire ID code to each of these FS groups, as was done for the dam ID codes. These approaches are further referred to as "pooled unknown sires" and "unique unknown sires" pedigree structures, respectively.

Traits of interest

The traits analysed in the present study are total body protein and lipid mass (TOTPROT and TOTLIPD, respectively), the proportions of total body protein that are present in the muscles (PROTMUS) or in the (sub-)cutaneous tissue (fatty tissue, skin and hair) plus bones (connective tissue protein, PROTCON), and the proportions of total body lipid that are present in the (sub-)cutaneous tissue (LIPDSUB), in the muscles (inter- and intramuscular fat, LIPDMUS), or in the bones (LIPDBON). A regression was fitted to adjust TOTPROT and TOTLIPD for empty body weight (EBWT), to adjust PROTMUS and PROTCON for TOTPROT, and to adjust LIPDSUB, LIPDMUS and LIPDBON for TOTLIPD. For the partitioning traits (*i.e.*, PROTMUS to LIPDBON), the effect of these adjustments is that the statistical inference that we produce is about their *deviations from the regression lines* as illustrated in Figure 1. It is these deviations that we are primarily interested in: the working hypothesis of this study is that they are heritable and hence animal-intrinsic.

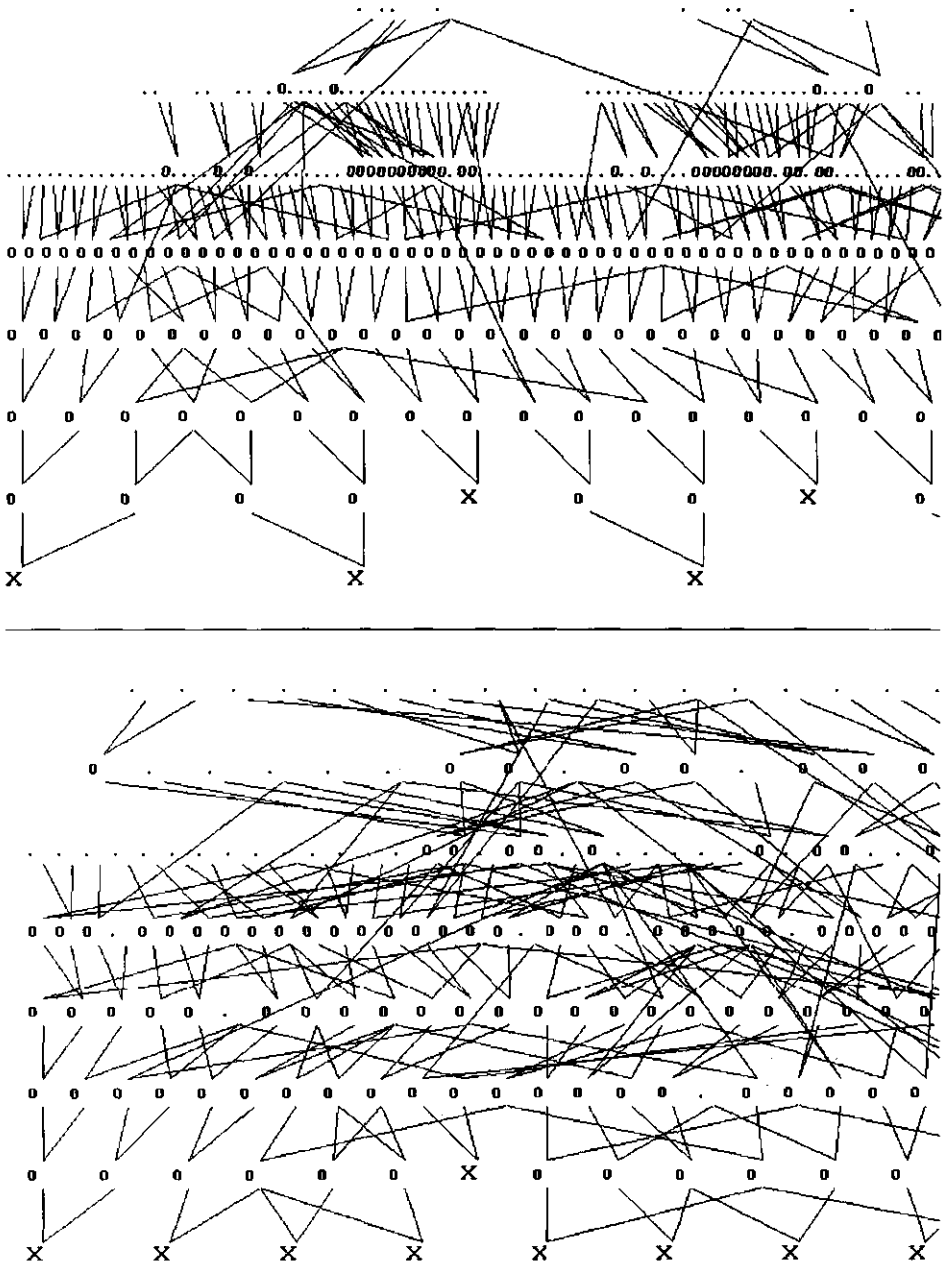


Figure 2 Pedigree structure of the full-sib groups with known grandparents. Full-sib groups are denoted by "X", ancestors by "0", base animals by a dot. Upper plot: Landrace; lower plot: Yorkshire. In addition to the ones shown here, the data contain 42 Landrace and two Yorkshire full-sib groups with unknown grandparents. These plots were made with the *PedigreeViewer* 2.4 package (Kinghorn, 1995).

Data analysis

An initial screening of the data produced the descriptive statistics in Table 1. The data fall into two distinct empty body weight groups (14.2 to 22.4 *versus* 62.5 to 93.2 kg), which is also discernible in body protein and lipid mass (2.4 to 3.9 *versus* 11.5 to 16.8 kg body protein, and 0.7 to 3.7 *versus* 7.8 to 29.1 kg body lipid). In Chapters 1 and 3 we obtained a satisfactory fit of the data by Jørgensen *et al.* (1985a; a subset of the present data) as well as of four other data sets, by applying logarithmic regression of the protein and (sub-)cutaneous lipid proportions on total protein or lipid mass. This still holds for the protein proportions in the present data, but the muscle lipid proportions were described much better with a third-degree polynomial, so that model was applied to all lipid depots. The RSD values in Table 1 are the residual standard deviations after adjustment of the data for the effects of breed, sex, and experimental batch and for the effects of either EBWT, TOTPROT, or TOTLIPD, by models (1), (2) or (3) described below. For the partitioning traits, these RSD values quantify the variation of the deviations from the regression lines. The residual coefficients of variation (RSD divided by the associated average value) are lower for the lipid-related traits (between 0.03 and 0.04 for TOTLIPD, LIPDMUS, LIPDSUB, and LIPDBON) than for the protein-related traits (between 0.07 and 0.16 for TOTPROT, PROTMUS, and PROTCON), as would be expected. Most RSD values, and all residual coefficients of variation, are lower in the Yorkshire than in the Landrace although they all follow the same pattern in both breeds.

Table 1 Distribution statistics of the analysed traits. Upper entries: Landrace, lower entries: Yorkshire.

trait	min	max	average	stdev†	RSD‡
EBWT (kg)	14.2	90.3	72.8	23.7	
	15.2	93.2	72.9	24.2	
TOTPROT (kg)	2.38	16.81	12.82	4.27	0.55
	2.60	16.76	12.97	4.36	0.48
TOTLIPD (kg)	0.80	29.12	14.08	6.15	2.23
	0.74	24.03	11.80	5.19	1.82
PROTMUS ($kg.kg^{-1}$)	0.358	0.626	0.527	0.051	0.017
	0.440	0.597	0.548	0.043	0.016
PROTCON ($kg.kg^{-1}$)	0.243	0.426	0.318	0.035	0.014
	0.259	0.391	0.300	0.029	0.012
LIPDMUS ($kg.kg^{-1}$)	0.175	0.467	0.315	0.055	0.029
	0.275	0.460	0.383	0.040	0.029
LIPDSUB ($kg.kg^{-1}$)	0.253	0.679	0.485	0.076	0.034
	0.269	0.570	0.400	0.047	0.029
LIPDBON ($kg.kg^{-1}$)	0.065	0.374	0.125	0.046	0.016
	0.088	0.270	0.148	0.038	0.016

†standard deviation of unadjusted data

‡residual standard deviation after fitting models (1), (2) or (3)

As a first check of the statistical significance of animal-intrinsic effects, the data were subjected to analysis of variance by making use of the MIXED procedure of SAS (1992) to fit the following statistical models to the data by REML (restricted maximum likelihood):

$$y = \mu + [breed \times sex] + b_1 \times \text{EBWT}:[breed \times sex] + [breed \times batch] + FSgroup:[\text{breed} \times batch] + residual \quad (1)$$

for TOTPROT and TOTLIPD,

$$y = \mu + [breed \times sex] + b_2 \times \ln(\text{TOTPROT}):[breed \times sex] + [breed \times batch] + FSgroup:[\text{breed} \times batch] + residual \quad (2)$$

for PROTMUS and PROTCON, and

$$y = \mu + [breed \times sex] + b_3 \times \text{TOTLIPD}:[breed \times sex] + b_4 \times (\text{TOTLIPD})^2:[breed \times sex] + b_5 \times (\text{TOTLIPD})^3:[breed \times sex] + [breed \times batch] + FSgroup:[\text{breed} \times batch] + residual \quad (3)$$

for LIPDSUB, LIPDMUS and LIPDBON.

Here y denotes the dependent trait; μ denotes its overall mean; [*breed* × *sex*] is the fixed interaction effect of breed and sex with six classes; covariates are EBWT, the natural logarithm of TOTPROT, or TOTLIPD and its 2nd and 3rd powers with regression coefficients b_1 to b_5, nested within [*breed* × *sex*]; [*breed* × *batch*] denotes the fixed interaction effect of breed and experimental batch with ten classes (there were eight batches, but Yorkshire data were recorded in two batches only); *FSgroup* denotes the random effect of the FS group, nested within [*breed* × *batch*]; and *residual* denotes the residual error not accounted for by the model. The *FSgroup* terms are assumed to have equal variance and zero covariance among each other; that is, their covariance matrix is diagonal. The same holds for the *residual* terms.

Because of the small size of the data sets and the incomplete pedigree information, the data of the two breeds were pooled and analysed together. Although this allows for a much more robust adjustment for fixed effects and covariates, it assumes that the variances of the random components of the statistical models are the same for the two breeds. Likelihood ratio tests showed that the residual variance estimates from preliminary within-breed analyses were not significantly different between the breeds ($0.15 < P < 0.9$) for any of the traits (although most of the Yorkshire estimates are lower, see the RSD values in Table 1). The same holds for the within-breed *FSgroup* variance estimates for TOTLIPD, PROTMUS, and PROTCON ($P > 0.8$). Within-breed *FSgroup* variance estimates for the other traits were more difficult to compare due to their instability in the Yorkshire breed: the analysis failed to produce a meaningful estimate for LIPDMUS in the Yorkshire (no convergence), and for TOTPROT, LIPDSUB and LIPDBON it produced extremely skewed confidence intervals that completely encompassed the corresponding intervals of the Landrace estimates.

The significance tests for the *FSgroup* effect and its associated variance components from models (1) to (3) quantify the differences among FS groups in each of the seven traits after adjustment for the effects of breed, sex, and experimental batch and the effects of EBWT, TOTPROT, or TOTLIPD, *i.e.* the animal-intrinsic variation of the regression deviations as far as it can be quantified under the assumption of absence of covariance between FS groups.

In the above analyses, animal-intrinsic variation was quantified in terms of the resemblance among littermates (full-sibs), but the pedigree structure shown in Figure 2 provides further information on genetic relationships *between* FS groups (*i.e.* among half-sibs, cousins, etc.) that allows for a more detailed quantification of the animal-intrinsic variation. In order to do so, variance components were estimated for the additive genetic effect, and for the litter-associated effect, on the seven traits according to the following statistical models:

$$y = \mu + [breed \times sex] + b_1 \times \text{EBWT}:[breed \times sex] + [breed \times batch] + litter + animal + residual \quad (4)$$

for TOTPROT and TOTLIPD,

$$y = \mu + [breed \times sex] + b_2 \times \ln(\text{TOTPROT}):[breed \times sex] + [breed \times batch] + litter + animal + residual \quad (5)$$

for PROTMUS and PROTCON, and

$$y = \mu + [breed \times sex] + b_3 \times \text{TOTLIPD}:[breed \times sex] + b_4 \times (\text{TOTLIPD})^2:[breed \times sex] + b_5 \times (\text{TOTLIPD})^3:[breed \times sex] + [breed \times batch] + litter + animal + residual \quad (6)$$

for LIPDSUB, LIPDMUS and LIPDBON.

Apart from the terms already defined in models (1) to (3), *litter* in models (4) to (6) denotes the random effect of the common litter environment, and *animal* denotes the random additive genetic effect. Because of the explicit inclusion of the *animal* effect, the *litter* effect in models (4) to (6) is more specific than the *FSgroup* effect in models (1) to (3) although it is coded for by the same litter ID values. The *FSgroup* effect represents all litter-associated variation, that is the additive, dominance, and epistatic genetic effects and any maternal and other common environmental effects; in contrast, the *litter* effect does not comprise the additive genetic effects as these are dealt with by the *animal* effect.

As in the above REML analyses, the *litter* terms are assumed to have equal variances and zero covariances; that is, their covariance matrix is diagonal. The same holds for the *residual* terms. The *animal* terms are characterised by a non-diagonal covariance matrix which is a multiple of the matrix of additive genetic relationships among the animals in the analysis (0.5 among full sibs or between parents and progeny, 0.25 among half sibs, etc.).

Models (4) to (6) were fitted with the maGGic 1.1 package (Janss, 1995); this program estimates variance components for the random effects in the model (*litter*, *animal*, and *residual*, in our case) by means of Gibbs Monte-Carlo Markov-chain sampling methodology (see Sorensen *et al.*, 1994, for a short introduction). Seven separate univariate analyses were conducted, each one with both the "pooled unknown sires" and the "unique unknown sires" pedigree structures, as described in the *Data* section above.

For each trait we have broadly followed the procedure suggested by Janss *et al.* (1997) for estimation of polygenic variance by (i) generating ten Markov chains with non-informative prior parameters, each chain of sufficient length to yield 48 "virtually independent" samples from the distributions of the *residual*, *animal*, and *litter* variances (which involved sampling at intervals of 400 to 1150 samples, as determined in an initial 100,000 samples chain) after discarding the initial 20 % of the chain to allow it to stabilise ("burn-in"), (ii) determining convergence of the Gibbs sampler by checking for absence of significant chain effects in a simple analysis of variance, and (iii) "post-analysing" the resulting 480 samples by calculating the mean and standard deviation of the variances (which may loosely be interpreted as the variance estimate and its standard error) and plotting their frequency distribution (the "posterior density"). The

latter provides another impression of the significance of the estimates: a distribution with a mode close to zero suggests non-significance; with symmetric distribution of the samples within the permissible parameter space (from zero to unity for variance components), the mean value is asymptotically equal to the mode, and the mode is equivalent to the REML estimate of the same variance.

The *animal* and *litter* variance components can be summarised in terms of the heritability $h^2 = \frac{\sigma_A^2}{\sigma_A^2 + \sigma_L^2 + \sigma_e^2}$ and the litter-associated component $c^2 = \frac{\sigma_L^2}{\sigma_A^2 + \sigma_L^2 + \sigma_e^2}$, where σ_A^2, σ_L^2 and σ_e^2 are the additive genetic, litter-associated and residual variances, respectively. The total genetic proportion of the variance of each trait is then approximated (overestimated, because of the maternal and other common environmental effects) by the "degree of genetic determination" $I^2 = h^2 + c^2$. This parameter ("I" for "individual") is similar to the "broad-sense heritability" as it was defined by Lush (1940) and further detailed by Falconer (1960); it quantifies the animal-intrinsic variance that we are interested in for our present purposes.

Results

Models (1) to (3) revealed highly significant ($P < 0.003$) contributions of the *FSgroup* effect to the overall variation of the data. The associated REML variance component estimates (column 2 of Table 2) are around 0.1 for TOTPROT, TOTLIPD and LIPDBON, 0.2 for LIPDMUS and LIPDSUB, 0.3 for PROTCON, and 0.4 for PROTMUS. Hence we may conclude that the data show significant FS group-associated (and hence animal-intrinsic) variance for each of the seven traits analysed, most clearly so for the body protein partitioning traits.

Table 2 REML estimates of *FSgroup* variance components, and Gibbs estimates of heritabilities (h^2), common litter-environmental components (c^2) and degrees of genetic determination (I^2) ± standard errors in the pooled Landrace and Yorkshire data.

		unique unknown sires†			pooled unknown sires‡		
trait	*FSgroup*	h^2	c^2	I^2	h^2	c^2	I^2
TOTPROT	0.110	0.181 ± 0.122	0.081 ± 0.057	0.262 ± 0.101	0.412 ± 0.208	0.068 ± 0.050	0.480 ± 0.188
TOTLIPD	0.121	0.216 ± 0.126	0.078 ± 0.058	0.294 ± 0.104	0.502 ± 0.227	0.060 ± 0.052	0.561 ± 0.204
PROTMUS	0.393	0.590 ± 0.223	0.137 ± 0.109	0.727 ± 0.142	0.212 ± 0.164	0.348 ± 0.090	0.560 ± 0.121
PROTCON	0.307	0.537 ± 0.204	0.112 ± 0.091	0.649 ± 0.144	0.326 ± 0.215	0.242 ± 0.100	0.568 ± 0.155
LIPDMUS	0.218	0.237 ± 0.157	0.118 ± 0.079	0.355 ± 0.114	0.142 ± 0.124	0.173 ± 0.072	0.315 ± 0.101
LIPDSUB	0.200	0.212 ± 0.144	0.133 ± 0.078	0.344 ± 0.109	0.174 ± 0.140	0.161 ± 0.071	0.334 ± 0.115
LIPDBON	0.113	0.109 ± 0.088	0.062 ± 0.045	0.171 ± 0.079	0.155 ± 0.114	0.066 ± 0.046	0.220 ± 0.105

† full-sib groups with unknown sires were assigned unique dummy sire ID codes

‡ full-sib groups with unknown sires were assigned the base Landrace or Yorkshire ancestor code as a pooled dummy sire ID

Columns 3 to 5 of Table 2 give the Gibbs variance component estimates and their "standard errors" (*i.e.*, the means and standard deviations of each of the seven sets of 480 samples mentioned in the previous section), as estimated with the "unique unknown sires" pedigree structure (see the *Data* section in **Materials and methods**). None of the c^2 estimates differs sig-

nificantly from zero nor, in most cases, do the h^2 estimates, although they show the same pattern as the REML *FSgroup* variance components. The I^2 estimates follow that same pattern too, but they are considerably larger than the REML *FSgroup* estimates, ranging from 0.17 for LIPDBON to 0.73 for PROTMUS. In addition, all these I^2 variance components are significantly different from zero, strongly so for the body protein partitioning traits.

The corresponding estimates from the "pooled unknown sires" pedigree structure are in columns 6 to 8 of Table 2. Some of the c^2 estimates are significantly different from zero, as are the h^2 estimates for TOTPROT and TOTLIPD. The I^2 estimates for PROTCON and for the body lipid partitioning traits are very close to those obtained with the "unique unknown sires" pedigree structure, but those for TOTPROT and TOTLIPD are considerably higher (with much larger standard errors) and the one for PROTMUS is somewhat lower.

Figure 3 shows the density distributions of the I^2 estimates obtained with the two approaches for dealing with unknown sire IDs. These plots confirm the inferences based on the estimates in Table 2.

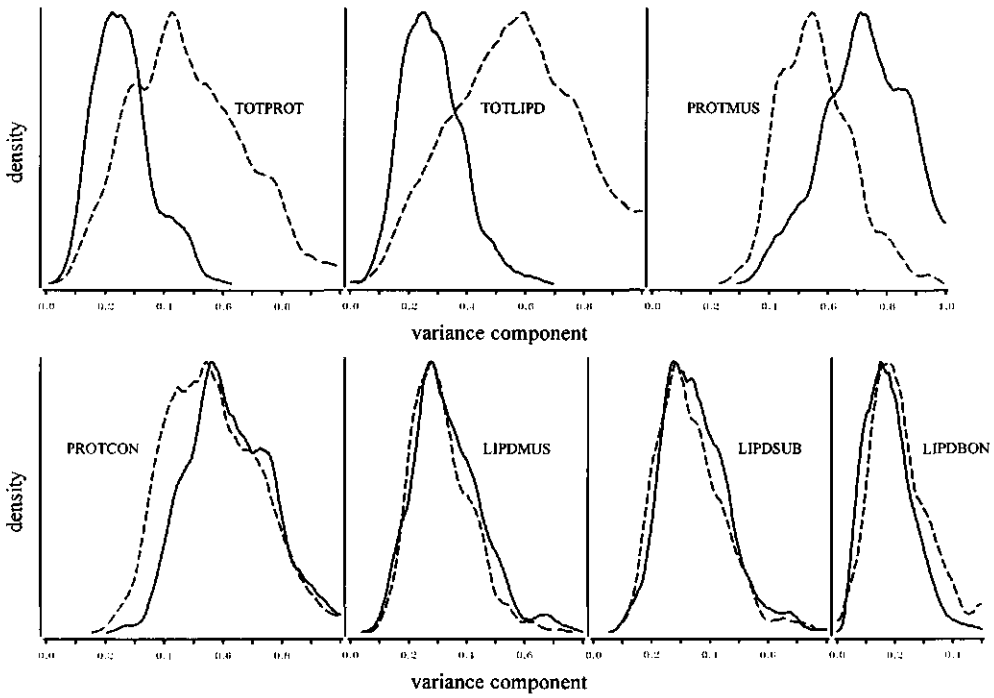


Figure 3 Frequency distributions (Gibbs posterior densities) of the I^2 estimates in the pooled Landrace and Yorkshire data, according to two approaches to deal with unknown sires of full sib groups (——: unique unknown sires; – – –: pooled unknown sires, see Table 2). The scale of the vertical axes is arbitrary, each plot has been stretched to reach the same maximum value.

Discussion

Data

The data set analysed in this study does not satisfy the requirements of a demanding statistical process such as variance component estimation: the number of observations is very limited, and the available pedigree information is incomplete, especially so for the Landrace pigs (the largest subset). But at the same time this is by far the largest data set of its kind currently available, and it is for this reason that publication of our results was considered justified. It seems unlikely that a more suitable data set will be created in the foreseeable future. This is unfortunate because our results have to be interpreted with caution; although they seem quite straightforward (see the *Interpretation of the estimates* section below), there is a strong need for independent confirmation.

Relation of the I^2 estimates to repeatability

We have quantified the proportion of variation between individual measurements of the partitioning traits that is animal-intrinsic, as opposed to that from measurement error. From a physiological point of view, the parameter of interest is the repeatability of the partitioning traits. Repeatabilities reflect the magnitude, as a ratio, of the animal-intrinsic variance of a trait, that is its genetic (additive plus dominance plus epistatic) variance plus variance due to permanent environmental effects (*i.e.* effects that exert their uniform influence on the animal during the whole trajectory of interest).

The expectation of the repeatability is then $\frac{\left[\sigma^2_A + \sigma^2_D + \sigma^2_{GI}\right] + \sigma^2_{Ep}}{\sigma^2_A + \sigma^2_D + \sigma^2_{GI} + \sigma^2_M + \sigma^2_{Ec} + \sigma^2_{Ep} + \sigma^2_e}$ whereas the realisation of I^2 can be written as $\frac{\left[\sigma^2_A + \sigma^2_D + \sigma^2_{GI}\right] + \sigma^2_M + \sigma^2_{Ec}}{\sigma^2_A + \sigma^2_D + \sigma^2_{GI} + \sigma^2_M + \sigma^2_{Ec} + \sigma^2_{Ep} + \sigma^2_e}$ in this particular data set. The terms in these equations denote the variances due to additive genetic effects (σ^2_A), genetic dominance effects (σ^2_D), genetic interaction effects (epistasis; σ^2_{GI}), maternal effects (σ^2_M), other common litter environmental effects (σ^2_{Ec}), permanent environmental effects (σ^2_{Ep}), and temporary environmental effects not accounted for by the other terms (σ^2_e). With a more favourably structured data set it might have been possible to obtain an estimator for I^2 without σ^2_M and σ^2_{Ec} in its numerator.

Thus the repeatability would exceed our I^2 estimate by the factor $\sigma^2_{Ep} - [\sigma^2_M + \sigma^2_{Ec}]$. Considering that our data were obtained from pigs that were separated from their littermates and housed individually at a liveweight of about 20 kg, σ^2_{Ec} can be expected to be small, especially at the higher liveweights. The role of maternal effects in lean- and fat-related production traits in growing pigs, although potentially significant in crosses between breeds (*e.g.* Bereskin, 1983; McLaren *et al.*, 1987), has been found to be limited within breeds (*e.g.* Steindel and Duniec, 1978; Lodde *et al.*, 1983; Van der Steen, 1983); hence we would expect σ^2_M to be small as well, again especially at the higher liveweights. It is difficult to envisage the mechanism through which any permanent environmental factors might cause the protein and lipid partitioning traits to differ between animals in uniform controlled laboratory conditions, and to speculate on the magnitude of σ^2_{Ep}. The above factor $\sigma^2_{Ep} - [\sigma^2_M + \sigma^2_{Ec}]$ may then be expected to be small, and our

I^2 estimates are likely to be fair approximations of the corresponding repeatabilities.

Interpretation of the estimates

The variance component estimates for TOTPROT and TOTLIPD provide an opportunity to compare the present results with the literature, which is the main reason why these traits were included in this study. The genetic variance of lean- and fat-related production traits is well-established in pigs and other species; for example, Stewart and Schinckel (1988) reviewed a large number of literature sources and report h^2 estimates centering around 0.41, 0.52, 0.48, and 0.47 for ultrasonic and carcass fat depth, lean percentage, and *m. longissimus dorsi* area in growing pigs. More specifically, Andersen and Vestergaard (1984) analysed the performance test data recorded from 1975 to 1982 in the Danish Landrace and Yorkshire breeding populations that our pigs had been sampled from, and report h^2 estimates for lean percentage and ultrasonic fat depth between 0.43 and 0.87. Hence bearing in mind that our "physiological" traits should be less sensitive to environmental factors than those production traits, one would expect I^2 estimates for TOTPROT and TOTLIPD of about 0.5, if not higher. Our estimates obtained with the "pooled unknown sires" pedigree structure agree with that expectation. The true nature of the data must be somewhere between this structure and the "unique unknown sires" pedigree structure, which produced much lower I^2 estimates for TOTPROT and TOTLIPD. This would suggest that the "pooled unknown sires" pedigree structure provides the better reflection of the truth. The two approaches differ further only in their estimation of the PROTMUS I^2; the estimate obtained with the "pooled unknown sires" pedigree is more in line with the estimates for the other partitioning traits, which may point into the same direction.

The difference between the Gibbs I^2 and the REML *FSgroup* variance component estimates should largely be due to the use of pedigree information. Making this information available has increased the analysis's power of detecting animal-intrinsic variation for all traits involved in this study.

In spite of the severe limitations of the data, the conclusion from this study as a whole is straightforward: the deviations from the trend lines as illustrated in Figure 1 are indeed partly animal-intrinsic, rather than just caused by experimental error.

Acknowledgements

Søren Andersen provided us with a six-generation pedigree for pigs that lived about fifteen years ago. Julius van der Werf, Steve Bishop and Ron Lewis made valuable comments on subsequent versions of the text.

Selecting for a slower decline in energy retention seems an attractive alternative to selecting for cold-bloodedness in domestic animals, with the possible exception of pigs and poultry under intensive management conditions.

S. Stephens (1991) *Biological aspects of feeding and growth in mice*. PhD thesis, University of New England, Armidale; p 17.

Chapter 6 is based on

P.W. Knap (2000) Time trends of Gompertz growth parameters in "meat-type" pigs. *Animal Science* **70**:39-49

Chapter 6

Time trends of Gompertz growth parameters

Previously published data from serial slaughter trials on growing pigs of five genotypes were re-analysed. Gompertz curves were fitted to body protein and lipid mass in order to estimate mature protein and lipid mass (P_∞, L_∞) and the rate parameter (B_{Gomp}) that was presumed to be equal for the protein and lipid curves. L_∞ was expressed as its ratio to P_∞, $R_{L\infty/P\infty}$. The maximum rate of protein deposition was derived as $P_{dep,max} = P_\infty \times B_{Gomp} / e$. The analysed data encompass body weights of 10 to 133 kg, 13 to 217 kg, 18 to 106 kg, 20 to 110 kg, and 11 to 145 kg. The Gompertz function fitted all data sets well, as judged by the standard deviations and distribution patterns of the residual terms. Autocorrelations among the residuals were non-significant.

Averaged over sexes (females and entire and castrated males), the P_∞ estimates were all close to 31 kg; the $R_{L\infty/P\infty}$ estimates ranged from 1.4 to 4.7 $kg.kg^{-1}$; the B_{Gomp} estimates ranged from 0.009 to 0.017 $kg.d^{-1}.kg^{-1}$. The resulting $P_{dep,max}$ estimates ranged from 110 to 193 $g.d^{-1}$. The genotypes were placed in 1969, 1976, 1984, 1990 and 1993. Plotting the estimates against time (year) showed distinct time trends for all parameters except P_∞. $R_{L\infty/P\infty}$ seems to gradually decline towards a plateau around unity, whereas B_{Gomp} and $P_{dep,max}$ increase linearly. These trends were confirmed by an analysis of body weight based on the same data plus data on three other genotypes that spanned the same time period. Analyses of the same protein and lipid data to fit a sigmoid growth function with a flexible point of inflection did not change the apparently absent time trend of P_∞. The estimates of the inflection points of the fitted protein accretion curves, expressed as proportions of P_∞, were indistinguishable from the fixed 0.368 value of the Gompertz function for the earliest three genotypes and then showed a tendency to increase, up to 0.46 for the 1993 population.

These time trends must be the consequence of a combination of changes in nutritional and other environmental factors and genetic changes. They cannot be the sole result of within-line selection for growth and body composition traits, since this should increase P_∞. It seems as if pig breeders have repeatedly initiated their sire lines from genetic resources with small mature size, to subsequently increase this trait as an indirect result of within-line selection.

Introduction

Since Whittemore and Fawcett (1974) published their first attempts to simulate the growth processes of the pig, several models have been developed that summarise the growing pig's genotype in terms of a few parameters (see Moughan *et al.*, 1995, and Baldwin and Sainz, 1995). The successful simulation of the growth performance of a particular pig depends as much on a correct parameterisation of its genotype parameters as on a detailed description of its (nutritive, climatic, health-related, etc.) environment. Most Whittemore-derived growth models are largely based on two genotype parameters: the maximum rate of daily protein deposition ($P_{dep,max}$) and the minimum ratio between daily lipid and protein deposition, both assumed to be constant between 20 and 100 kg body weight. An alternative approach is that of Emmans (1988), which uses three genotype parameters: mature body protein mass (P_∞), mature body lipid mass (L_∞), and the rate parameter B_{Gomp} of the Gompertz curves that potential body protein growth and desired body lipid growth are presumed to follow. L_∞ is more conveniently expressed as its ratio to P_∞ ($R_{L\infty/P\infty}$).

The actual value of all these parameters is critically important for model output. Although pig breeding has resulted in significant genetic change in growth and body composition traits over the last few decades, little attention has been paid to the change of model genotype parameters over time. As a consequence, growth simulations are often carried out without detailed knowledge of the status of the genotype under concern. In the present study, five pig populations that were reported upon between 1971 and 1994 have been characterised in terms of the above mentioned genotype parameters ($P_{dep,max}$, P_∞, $R_{L\infty/P\infty}$ and B_{Gomp}).

The aim of this study is to quantify the time trends of these parameters, and to provide evidence for (or against) statements such as "...it would appear that in the last two decades of positive selection for lean tissue growth rate in the pig, values for [P_∞] have risen from 35 to 50 kg" (Whittemore, 1994; p. 57) and "...it would [...] be expected that, over time, as commercial selection proceeds, [...] population mean values for [P_∞] and [B_{Gomp}] will increase, and that of [$R_{L\infty/P\infty}$] will decrease" (Emmans and Kyriazakis, 1998, p. 193).

Model

According to the potential growth rules of Emmans (1988), potential (*i.e.* not limited by the environment) body protein mass follows a sigmoid growth pattern up to its asymptotic value P_∞. Desired (*i.e.* not disturbed by nutritional imbalance) body lipid mass follows a similar pattern up to L_∞. These two sigmoid growth patterns are modeled by Gompertz functions (see model (1) in the **Methods** section) with the same single value of their rate parameter, as a consequence of which protein and lipid mass are allometrically related to each other. The rate parameter B_{Gomp} represents the specific growth rate $\frac{dy / dx}{y}$ at the curve's point of inflection.

The maximum rate of protein deposition derives from the other parameters as $P_{dep,max} = B_{Gomp} \times P_\infty / e$ (Ferguson and Gous, 1993a), where e is the base of the natural logarithm.

Data

Use was made of data described by Doornenbal (1971, 1972; further referred to as "Doornenbal"), Tullis (1981; "Tullis"), Noblet *et al.* (1994) and Quiniou and Noblet (1995; "Noblet"),

and Van Lunen (1994; "Van Lunen"). All these data had been collected in serial slaughter trials, and all pigs were reported to have been fed *ad libitum*. Apart from that fact, little information is available to assess the extent to which the various dietary conditions may have allowed the genotypes' growth potential to be fully expressed. Noblet's data points were recreated from readings from a graph (Quiniou and Noblet, 1995; figure 1) and allometric regression parameter estimates per genotype (Noblet *et al.*, 1994; table 2). The other data had been listed in the respective sources, in the case of Doornenbal and Van Lunen as means of two to four animals, in the case of Tullis as individual observations.

In the late sixties, Doornenbal measured body composition in 90 Canadian Lacombe females and castrated males, at about 10 kg BW intervals between 44 and 211 days of age (10 to 133 kg BW). The Lacombe, a synthetic based on Berkshire, Chester White and Landrace lines, was one of the first pig populations to be systematically selected for growth and body composition traits (Briggs, 1983), and Doornenbal's pigs probably represent the most advanced western "meat-type pig" genotypes available in the sixties.

In 1979, Tullis measured body composition in 42 crossbred males, females and castrated males, at five- to seven-week intervals between 52 and 332 days of age (13 to 217 kg BW). These pigs were of an "unimproved British Large White × Landrace cross" and represent the genotypes present in Britain before the modern selection programmes were initiated in the mid-seventies (C.T. Whittemore, personal communication, 1998).

Around 1990, Noblet measured body composition in 58 French Large White (LW) males, females and castrated males, at about 20 kg BW intervals between 56 and 180 days of age (18 to 106 kg BW). These pigs were "recruited from a population whose sire line had been isolated from commercial genetic selection for many years" (N. Quiniou, personal communication, 1999), and should roughly represent the western European genotypes of the mid-eighties.

In the same series of experiments, Noblet measured body composition in eight males of a synthetic sire line, at about 20 kg BW intervals between 65 and 150 days of age (20 to 110 kg BW). This synthetic had been based on Hampshire, LW, Pietrain and Duroc lines (N. Quiniou, personal communication, 1999).

In 1993, Van Lunen did the same in 60 males and females of another synthetic sire line, at 10 kg BW intervals between 55 and 223 days of age (11 to 145 kg BW). This synthetic had been based on LW, Pietrain and Landrace lines (O.I. Southwood, personal communication, 1999). Noblet's and Van Lunen's synthetic sire lines represent the most advanced "meat-type pig" genotypes available in the early nineties.

In addition, use was made of three further data sets, described by Walstra (1980; further referred to as "Walstra"), White *et al.* (1995; "White"), and Andersen and Pedersen (1996; "Andersen"). These authors did not measure body protein or lipid, but serial body weights.

Walstra measured body weight in 198 *ad libitum*-fed Dutch Landrace males, females and castrated males, at six-week intervals from 1 to 211 days of age and further at 350 days and at maturity (up to 296 kg BW). These pigs were recruited from an experimental herd that had been founded in 1966 (E.W. Brascamp, personal communication, 1999), and should represent the western European genotypes of the mid-sixties.

White measured body weight in ten *ad libitum*-fed US Yorkshire females and castrated males, at irregular intervals from 1 to 171 (castrates) and 260 (females) days of age (up to 150 kg

BW). These pigs were purchased from an Illinois Yorkshire breeder in 1990 (D.G. McLaren, personal communication, 1999). Given the fact that genetic change in growth and carcass traits has been practically absent in the US Yorkshire population up to 1989 (see Lofgren *et al.*, 1994), the genetic level of these pigs is most likely comparable to Tullis's "unimproved" genotype. This would place White's genotype in the late seventies on the horizontal axes of our time trend plots.

Andersen measured body weight in 192 *ad libitum*-fed female and castrated male pigs of a Danish [Hampshire × Duroc] × [Landrace × Yorkshire] terminal cross, twice weekly between 30 and 115 kg BW (from 10 to 100 days on-test). These pigs represent the Danish nucleus levels of 1989 (S. Andersen, personal communication, 1999).

Methods

Parameter estimation

Body protein and lipid mass (P and L, in kg) as reported by Doornenbal, Tullis, Noblet, and Van Lunen were related to age (in days) by making use of the MODEL procedure of SAS (1993) to apply a maximum likelihood routine to fit Gompertz functions to the data for each genotype and sex according to the model

$$\begin{aligned} P &= P_{\infty} \times e^{-e^{-B_{Gomp} \times (age - t_P^*)}} \\ L &= L_{\infty} \times e^{-e^{-B_{Gomp} \times (age - t_L^*)}} \end{aligned} \qquad (1)$$

where P_{∞} and L_{∞} are the asymptotic values that represent mature protein and lipid mass (in kg), and t_P^* and t_L^* denote the x-coordinates of the points of inflection of the estimated P and L curves (in days). The protein and lipid curves were forced to have equal estimates for the Gompertz rate parameter B_{Gomp} to accommodate Emmans's (1988) requirement of allometry between protein and lipid growth. $R_{L\infty/P\infty}$ is estimated as $\hat{L}_{\infty} / \hat{P}_{\infty}$, the maximum rate of protein deposition as $\hat{P}_{dep,max} = \hat{B}_{Gomp} \times \hat{P}_{\infty} / e$.

Time trends

The method described above produces parameter estimates for each genotype by sex subclass. Because the dataset is unbalanced (not all three sexes are represented in each data set), and some of the parameters must be expected to differ between sexes, the estimates were further analysed using the GLM procedure of SAS (1990b) with a simple ANOVA model comprising the effects of genotype and sex:

$$y = \mu + Genotype + Sex \qquad (2)$$

where y denotes the estimates of P_{∞}, $R_{L\infty/P\infty}$, B_{Gomp} or $P_{dep,max}$ from model (1).

This produces least-squares means for the genotypes adjusted for sex effects. To describe the time trends of the parameters, these least-squares means have been related to time (year) by placing Doornenbal's population in 1969, Tullis in 1976, Noblet's LW population in 1984, and Noblet's and Van Lunen's synthetic sire lines in 1990 and 1993, respectively. These allocations in time, although somewhat arbitrary, reflect the backgrounds of the genotypes as described in the **Data** section.

Results

The data points and the Gompertz curves fitted through these by model (1) are in Figure 1. The most striking development from Doornenbal's pigs to Noblet's and Van Lunen's synthetic sire lines is the gradual descent of the lipid growth curves towards the same level as the protein growth curves.

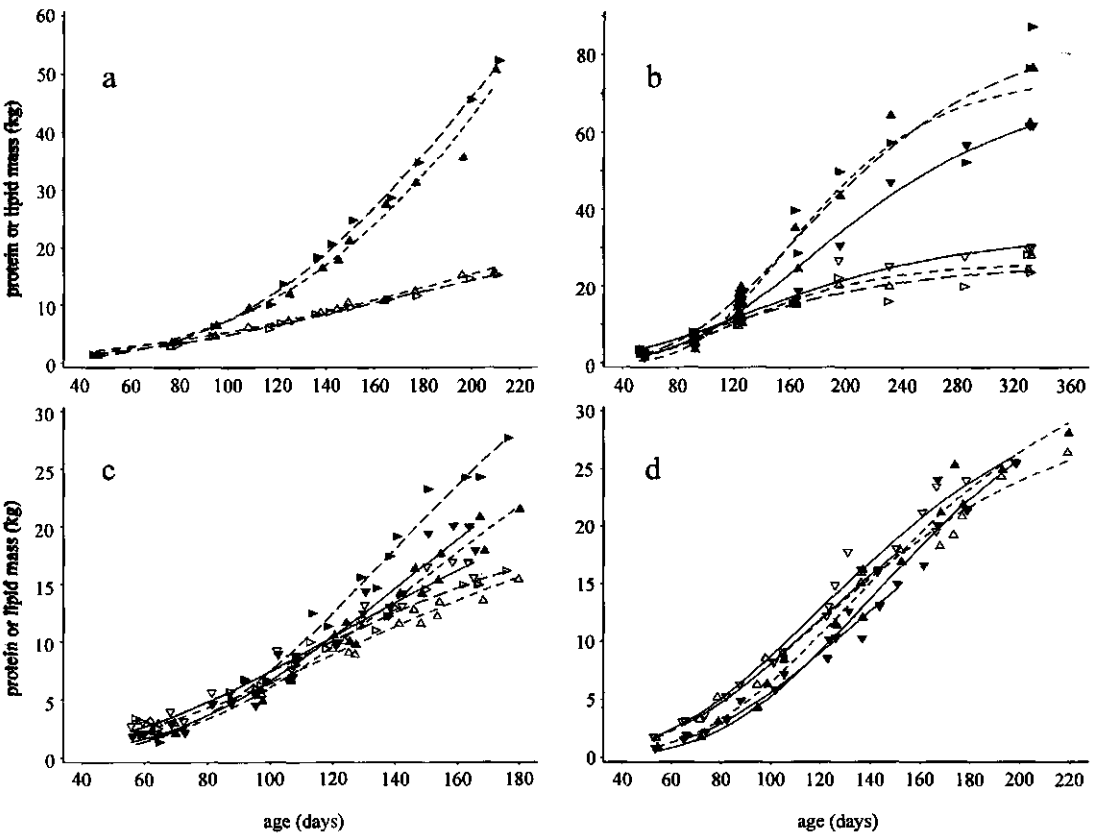


Figure 1 Body protein (open symbols) and lipid (solid symbols) mass in relation to age in serial slaughter trials with growing pigs. Scatter plots: experimental data (a: Doornenbal, 1971, 1972; b: Tullis, 1981; c: Quiniou and Noblet, 1995; d: Van Lunen, 1994 (full curves), and Quiniou and Noblet, 1995 (up to 150 days)). Line plots: Gompertz curves through scatter plots with equal growth rate parameters for protein and lipid within sex (▼——: entire males; ▲– – –: females; ►— — —: castrated males), according to model (1). See the text for details.

The associated parameter estimates are in Table 1. In Tullis's data, $1.89 \leq RSD \leq 2.40$ kg for the protein curve and $3.46 \leq RSD \leq 6.78$ kg for the lipid curve; this is the only data set with individual observations, and its residuals are spread much wider at the later ages. In the other data sets, $0.34 \leq RSD \leq 1.20$ kg for protein, and $0.36 \leq RSD \leq 2.11$ kg for lipid. In all data sets, $0.91 \leq R^2 \leq 0.99$ for protein and lipid. The sampling correlations between the P_∞ and B_{Gomp} estimates were generally strongly negative (in Tullis's data, $-0.81 \leq r \leq -0.92$; in the

Table 1 Estimates of genotype parameters (± approximate standard errors) according to model (1)

source[†]	breed[‡]	sex[§]	P_∞ (kg)	$R_{L\infty/P\infty}$ ($kg.kg^{-1}$)	B_{Gomp} ($kg.d^{-1}.kg^{-1}$)	$P_{dep,max}$ ($g.d^{-1}$)
Doornenbal	Lc	C	28.8 ± 5.8	5.16 ± 0.50	0.0093 ± 0.0016	99 ± 4
		F	31.8 ± 11.1	4.72 ± 0.80	0.0089 ± 0.0021	105 ± 12
Tullis	LWL	C	25.8 ± 2.7	3.39 ± 0.27	0.0120 ± 0.0049	113 ± 37
		F	27.6 ± 1.0	2.89 ± 0.14	0.0135 ± 0.0015	137 ± 12
		M	32.8 ± 5.8	2.13 ± 0.32	0.0121 ± 0.0034	146 ± 20
Noblet	LW	C	24.5 ± 8.2	2.22 ± 0.22	0.0141 ± 0.0037	127 ± 11
		F	30.0 ± 8.6	1.84 ± 0.19	0.0110 ± 0.0021	121 ± 11
		M	32.8 ± 18.0	1.48 ± 0.17	0.0124 ± 0.0046	149 ± 28
	S	M	38.5 ± 14.5	0.97 ± 0.28	0.0142 ± 0.0045	201 ± 25
Van Lunen	S	F	31.8 ± 3.6	1.19 ± 0.08	0.0160 ± 0.0024	187 ± 10
		M	33.2 ± 6.0	1.09 ± 0.16	0.0173 ± 0.0052	212 ± 29

[†] Doornenbal (1971, 1972); Tullis (1981); Noblet *et al.* (1994) and Quiniou and Noblet (1995); Van Lunen (1994).

[‡] Lc: Lacombe; LW: Large White; LWL: Large White × Landrace cross; S: synthetic sire line.

[§] C: castrated male; F: female; M: entire male.

other data sets, $-0.95 \leq r \leq -0.99$). The sampling correlations between the P_∞ and $R_{L\infty/P\infty}$ estimates ranged from –0.37 to +0.99, those between the $R_{L\infty/P\infty}$ and B_{Gomp} estimates from –0.99 to +0.25. Plots of the residuals in relation to age did not reveal discernible patterns for any of the data sets. Analysis of the residuals by fitting a second-order autoregressive error model with the AUTOREG procedure of SAS (1993) produced non-significant Durbin-Watson statistics for protein ($0.19 \leq P \leq 0.99$) and lipid ($0.21 \leq P \leq 0.99$) in all data sets, indicating non-significant autocorrelations among the residual terms. It may be concluded that the Gompertz function fitted the data generally very well, although it will be difficult to interpret the individual regression parameter estimates *per se*.

As expected, some parameters show significant differences among the sexes: the P_∞ estimates decrease in the order males > females > castrates ($0.04 \leq P \leq 0.06$), the $R_{L\infty/P\infty}$ estimates are higher in castrates than in males or females ($P = 0.06$) and the $P_{dep,max}$ estimates are higher in males than in castrates or females ($P = 0.05$). The least-squares means of the growth curve parameters for the five genotypes, adjusted for sex effects, from model (2) are in Figure 2. See the **Discussion** section for the trend lines in this Figure.

The data sets analysed here had not been sampled from the same genetic population. On the contrary, the genotypes involved represent a wide range of pig breeds and selection programmes, and it is even unclear to what extent each genotype is representative for the state of the art of the pig industry at its time. Each data point in Figure 2 can only provide a point estimate from a much wider range, without any information of its location within that range.

Nevertheless, the differences among the data sets reflect the industry-wide genetic change over the 25 years preceding the early nineties, in combination with the change in nutritional and other environmental conditions during the same period.

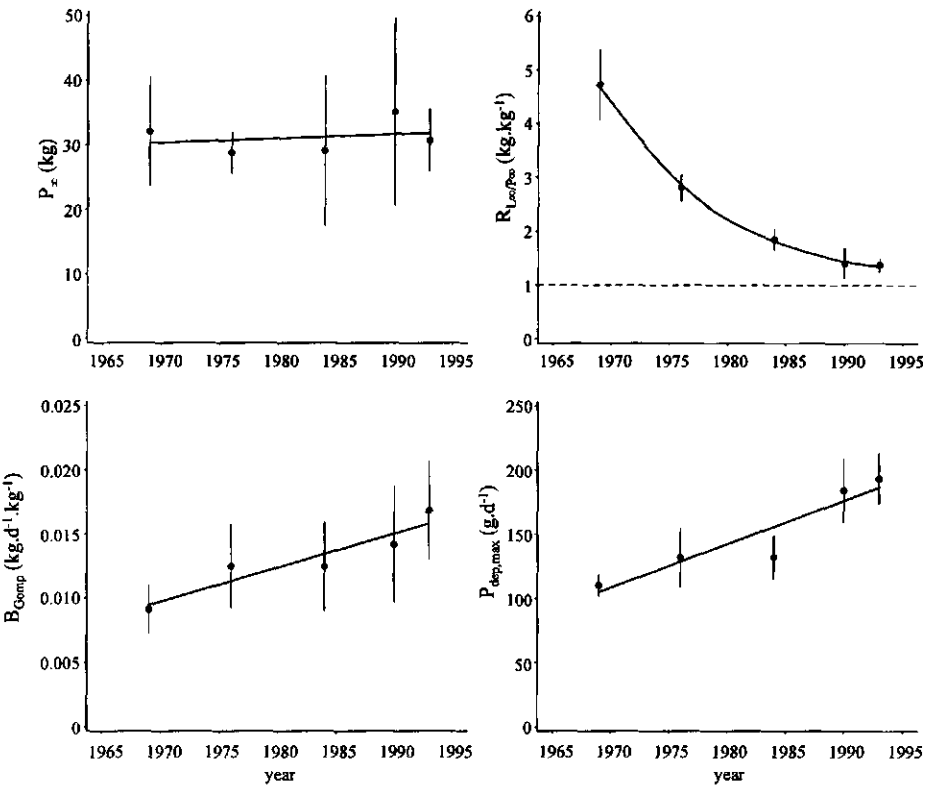


Figure 2 Time trends of mature body protein mass (P_∞), the ratio between mature body lipid and protein mass ($R_{L\infty/P\infty}$), the Gompertz growth rate parameter (B_{Gomp}), and maximum daily protein deposition ($P_{dep,max}$) as estimated for growing pigs of five genotypes. The data points are least-squares means of the within-sex estimates from Table 1, averaged over females and entire and castrated males. The vertical bars represent the average standard errors of those estimates. The trend lines are spline interpolation plots through the points shown. See the text for details.

According to our least-squares means in Figure 2, this combined change has resulted in a doubling of the growth rate parameter from B_{Gomp} = 0.009 to 0.017 kg.d^{-1}.kg^{-1} and a consequent increase of the maximum rate of protein deposition from $P_{dep,max}$ = 110 to 193 g.d^{-1}. The ratio between mature body lipid and protein mass has been reduced from $R_{L\infty/P\infty}$ = 4.7 to 1.4 kg.kg^{-1}, largely due to a drastic reduction of mature body lipid mass from 151 to 42 kg. Mature body protein mass shows little change around P_∞ = 31 kg. All these figures hold for a balanced combination of females and entire and castrated males.

Discussion

Model

To check the assumption of equal rate parameters for the protein and lipid curves, the analyses with model (1) were repeated while allowing for separate B_{Gomp} estimates. In each data set, likelihood ratio tests showed that the estimates for the two fractions were not significantly different ($0.53 \leq P \leq 0.97$). Hence these data present no reason to abandon this assumption.

Earlier analyses

Some of these datasets had been analysed earlier to estimate the same parameters as in the present study. Emmans (1988; p.160) produced "equations with the same value of the rate parameter for each component" (Emmans and Kyriazakis, 1998) for Doornenbal's castrates, resulting in estimates remarkably close to ours from model (1). However, his least-squares analysis of Doornenbal's females led to unrealistic estimates. Whittemore *et al.* (1988) fitted Gompertz curves to the weekly BW recordings in relation to age of six of Tullis's pigs (two of each sex) that had been slaughtered at the highest end weights. The within-sex estimates of mature body weight and B_{Gomp} from these analyses can be combined with the allometric coefficients that relate P and L to BW, which these authors estimated on Tullis's whole data set. This produced estimates that differ considerably from ours, which is not surprising in view of the combination of parameter estimates from different subsets of the data. In terms of more complete use of information, our estimates from Tullis's data are to be preferred. Van Lunen and Cole (1998; table 4) fitted Gompertz curves to Van Lunen's P and L recordings in relation to age, and produced estimates that deviate somewhat from ours, due to the fact that they allowed for separate B_{Gomp} estimates for P and L. Hence their estimation procedure was not consistent with the simpler model used in the present study.

Data

Emmans's (1988) rules are *potential* growth rules. That is, model (1) is supposed to represent the genotype potential, which will only be fully expressed in an entirely non-limiting environment, in terms of nutrition, climate, health and other stressors. It is unlikely that any of the data analysed here were produced in such conditions. Hence the estimates in Table 1 may deviate from the true genetic potential of the associated populations as a result of suboptimal environmental conditions. For example, a shortage of feed protein would bias the P_∞ estimate downwards and the $R_{L\infty/P\infty}$ estimate upwards, and would delay the protein accretion pattern so that the point of inflection of the protein growth curve would be shifted towards a higher proportion of P_∞. A shortage of feed energy would bias the $R_{L\infty/P\infty}$ estimate downwards and delay the lipid accretion pattern. Both conditions would bias the B_{Gomp} estimate downwards. Other environmental factors can have similar effects (*e.g.* Holck *et al.*, 1998; figure 3).

It follows that the results of the present study cannot be interpreted in terms of purely genetic trends independent from dietary or other environmental influences. Indeed, it is difficult to think of any realistic experimental setting that would allow for the unlimited expression of the growth potential of prepubertal pigs, most notably because of constraints imposed by immunological and social stressors. But a "fair" comparison of the five genotypes analysed here would have included, at the very least, a series of dietary treatments that was optimised in terms of energy and amino acid supply to match the nutrient requirements that will have changed, both over the years and within each genotype with ageing.

None of the sources from which our data were taken gives sufficient information on its experimental conditions to judge such issues. Only Van Lunen reports on the realised feed intake of his pigs. This is relevant because the estimates for this genotype are the most extreme. Van Lunen's pigs increase their daily feed intake continuously with increasing body weight over the entire course of the data. The same holds for their body lipid to protein ratio. These

data show no sign of systematic deviations of the parameter estimates as a result of nutritional deficiencies

What follows in the present discussion is an interpretation of the results of our analysis that largely ignores such possible confounding with environmental factors. That is, we attempt to interpret the data as if they fully reflect the genetic growth potential of the genotypes involved or, at least, as if all five genotypes were constrained to the same extent. Given the lack of other information this seemed to be the best that could be done with these data.

The strongly negative sampling correlations between our P_∞ and B_{Gomp} estimates from each data set (due to the data running up to an insufficient stage of maturity) make it difficult to interpret these estimates separately. For practical purposes it may be better to consider their product, *i.e.* $P_{dep,max}$. It should be kept in mind that the P_∞ and L_∞ estimates from these particular analyses are not equivalent to the mass of protein and lipid as it could be physically measured in a mature pig. Bünger and Schönfelder (1984; figure 2) showed in laboratory mice that the asymptotic values of sigmoid growth curves, as estimated from body weight measurements that run up to first mating, may underestimate the eventually realised BW by 30 %. Thompson *et al.* (1996; p. 274) noticed a similar thing in pigs.

Time trends of growth parameters

The trend lines in Figure 2 are spline interpolation plots that represent our subjective interpretation of the least-squares means. As a tentative view of the future time trends of the genotype parameters it may then be inferred that P_∞ will show a very limited increase, that $R_{L\infty/P\infty}$ seems to reach a plateau around a unity ratio between mature lipid and protein mass, and that the steady increase of B_{Gomp} and $P_{dep,max}$ shows no signs of plateauing as yet. It must be stressed that this derives from a between-genotypes analysis, reflecting the effects of a replacement of one genetic population by another. This is an entirely descriptive analysis, it does not attempt to (and cannot) explain the observed changes in the genotype parameters in terms of selective breeding for growth and body composition traits within populations. Extrapolation of the trend lines in Figure 2 towards the advanced western "meat-type pig" genotypes of the year 2005 would then forecast population means around P_∞ = 33 kg, $R_{L\infty/P\infty}$ = 1.0 $kg.kg^{-1}$ (*i.e.*, L_∞ = 33 kg), B_{Gomp} = 0.019 $kg.d^{-1}.kg^{-1}$, and $P_{dep,max}$ = 230 $g.d^{-1}$ for balanced combinations of females and entire and castrated males. This forecast is consistent with the surmise of Emmans and Kyriazakis (1998) with regard to $R_{L\infty/P\infty}$ and B_{Gomp} as quoted in the **Introduction** section.

However, Figure 2 seems to contradict the ideas of Whittemore (1994) and Emmans and Kyriazakis (1998) about the development over time of P_∞ (also quoted in the **Introduction** section). These ideas were largely based on reasoning about the mechanisms of selective breeding for growth and body composition traits *within* populations, and we have confirmed this reasoning by simulation studies (unpublished). Although the predominant strategies for selective breeding for growth and body composition traits *within* "meat-type" pig populations are likely to lead to increasing P_∞ levels within populations over time, the present study suggests that sire line development *across* populations (breeds) has *not* favoured genotypes with larger mature size. It looks as if pig breeders have consistently initiated their sire lines from genetic

resources with small mature size, to subsequently increase this trait as an indirect result of within-line selection for growth and body composition traits.

This suggestion is further supported by the serial body weight measurements by Walstra, White, and Andersen. These data were analysed together with the BW measurements of Doornenbal, Tullis, Noblet and Van Lunen, applying models (1) and (2) again. This analysis produced the estimates for mature body weight (BW_∞, in kg) and the associated Gompertz rate parameter (B_{BW}, in $kg.d^{-1}.kg^{-1}$) in Figure 3. The least-squares estimates from Walstra's, White's and Andersen's data fit satisfactorily into the pattern set by the other sources, supporting our above surmise that "sire line development across populations (breeds) has not favoured genotypes with larger mature size".

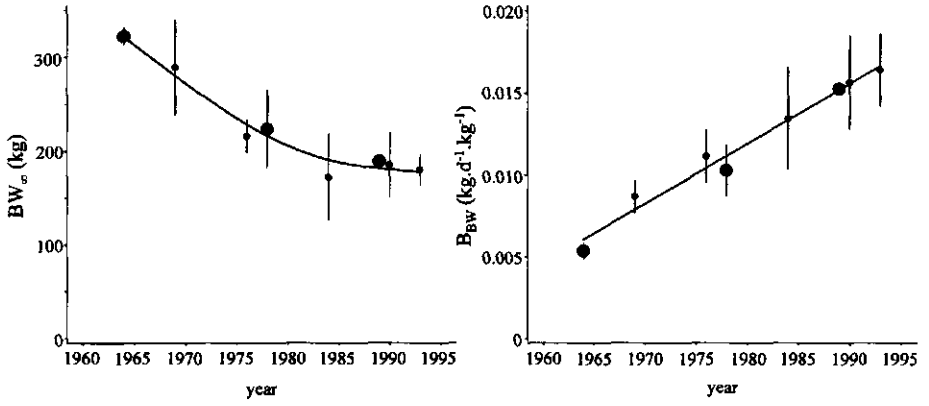


Figure 3 Time trends of mature body weight (BW_∞) and the associated Gompertz growth rate parameter (B_{BW}) as estimated for growing pigs of eight genotypes. Small symbols: the same data sources as in Figure 2. Large symbols: data from Walstra (1980); White *et al.* (1995); Andersen and Pedersen (1996). Further details as in Figure 2.

In contrast, mature size of dam lines *does* seem to have increased. Much of the evidence for this is more or less anecdotal, for example in Williams (1995, p. 108): "these changes can be explained with the simple assumption that modern genotypes differ from traditional ones only in their [mature] body size"; in Mackenzie and Revell (1998, p. 108): "an increase in mature body size [...] has undoubtedly occurred over the past few decades"; in Noblet *et al.* (1998, p. 113): "over the last 30 years [...] the mature weight of sows has increased"; and in Whittemore (1998, p. 183): "over the past two decades sow mature size has increased by some 30 %". This would agree with the notion that frame size has always been an important "informal" selection objective for maternal breeds all over Europe. However, data to properly substantiate such statements are very scarce in the literature, and are often difficult to interpret because of time differences in restricted feeding levels and in the general metabolic load of reproduction. For example, Klusáček and Diblík (1990) compared mature BW of the Czech Improved White and Prešticе breeds, as measured in 1987, with data from 1956 and 1963-65, respectively. Mature BW had increased in Czech Improved White females but decreased in males, and *vice versa* in Prešticе. No information is given on (a possible change in) feeding regimes.

Thompson *et al.* (1996) analysed growth data of castrates and females of five pig genotypes and found a very low correlation of BW_∞ with $P_{dep,max}$, but high positive correlations of BW_∞ with the stage of development (in terms of either age, BW, or BW/BW_∞) at the points of inflection of the protein accretion curves. By contrast, Emmans (1989; table 8.7) and Hancock *et al.* (1995) analysed BW growth data of males and females of nine turkey genotypes, and body protein growth data of males and females of six broiler chicken genotypes, respectively. Their results point into the opposite direction, showing high positive correlations of BW_∞ with maximum BW growth or $P_{dep,max}$, and very low correlations of BW_∞ with age at the points of inflection of the growth curves.
Our results and those of Thompson *et al.* (1996) suggest that selection for increased lean growth in pigs does not increase mature size to the same extent as has been the case in poultry. Instead, the reduction of mature size (BW_∞) depicted in Figure 3 is the consequence of a reduction in mature lipid mass.

Verification: within-genotype trends

The above interpretations require verification in terms of within-genotype trends with the exclusion of non-genetical factors. For a reliable comparison over time of genotype-intrinsic growth patterns, nutritional and other environmental factors should be controlled so as to continuously maximise the expression of the genotype's growth potential. Data should be collected on animals from the same genetic population over an extended period of time (see Oksbjerg *et al.*, 1997, and Petersen *et al.*, 1997, for an example), ideally making use of semen or embryo freezing techniques.

Alternative models

Daily protein deposition (the derivative of protein mass with respect to time) in pigs starts in early life at a very low level to increase rapidly towards a maximum and subsequently decline to zero. Emmans (1988) described the course of body protein mass over time with a sigmoid function in order to accommodate this pattern of the derivative, and chose the Gompertz function because it has its point of inflection at a relatively early stage (proportionally $1/e \approx 0.368$) of maturity. This allows for the decline of the derivative to take place much more slowly than its initial increase, which is what has been observed in real life (*e.g.* Oslage, 1966; Tullis, 1981; Black *et al.*, 1986).

The Gompertz function has some convenient statistical properties. One of these was exploited by Ferguson and Gous (1993a) to provide a low-cost alternative to the expensive measurement routines that had to be carried out to produce the data analysed in the present study. They proposed (i) to measure protein and lipid deposition rates twice only, at a very early and a much later stage, (ii) to express these rates as specific growth rates by dividing them by the current protein or lipid mass, and (iii) to regress these specific growth rates on the natural logarithm of current protein or lipid mass, respectively. If protein and lipid mass follow Gompertz curves in relation to time, as assumed for our model (1), then the slopes of the resulting regression lines provide an estimate of $-B_{Gomp}$ (assuming that the regression analysis (iii) would be forced to produce equal regression coefficients for both curves), whereas the x-intercepts provide estimates of $\ln(P_\infty)$ and $\ln(L_\infty)$. The main disadvantages of this linearisation are that it does not allow for the detection of sytematic deviations of the data from the Gom-

pertz pattern (two observations do not allow for the detection of curvilinearity), and that it cannot produce meaningful standard errors of the estimates. Ferguson and Gous (1993b) used this method to analyse data obtained from South-African "unimproved Large White × Landrace" pigs. This produced P_∞, $R_{L\infty/P\infty}$ and B_{Gomp} estimates of [28.4, 3.89, 0.0120] for females and [38.7, 2.60, 0.0107] for entire males, which would locate this genotype around 1974 on the horizontal axes of our Figure 2.

Nevertheless, the use of the Gompertz function (or any other specific member of the Richards family of sigmoids; see Fitzhugh, 1976, for background information) may result in biased estimates of the asymptotic values (P_∞, L_∞, BW_∞) because the fit of the observations is forced to accommodate a fixed y-coordinate (as a proportion of the asymptote) of the point of inflection. The points of inflection of protein and lean tissue growth patterns in pigs of different genotypes have been estimated to occur at different body weights (Schinckel and De Lange, 1996; Thompson *et al.*, 1996), although those differences were considerably reduced after scaling BW for BW_∞. Black *et al.* (1995) describe potential protein growth with the general Richards function, which has a point of inflection with a variable y-coordinate (Fitzhugh, 1976). Attempts to fit the form of the Richards function proposed by Black *et al.* (1995; p. 89) to the BW observations of Walstra, Doornenbal, Tullis, White, Andersen, Noblet and Van Lunen either failed because the MODEL procedure did not converge, or produced BW_∞ estimates similar to those in Figure 3.

Schinckel and De Lange (1996) make use of a three-parameter function with a flexible point of inflection ($Y = Y_\infty \times [1 - e^{-m \times X^a}]$; Bridges *et al.*, 1986). This function fitted the protein and lipid data of Doornenbal, Tullis, Noblet and Van Lunen rather well. The estimates of the y-coordinates of the points of inflection of the Bridges protein growth curves are in Figure 4. The corresponding estimates for the lipid growth curves were all around $0.48 \times L_\infty$, and the P_∞ estimates were similar to the ones from the Gompertz analyses. These results lead to three conclusions.

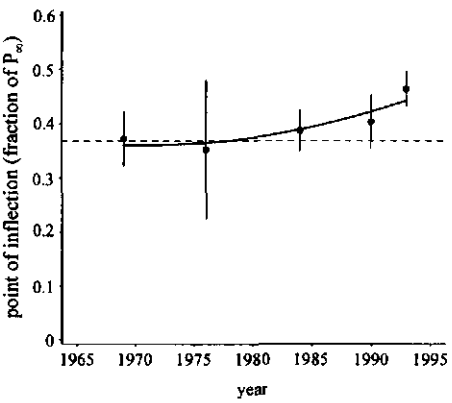


Figure 4 Time trends of the point of inflection of Bridges curves that relate body protein mass to age in growing pigs of five genotypes. The y-variable is expressed as a proportion of the associated estimates of mature protein mass (P_∞). The dotted line indicates the fixed value of the point of inflection of the Gompertz function. Further details as in Figure 2.

First, Doornenbal's, Tullis's and Noblet's LW pigs show a protein deposition pattern with a free point of inflection that is indistinguishable from the fixed Gompertz value of $0.368 \times P_\infty$, whereas Noblet's and Van Lunen's synthetic sire lines show an increasing tendendy towards a higher value (Figure 4). This suggests that either the intrinsic shape of the protein accretion curve in modern "meat-type pig" genotypes is changing, or the amino acid supply of these pigs had been less adequate than in the earlier experiments.

Second, the lipid deposition pattern in all five genotypes shows a free point of inflection substantially larger than the corresponding Gompertz value. Presuming that the Gompertz function provides a true reflection of the pig's lipid growth potential, this suggests that the energy supply had been less than optimal in at least the earlier experiments. Alternatively, it may suggest that the Gompertz function does not provide this true reflection.
Third, although our Gompertz estimates of P_∞ may be biased downwards because of lack of data towards maturity, because of unjustifiedly forcing a fixed point of inflection on the data, and/or because of environmental factors that could not be taken account of, this deficiency is *not* remedied by fitting a sigmoid with a flexible point of inflection (such as Bridges's) to these same data.

Conclusions

For a reliable comparison of genotype-intrinsic growth patterns over time, nutritional and other environmental factors should be controlled so as to continuously maximise the expression of the genotype's growth potential. Data should be collected on animals from the same genetic population over an extended period of time, and up to a much more advanced stage of development than has been customary in past experiments.

Ignoring the above, and accepting a possible confounding of the genotype contrasts in this study with nutritional and other environmental factors, it looks as if between-genotypes selection between "meat-type" pig populations has greatly reduced mature body lipid mass while leaving mature body protein mass practically unchanged. The growth rate of both body fractions has substantially increased, and the peak of the protein accretion curve seems to have shifted towards more mature stages of development.

Acknowledgements

Valuable contributions were made by Allan Schinckel, Martin Verstegen, Gerry Emmans, Colin Whittemore and Ella Luiting. Søren Andersen, Pim Brascamp, Dave McLaren, Nathalie Quiniou and Olwen Southwood helped in locating the various data sets in the time dimension. However, any errors made in that process are the sole responsibility of the author.

Grisen står i sin grisebing
og tenker dypsindig på viktige ting.

Inger Hagerup (1982) *Så rart.* Aschehoug, Oslo; p. 42.

Ik ben verbaasd over de grove wijze waarop men hier is opgetreden. Het is duidelijk dat men zich meer heeft beziggehouden met de vraag: 'wat is daar aan de hand?' dan met de kwestie: 'wat kunnen we er mee doen?'. Maar dat noemt men nu eenmaal wetenschap...

Marten Toonder (1968) *De kwade inblazingen.* In *Zoals mijn goede vader zei* (1983) De Bezige Bij, Amsterdam; p. 117.

Section 7 of Chapter 7 is based on parts of

P.W. Knap and S.C. Bishop (2000) Relationships between genetic change and infectious disease in domestic livestock. In *The challenge of genetic change in animal production* (eds. W.G. Hill, J.C. McKay, G. Simm and A.J. Webb). British Society of Animal Science, Edinburgh. BSAS occasional publication 27.

Chapter 7

General Discussion

Sections 1 and 2 of this chapter deal with some details of protein turnover and thermoregulation that could not be included in Chapters 1 or 3, either because there was insufficient space in the associated journal articles, or because this information was discovered after these had been published.
In **section 3** we discuss various aspects of between-animal variation, the most relevant feature of Chapters 2 and 4. These are (i) ways to estimate variation of traits associated with model parameters, (ii) the magnitude of variation in maintenance requirements and (iii) the way this variation is partitioned, and (iv) priorities for further stochastic modeling of growth metabolism.

The question that led to the study carried out in this thesis was quoted in section 2 of the Introduction chapter: "to what extent [can] differences in maintenance requirements be attributed to differing proportions of the different organs and tissues of the body, each having different metabolic rates"? (Webster, 1988). Chapters 2 and 4 provide a part of the answer, summarised in point (iii) of the previous paragraph. Hence section 3.3 of this chapter adresses the question that this study started with, and it must be seen as the heart of this thesis.

Sections 4 and 5 deal with some issues of body protein and lipid partitioning (Chapter 5) and protein and lipid growth curves (Chapter 6) that were not discussed in those chapters, including ideas about the further testing of the results obtained there.
In **sections 6 and 7** we describe some aspects of membrane transport and immunocompetence, two important maintenance functions that have not been explicitly dealt with in this study due to insufficient quantitative information, but that would be strong candidates for mechanistic modeling according to section 3.
Section 8 deals with a statistical problem that was encountered but not further elaborated in Chapters 2 and 4. This concerns the effect of measurement errors on the independent variables of a regression analysis.
Section 9 discusses the use of growth models, such as the ones evaluated in Chapters 1 and 3, in animal breeding.
Finally, some short concluding remarks are made in **section 10**.

1. Protein turnover: Chapters 1 and 2

1.1. Model assumptions

In the model extension of Chapter 1, the synthesis of body protein was assumed to require 410 kJ metabolisable energy (ME), the equivalent to 5 mol ATP, per mol arranged peptide bonds. The latter value was taken from Van Es (1980), who expressed a considerable uncertainty about it. In fact, more than a decade later this uncertainty had not been eliminated when Lobley (1993) referred to two studies that "attempted to actually quantify the energy cost of protein synthesis". He concluded that "in both cases the number of ATP molecules (7 to 10) apparently required per mole peptide bond synthesis exceeded considerably the minimum theoretical value". Kelly *et al.* (1993) wrote, in the same book that "when it is considered that something as basic as the real *in vivo* energy cost of synthesis of a peptide bond is not really known (Van Es, 1980), it is evident that a great deal of quantitative metabolic information must yet be garnered in order to create mechanistic models that are accurate". Hence it is quite likely that our model extension will have to be reparameterised when new information

on this issue becomes available.

Perhaps the most significant aspect of that model extension in Chapter 1 is the separation of protein turnover into the turnover of already present body protein (with ME requirements $ME_{turn,pres}$) and the repeated synthesis-and-breakdown of newly deposited protein (with ME requirements $ME_{turn,dep}$) in equation (2) of that Chapter. This separation relates, of course, to the distinction between cellular maintenance functions and deposition-related processes that was mentioned in section 1 of the Introduction chapter. See also equation (8) in Chapter 1 and the subsequent paragraph, which discusses the separation of ME_{maint} into a fixed part ($ME_{maint,indep}$) and a part that varies with body protein content and composition (the costs of turnover of present body protein). Moreover, the initially fixed value for the ME costs of protein deposition of 53 $MJ.kg^{-1}$ is made dependent on the composition of protein deposition, which changes during development. Deviations from the average value of the latter portion would be allocated to ME_{maint} in a fixed system. In equations (7) and (8), the original ME_{maint} has effectively been replaced by the sum of $ME_{maint,indep}$ and the protein turnover and deposition costs.
This separation goes back to Whittemore and Fawcett (1974) and Reeds (1989). The latter distinguished between two protein turnover components: "an essentially inevitable turnover associated with the maintenance of cell function and a variable turnover associated with growth". The earlier literature (as cited in Riis, 1983a, and Simon, 1989) on protein turnover and its energy requirements shows a considerable amount of confusion about the effects of feeding level (or growth rate) and age on the overall turnover rate, and the above distinction seemed the logical way to eliminate much of that confusion.

The non-turnover-related proportion of ME_{maint} (*i.e.*, $ME_{maint,indep}$) is estimated to comprise 65 % of total ME_{maint} in Chapter 1, but in Table 3 of Chapter 2 and Table 2 of Chapter 4 it covers usually only 55 % or so. This is because "total ME_{maint}" in those tables is simply the sum of $ME_{maint,indep}$ and the ME requirements of protein turnover, and the latter includes not only $ME_{turn,pres}$ but also $ME_{turn,dep}$. $ME_{turn,pres}$ is a usual component of the maintenance requirements (as used in Chapter 1), but $ME_{turn,dep}$ is commonly treated as part of the ME costs of protein deposition. So total ME_{maint} in the above tables comprises more than $ME_{maint,indep}$ / 0.65, and ME_{turn} appears to cover more than 35 % of the total. In other words: "total ME_{maint}" in the above mentioned tables and "ME_{maint}" in Chapter 1 are not exactly equivalent.

1.2. Contribution of protein turnover to energy expenditure

The contribution of simulated protein turnover ($ME_{turn,pres}$ + $ME_{turn,dep}$) to daily energy expenditure ranged from 16 % (at low $P_{dep,max}$ and high feeding level) to 26 % (*vice versa*) in the simulations of Chapter 1. We noticed there that similar fractions were obtained by Gill *et al.* (1989) when simulating protein metabolism of young growing lambs. Similarly, Beckerton (1976) estimated the energy cost of protein synthesis in 20 kg lambs at 19 % of total heat production); Lobley *et al.* (1980) conclude that "the energy costs of protein synthesis [in cattle] would account for 14-19 % of heat production, and based on [another analytical technique] the values would increase to 24-31 %"; Reeds *et al.* (1985, 1987) measured the contribution of protein synthesis to total body energy expenditure at 15 to 22 % in growing pigs; Milligan

and Summers (1986) made the tentative estimation of the contribution of protein re-synthesis to basal energy expenditure as 9 to 12 %, of protein degradation possibly up to 15 %; and Webster (1988) states that; "the two largest contributors to the cost of cell maintenance are protein turnover and Na^+/K^+ transport, each contributing, very approximately, 20 % to total heat production"). Sève and Ponter (1997) argue towards an "estimate of 30 % of total heat production associated with protein synthesis in a 35 kg pig, bearing in mind that about half this amount is part of the energy requirement for maintenance". Of course, the various contributions to energy expenditure of protein turnover can only be reasonably compared when the respective feeding levels are known.

Figure 6 of Chapter 1 presented the cumulative distribution of protein synthesis over turnover of present protein pools (muscle, connective tissue, liver, gastro-intestinal, blood plasma and "other") and repeated synthesis of protein newly deposited into those pools, as predicted by our simulations. These patterns can be compared, to some extent, to the "contribution of major tissues to whole-body protein synthesis" of lambs aged 1 week or 8 months, as given in figure 14.5 of Lobley (1993) and reproduced here in Figure 1.

The comparison is not straightforward: the protein turnover of the 8 month-old ruminant lambs is dominated by the gastro-intestinal pool and by what may be wool growth, and the 1 week-old preruminant lambs are in a much earlier stage of development than our simulated 23 to 100 kg pigs. Nevertheless, many aspects of both partitioning patterns are very similar.

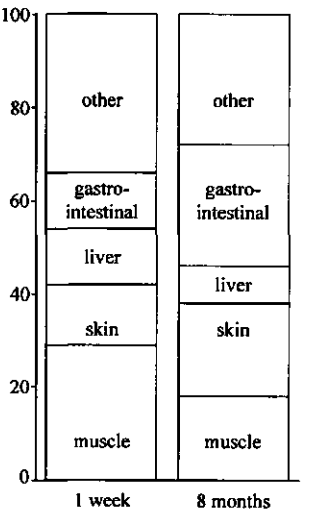


Figure 1 Proportional contribution (in %) of major tissues to whole-body protein synthesis in lambs at two ages. After Lobley (1993), modified.

1.3. Variation in turnover rates

All the variation in protein turnover that was generated in the simulations of Chapter 2 is necessarily due only to variation in body composition. The fractional rate of synthesis of newly deposited protein (FRS_{dep}) was assumed to be a constant, as were the turnover rates of present protein (TR_{pres}) for each pool. However, the discussion associated with Figure 5 in Chapter 1 suggested that this may be an oversimplification, and that FRS_{dep} levels may vary among genotypes, or over time.

The literature provides little, if any, evidence on the protein pool level for this notion, and it is in contradiction with Emmans's (1994) findings of constant energetic efficiencies of protein deposition over genotypes and degrees of maturity in chickens and cattle. On the other hand, on the whole animal level, Oddy (1993) and Oddy *et al.* (1998) found diminished protein degradation rates, but unaffected protein synthesis rates, in sheep and cattle genotypes that had been selected for increased weaning weight and yearling growth rate. The energy costs of protein degradation are much lower than those of protein synthesis (see Chapter 1); hence Oddy's findings may have influenced the energy balance of his animals to a limited extent only .

The experimental results of Weiler *et al.* (1997, 1998) may be of some relevance for this particular discussion. These authors studied endocrine profiles in pigs of widely different genotypes (Large White, Meishan, and wild boar). They found significantly different profiles of IGF, cortisol, oestradiol and testosterone in relation to age, and were able to relate these to the genotypes' growth protential. Weiler *et al.* (1997) associate these hormones with anabolic and catabolic processes, more specifically with protein synthesis and degradation. Claus and Weiler (1994) describe the way protein synthesis in pigs is regulated by the hormones above mentioned, which we have summarised in Figure 2; see also Evers (1989). Such functional relations between hormone profiles and anabolic and/or catabolic action, combined with individual variation in hormone profiles (as reported by U. Weiler, personal communication, 1997), would lead to individual variation in anabolic and/or catabolic rates. Detailed information on individual profile differences is clearly needed before the value of these processes for the quantification of possible variation in protein turnover rates can be assessed.

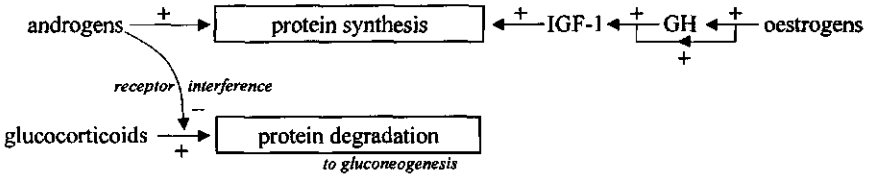


Figure 2 Endocrine regulation of protein synthesis in pigs. GH: growth hormone; IGF: insulin-like growth factor. After Claus and Weiler (1994).

Assuming that such individual variation is partly of a genetic nature, this would suggest possibilities for genetic selection for more energetically efficient protein metabolism in the meat-producing livestock species, which would be highly desirable from a production-economical point of view. This was recognised by Baldwin *et al.* (1980) who notice that "when considerable biological variation exists, opportunities for improvement are embedded within the variation".

These authors attempted to identify basic characteristics or parameters of metabolism that might be manipulated in animal production, and the inherent constraints imposed upon such manipulation. Based upon observed differences between highly efficient and "average" ruminant animals, their estimated scope for improvement of production efficiency is 20 %, and they argue that this might be achieved by (i) direct selection for metabolic efficiency. Alternatively, apparent ME_{maint} might be manipulated, for example through (ii) selection of animals "that could handle high nutrient intakes" and the associated increased levels of nutrient processing functions, "without experiencing increases in relative weights of high energy-requiring tissues" such as liver, heart and intestine. This would effectively reduce ME_{turn}, according to the findings of Chapter 1. These authors argue against careless attempts to manipulate turnover rates as such, pointing to the disturbance of homeostasis that would likely result from this. A third opportunity for improvement of animal efficiency is through (iii) manipulation, hormonal or otherwise, of the biochemical patterns of nutrient use. For example, in ruminants, if the use of long chain fatty acids for maintenance processes and volatile fatty acids for body lipid synthesis could be reversed into the opposite pattern (using long chain

fatty acids for lipid synthesis), these authors claim that ME_{maint} could be reduced by 20 %.

Conventional selection for feed efficiency of production in livestock species may have resulted in some shift in the metabolic components of protein turnover, but this is not very likely, for two reasons. First, feed efficiency has not been a common direct selection trait in livestock breeding because of the high costs involved with individual feed intake measurement (*cf.* Knap and Van der Steen, 1994). If and when it was applied (most notably in pig breeding), this was always as part of a more elaborate breeding objective incorporating several other traits (see the various chapters in Groeneveld, 1993). Second, the main consequence of such selection has been a shift in body composition towards a higher lean-to-fat ratio (see Chapter 6 for an illustration of this principle). This has provided a convenient characteristic with much "elasticity" for genetic change in gross efficiency without the need for changes in complicated metabolic processes.

Of course, with the advent of molecular genetic technology and its much larger power to focus breeding activities on specific physiological functions, the above mentioned metabolic processes might form an interesting target for marker-assisted selection or transgenesis. This would, perhaps, reflect the above ideas of Baldwin *et al.* (1980) in their most ultimate form. In that respect, the reservations of these authors towards direct manipulation of "the 'futile' cycles of intermediary metabolism, of macromolecular turnover [...], and of ion pumping" (Reeds *et al.*, 1985) because of the likely loss of homeostasis is noteworthy. This was supported by Summers *et al.* (1986), who wrote: "the extent to which one may potentially alter or reduce the energy cost of any ATP-consuming process to benefit productive output is restricted both thermodynamically and by biological function". As we mentioned in section 2 of the Introduction chapter, maintenance (and with it, protein turnover; see Hawkins, 1991, and Hawkins and Day, 1996) should be regarded as a set of fitness functions. Manipulating these requires care and background information, and the latter is very scarce at present.

1.4. Protein turnover and immune system activation

An obvious case of accelerated protein turnover rates is the case of tissue repair after damage caused by infection or trauma. Garlick and Fern (1985; tables 2 and 4) illustrate this at the whole-body level where protein synthesis and breakdown levels increased by 17 to 47 %, and by 21 to 82 %, respectively. They found different responses for different tissues (protein synthesis decreased in muscle, liver and intestinal mucosa, but increased in spleen, lung and heart). Similar information was presented by Carli *et al.* (1989) for humans recovering from surgery. Neither of these authors present any information on the accuracy of their estimates.

These findings suggest that a dynamic model that deals with protein turnover and immune system activation (as discussed by Knap and Bishop, 2000) should explicitly consider the interactions between these functions.

2. Thermoregulation: Chapters 3 and 4

2.1. Ambient temperature and vasomotor action

The thermoregulatory rules that were implemented in Chapter 3 (as described in its Appendix II) were largely taken, as such, from the literature. One of the few original contributions to the theory of thermoregulation in pigs that was made here is the relationship between subcutaneous fat depth and tissue insulation that was derived in Appendix III of that chapter. Of course, for our

purpose of clarifying the dependence of maintenance-related processes on body composition, this relationship is particularly important. But it is puzzling that the established literature on thermoregulation in pigs (a species proverbial for the size of its subcutaneous fatty tissue) more or less ignores the issue.

The sow data of Holmes and McLean (1974) that were included in Figure 7 of Chapter 3 were measured at environmental temperatures of 6 and 8 °C (the plotted $I_{tissue,cold}$ values are the within-sow averages) and 23 °C ($I_{tissue,hot}$); in addition, these authors report measurements on the same sows taken at 13 and 18 °C. Taken together these data illustrate the gradual increase of b_{SHL} as a result of vasodilation over the thermoneutral zone that was depicted in Figure 1 of Chapter 3: see Table 1, where the regression coefficients represent $\frac{d(I_{tissue})}{d(BF)}$. The lower critical temperatures of these sows ranged from 10 to 17 °C. Accordingly, the measurements taken at 6 and 8 °C show essentially the same high regression coefficient, suggesting that peripheral vasoconstriction became complete somewhere between 13 and 8 °C, so that fatty tissue insulation is directly related to its depth at the lower temperatures.

From 8 to 18 °C, and to a lesser extent also from 18 to 23 °C, a gradually increasing vasodilation causes tissue insulation to become less and less dependent on fat depth until the regression is essentially zero (and vasodilation must be complete) at 23 °C.

Table 1 Regression statistics of tissue insulation (in °C.m².W⁻¹) on backfat depth (in mm) in sows at various environmental temperatures. Data from Holmes and McLean (1974).

T_{env} (°C)	regression	correlation
6	0.00233	0.78
8	0.00240	0.82
13	0.00161	0.58
18	0.00046	0.23
23	0.00032	0.15

Table 2 Regression statistics of tissue insulation (in °C.m².W⁻¹) on (sub-)cutaneous tissue depth (in mm) in humans at various environmental temperatures. Data from LeBlanc (1954).

T_{env} (°C)	regression	correlation
10	0.0133	0.97
16	0.0087	0.98
21	0.0037	0.63

Similar measurements had already been published by LeBlanc (1954), who measured skin temperature and (sub-)cutaneous tissue depth at 20 measurement sites on the body of six humans at three environmental temperatures, to derive tissue insulation values. The statistics from this study that correspond to the ones in Table 1 are in Table 2. The regression coefficients from the two data sets differ in magnitude mainly because the measurement sites on the body were different. Apart from that, the patterns show much similarity.

2.2. Fat depth and tissue insulation

In accordance with the published data mentioned in section 2.1, Ingram (1964) writes: "as pigs age, they are able to tolerate lower skin temperatures and so reduce their rate of heat loss.

This ability may be related to an increase of subcutaneous fat since [it has been] shown that fat men have lower skin temperatures than have controls". In his thermoregulation model, McArthur (1987) expresses tissue heat resistance in units per cm (probably cm tissue depth), and Curtis (1988) mentions a tissue insulation value of 0.009 °C.m^2.W^{-1} per mm fat depth in pigs, but does not document this. But the notion of tissue insulation as a logical dependant of subcutaneous fat depth seems to have been largely overlooked in the modern literature. For example, Black *et al.* (1999) mention a possible relation between heat loss and backfat depth in pigs, but state that little is known about the quantitative relationship. More seriously, the classical thermoregulation model of Bruce and Clark (1979) on which most subsequent attempts at modelling have been based, ignores the relationship as well.

The interpretation of the regression coefficients of I_{tissue} on T_{env} is complicated. The HP figures that we used in equation (A3-1) of Chapter 3 reflect whole-body heat loss, whereas the skin temperatures that they were combined with to calculate I_{tissue} were measured on the central part of the body (on the back, mostly) as opposed to the extremities (legs, tail and ears). This is relevant because cold-induced vasoconstriction is initiated earlier (*i.e.*, at a relatively high temperature), and seems to be much more severe, in the extremities than on the trunk in pigs (Stombaugh and Roller, 1977) as well as in other species; McArthur (1981a) deals with this issue in more detail. Hence in cold conditions, T_{skin} is much lower on the extremities than on the central part of the body. As a result, the use of trunk skin temperatures for T_{skin} in that equation (A3-1) overestimates whole-body skin temperature and hence underestimates whole-body I_{tissue}. Blaxter (1989) suggested that the role of vasoconstriction in the extremities is more pronounced in animals with poorer trunk insulation (*i.e.* lower BF levels). If this were the case, this negative bias in I_{tissue} would be larger in pigs with lower fat depth, causing the regression of I_{tissue} on BF to be overestimated. To quantify this effect, we could make use of the piglet data by Stombaugh and Roller (1977) because they reported their measurements of skin temperature on the extremities and surface area of the various parts of the body. Such data were not available for pigs with higher BF levels; to approximate these, we have used Whittow's (1962) data on one-year-old cattle.

Table 3 Bias in tissue insulation estimated from trunk skin temperatures versus whole-body (overall) skin temperatures.

animal type	T_{env}	T_{body}	$T_{skin,trunk}$	$T_{skin,overall}$	bias[†]
one month-old piglets[‡]	15	39.2	32.5	28.3	–0.385
one year-old cattle[§]	5	38.5	20.5	18.0	–0.123

† $\text{bias} = \dfrac{T_{skin,overall} - T_{skin,trunk}}{T_{body} - T_{skin,overall}}$ ‡ data from Stombaugh and Roller (1974) § data from Whittow (1962)

The relevant statistics are in Table 3. The bias has been calculated as the difference between overall and trunk skin temperature, expressed as a proportion of the "true" temperature gradient between body core and overall skin temperature. These values show that the negative bias in estimated I_{tissue} is, indeed, much larger in animals with poorer trunk insulation. When we assume that the cattle value holds for pigs at the higher BF extreme in Figure 7 of Chapter 3 (*i.e.* at 45 mm BF) and make use of the linear relation $I_{tissue} = b_0 + b_1 \times BF$ suggested by that Figure, the "true" b_1 value is more closely approximated by

$$b_1^* = \frac{\frac{1}{1-0.123} \times (b_0 + b_1 \times 45) - \frac{1}{1-0.385} \times (b_0 + b_1 \times 3)}{45 - 3}$$

where $b_0 = 0.05$ and $b_1 = 0.0022$ are the (biased) estimates from the regression in Appendix III (Figure 7) of Chapter 3, and the values 3 and 45 represent the lower and higher extremes of BF in that Figure. The resulting value is $b_1^* = 0.0019$ °C.m^2.W^{-1} per mm BF, which suggests that the rounded value of 0.002 °C.m^2.W^{-1} per mm BF that was arrived at in Chapter 3 can be retained. This value is much smaller than the one given by Curtis (1988) and quoted at the beginning of this section, probably because of differences in sites of fat depth measurement, as in the comparison between Tables 1 and 2.

2.3. Vasoconstriction and vasodilation patterns

Black *et al.* (1986) write: "Tissue thermal resistance [...] falls abruptly once [T_{env}] exceeds [T_{LC}] because of vasodilation. The literature provides few direct measurements of tissue thermal resistance over the whole surface of the pig after vasodilation. Bruce (1981) suggested that the value was zero. However, this implies that there is no temperature gradient between the body core and the skin of pigs exposed to hot conditions, and is not supported by experiments (Stombaugh and Roller, 1977)".

Actually, Bruce (1981) assumed that in "a pig under heat stress [...] the tissue insulation is reduced by vasodilation to a negligible amount". And actually, Stombaugh and Roller (1977) found in their 30 to 60 day-old piglets at T_{env} = 37.6 °C a hypothalamic temperature of 39.7 °C, whereas skin temperatures on trunk and extremities ranged from 39.3 (on the upper hind leg) to 39.9 °C (on the tail). However, the rectal temperature of these piglets was 40.7 °C at this T_{env} level, and the authors used the difference of rectal temperature and skin temperature to calculate whole body tissue insulation I_{tissue} = 0.06 °C.m^2.W^{-1}. When using hypothalamic temperature instead, the resulting figure is 0.013 °C.m^2.W^{-1}, which may indeed be regarded as a "negligible amount". It seems to be very much a matter of how "core temperature" is defined and measured; Bligh (1973) deals with this issue in more detail.

At environmental temperatures below T_{LC}, Stombaugh and Roller (1977) calculated average I_{tissue} values in their 30 to 60 day-old piglets of 0.048 °C.m^2.W^{-1} on the trunk and 3.50 °C.m^2.W^{-1} on the extremities. These values must be compared to 0.030 and 0.63 °C.m^2.W^{-1}, respectively, in 8 to 12 day-old piglets (Stombaugh *et al.*, 1973). The authors conclude that "the 8 to 12 day-old piglets with less subcutaneous fat and smaller body size had higher [cold] tissue conductance values. This difference was apparent for both [trunk] and extremity body regions."

When T_{env} was reduced to below 30 °C, "in the 8 to 12 day-old piglets [...] vasomotor responses continued to increase tissue insulation as T_{env} decreased to approximately 10 deg below thermoneutrality [T_{LC} = 33 °C]. Although vasomotor responses continued to be recruited as [T_{env}] decreased below thermoneutrality (30 °C), the [30 to 60 day-old piglets] reached maximal values of tissue insulation at a much milder degree of cold stress", *i.e.* at T_{env} = 25 °C. In other words, the very young piglets attained complete vasoconstriction only at temperatures 10 deg below T_{LC}, whereas this deficit was reduced to 5 deg below T_{LC} in the older ones. Apparently, the piglet needs some months to fully develop its vasomotor responses,

most notably in the extremities. The authors cite Mount (1968) to state that "previous work with both piglets and young swine has indicated that the vasomotor responses were fully developed at [T_{LC}]. Most of this work considered skin surface temperatures only on central body regions".
Fuller and Boyne (1972; see Table 2 in Chapter 3) suggest that "the thermal conductance of the pig continues to diminish with falling temperature below [T_{LC}.] The thermal conductance at very low temperatures may therefore be appreciately lower than when measured closer to [T_{LC}]".

The issue of development of vasomotor responses in very young mammals was also raised by Gonzalez-Jimenez and Blaxter (1962), who studied thermoregulation in 2 to 30 days-old calves. They notice that these calves were much more poorly insulated than adult steers, and suggest that this is "due to changes in the blood supply to the skin rather than to morphological changes in skin thickness which could hardly change so rapidly with age".

The evidence on this matter is clearly conflicting. In the approach outlined in equations (5) and (6) of Chapter 3, $b_{SHLcold}$ is supposed to be fully operational below T_{LC}; if in reality peripheral vasoconstriction is attained only gradually (first in the extremities and later on the central part of the body, see section 2.4) with decreasing sub-critical T_{env}, our model would overestimate $I_{tissue,cold}$ at T_{env} close to T_{LC} which would lead to underestimation of SHL_{cold} and HP in such conditions. This is in contradiction with the findings in Chapter 3, where cold HP is mostly *over*estimated (Figure 3).

2.4. The relevance of modeling thermoregulation

The main conclusion from Chapter 4 is that thermoregulatory processes do not contribute much to the between-animal variation in maintenance requirements (see section 3.3). Hence from the point of view of pig breeding, there is little reason for explicit consideration of thermoregulation when attempts are made at analysing the physiological variation within a population. But the process can be significant for prediction of growth and carcass traits as such.

This can be illustrated with data from Kovac and Groeneveld (1990), who analysed performance test results obtained in Germany between 1979 and 1987, and from Deo *et al.* (1981), who studied the growth of pigs kept in experimental facilities in Uttar Pradesh (India). Figure 3 shows the estimated seasonal trends (adjusted for genetic and other environmental effects) of growth rate in these data. These examples show the extent to which growth traits can be influenced by changes in the environmental temperature, even in confined conditions with climate control.
The amplitudes of the German and Indian data in Figure 3 are about 50 and 25 $g.d^{-1}$, respectively, ranging from 6 to 9 % of the corresponding overall mean level. Similar patterns were reported for pigs in Taiwan by Hsia and Lu (1989), with amplitudes of 6 % for growth rate and 13 % for *ad libitum* feed intake. Gourley *et al.* (1989) and Brumm *et al.* (1990) monitored feed intake of growing pigs in commercial conditions in Iowa and Minnesota (USA), and report amplitudes of 11 and 18 % for pigs housed indoors and outdoors, respectively. Likewise, Labroue *et al.* (1999) analysed French performance test results and found seasonal amplitudes of residual feed intake of about 220 $g.d^{-1}$, or 10 % of the mean feed intake level, as indicated in Figure 3 (results kindly provided by F. Labroue, personal communication,

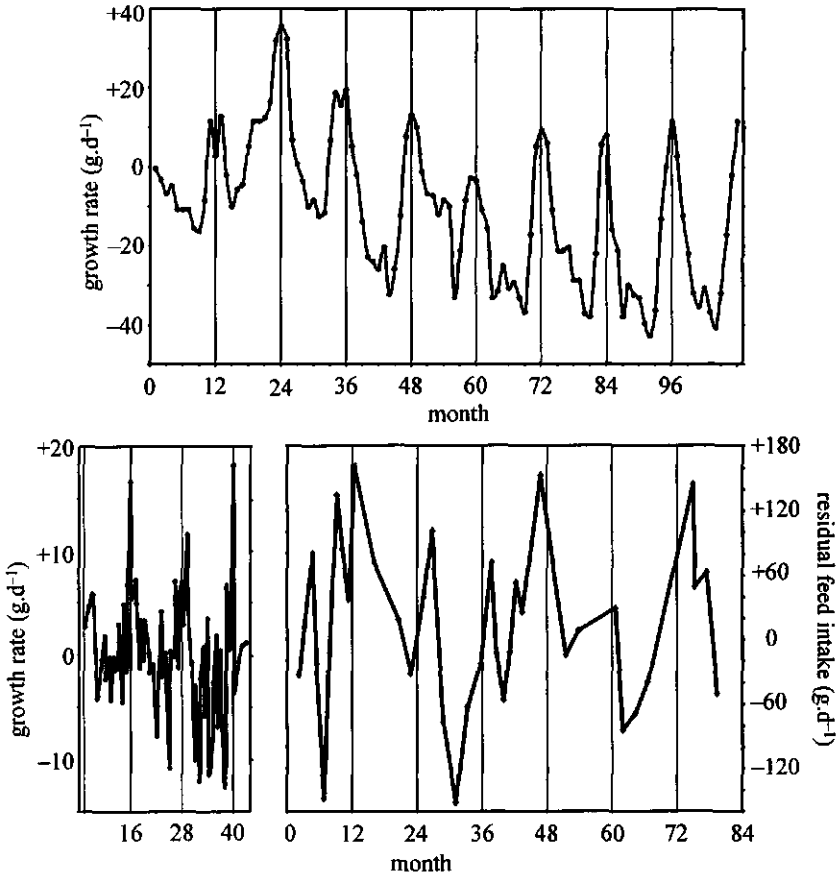


Figure 3 Environmental trends of growth rate and residual feed intake in pigs. Top: German performance testing data from 1979 to 1987 (data from Kovac and Groeneveld, 1990; average growth rate 791 $g.d^{-1}$). Bottom left: Indian (Uttar Pradesh) experimental data from 1972 to 1974 (data from Deo *et al.*, 1981; average growth rate 263 $g.d^{-1}$). Bottom right: French performance testing data from 1988 to 1994 (data from Labroue *et al.*, 1999; average feed intake 2168 $g.d^{-1}$).

1999). In all these cases, feed intake and/or growth rate were reduced in the warm season.

The above results show significant seasonal effects on *ad libitum* feed intake and growth rate of growing pigs, less pronouncedly so in temperate climates and in conditions with climate control. But climate has a significant influence on growth and feed intake even in performance test facilities with their explicit intention of reducing environmental variation.

2.5. Thermal adaptation

As was noticed in Chapters 3 and 4, the most poorly covered component of thermoregulatory functions (both in the model extension that was evaluated in Chapter 3 and in the literature on growing pigs at large) is the set of processes that the animal may invoke to acclimatise, *i.e.* to gradually adapt its body functions to prolonged cold or hot conditions. The lack of fit of our

model to the "cold" experimental data of Fuller and Boyne (1971, 1972) in Figure 5 of Chapter 3 is most probably due to the fact that this experiment lasted a long time (from 25 to 85 kg BW) and was conducted at very low effective environmental temperatures, which would have provided a strong urge for these pigs to acclimatise. The model, however, does not provide for such actions, mainly because there is a severe lack of quantitative information on the issue in the literature.

Most acclimatisation strategies involve some redistribution of organ or tissue matter, and hence require growth (and time) to get established. An example is the re-partitioning of body lipid towards the subcutaneous depot in cold conditions, as reported by Le Dividich *et al.* (1987) and Rinaldo and Le Dividich (1991). Demo *et al.* (1995) give similar results, and also report a shift in coat depth towards longer hair in the cold. But these findings cannot be generalised, at least not to other species: Pond (1992) compared the lipid partitioning in domestic and/or temperate climate-carnivores *versus* wild polar bears, and failed to find the corresponding contrast. The blood vessel supply of the subcutaneous fatty tissue is another aspect that can be modified to suit thermal acclimatisation (Ingram and Weaver, 1969). These same authors (Weaver and Ingram, 1969) report adaptations in the overall morphology of growing pigs that cause a decrease of the body surface in cold conditions, mainly by reducing the size of the extremities (limbs, ears) and arranging for a more compact trunk shape.

The modeling of thermal adaptation in growing pigs would require an experiment to produce quantitative estimates of the associated parameters. In order for the outcome to be applicable to a satisfactory range of genotypes and environmental conditions, the experimental variates (and their tentative ranges) would be body size (in terms of BW from, say, 15 to 150 kg), fatness (in terms of P2 backfat depth at 100 kg BW, from 5 to 20 mm) and metabolic intensity (in terms of growth rate, from 400 to 1400 $g.d^{-1}$). T_{env} could be varied over the same range as in Chapter 4 (T_{LC} – 5 to T_{UC} + 5 °C), and the pigs would be subjected to these treatments for at least two months. Traits to be measured repeatedly are BW, body surface area and its proportion that is wet, daily ME and protein intake, body protein mass, the size of the subcutaneous and other lipid depots, coat depth, rectal temperature and the temperature of the skin and the coat surface (if any) at some representative sites on the body (see section 2.3) and evaporative and sensible heat loss. This would require a respiration trial for the measurement of heat loss, with regular CT scanning for the measurement of protein and lipid depots (and, conveniently, surface area). Metabolic intensity and fatness could be regulated by a careful choice of genotypes: females of a slow growing lean genotype (*e.g.* Belgian Pietrain), castrates of a slow growing fat one (*e.g.* Meishan) and entire males of a fast growing lean one (a modern synthetic sire line). Further regulation could be accomplished by using different feeding levels of a high-density diet, such as *ad libitum* and about 75 % of that level.
Assuming three genotypes, four BW ranges (15-30 kg, 30-60 kg, 60-100 kg, 100-150 kg), two feeding levels and five T_{env} levels, this trial would require $3 \times 4 \times 2 \times 5 = 120$ subclasses with about five individuals each. Heat loss and skin temperature measurement, and CT scanning, would be at biweekly intervals (5 records per pig).

The observations would be analysed in order to describe the T_{env}-dependent development over time of the relevant parts of the model extension in Chapter 3. These include: (i) coat insulation, as a possible extension of equation (A2-2); (ii) tissue insulation in relation to fat

depth and BW as in equations (A2-2) and (A3-1); (iii) subcutaneous lipid mass and fat depth in relation to total body lipid mass and BW (Appendix III); (iv) the parameters of the Meeh formula that relates body surface area to BW (first paragraph of Appendix II); (v) the parameters of equations (A2-8) to (A2-10), relating cold and hot EHL, and EHL due to wet skin, to BW; and (vi) heat production (= SHL + EHL) adjusted for protein and lipid deposition as an estimate of ME_{maint} in equation (3).

2.6. Thermoregulation and protein turnover

There is a logical functional link between thermoregulation and protein turnover: increased protein turnover and its associated increased metabolic intensity lead to increased heat production, which would reduce both the lower and the upper critical temperature. This relationship is implicitly dealt with in the model described in Chapter 3, among other things related to feed intake reduction. See also Webster (1983). Acclimatisation to prolonged hot conditions may be directed towards a reduction of the high protein turnover associated with the visceral organs (Black *et al.*, 1999): "exposure of pigs to hot conditions for extended periods reduces the weight of visceral organs, particularly the liver, digestive tract and heart (Sugahara *et al.*, 1970; Rinaldo and Le Dividich, 1991)". This notion would imply that the organ mass reduction is triggered by the need to reduce heat production by saving on protein turnover. Alternatively, it may be the logical consequence of the reduced feed intake at high environmental temperatures, which would require less capacity for nutrient processing.

3. Between-animal variation: Chapters 2 and 4

Stochastic simulation has been applied in animal science in general, and focused on pig production in particular, for the evaluation of breeding schemes (De Vries *et al.*, 1990; Satoh *et al.*, 1992; Appel *et al.*, 1995; Krieter, 1995; MacKenzie, 2000), reproductive strategies and sow replacement policies (Singh, 1986; Huirne *et al.*, 1988; Pomar *et al.*, 1991; Faust *et al.*, 1993), climate regulation strategies (Bridges *et al.*, 1992), diet formulation (Dyer, 1991), epidemiology of contagious diseases (Damrongwatanapokin, 1993; see also De Jong, 1995), and animal health management (Marsh and Morris, 1993).
Stochastic *growth* models are less well-developed. The concept of "constructing theories of population structures" was introduced by Emmans and Fisher (1986) who noticed at the same time that "including stochastic elements in models for predicting nutritional response has not received a lot of attention". Somewhat later, Emmans (1989; table 8.13, figure 8.14) illustrated the use of such models for diet formulation in poultry. An example in pigs is Ferguson *et al.* (1997) who simulated variation in the growth potential parameters (P_∞, $R_{L\infty/P\infty}$ and B_{Gomp}) of the same model that was used in our Chapters 3 and 4, and attempted to derive reasonable values for their coefficients of variation. The *application* of stochastic growth models is to use them as a research tool to obtain answers to questions; this is somewhat of a novelty in pig production. Among these questions are the ones addressed in this thesis such as: how important is variation in body composition for variation in maintenance requirements? Stochastic growth models can also be used as a developmental tool to optimise product development procedures (*e.g.* which performance testing regime maximises the expression of genetic potential for feed efficiency?).

The most difficult aspect of model development is usually the proper parameterisation of the

model for the system (in pig terms: the population) of current interest. As was argued in the first paragraph of the Discussion section of Chapter 3, attempts at pig growth simulation commonly fail to take into account that pig genotypes differ considerably in their elements of the relevant model equations (see Chapter 6), and hence should be properly characterised in the model for its predictions to be meaningful. Such a characterisation is complicated and laborious enough for population mean levels of model parameters, and even more so for their coefficients of variation and the possible co-variation. Many of the traits that must be measured to quantify the value of the current generation of growth model parameters can only be measured in a group of animals from the same population, each animal contributing a data point to a population-specific curve that must be described because the model parameter depends on its slope, intercept, point of inflexion, asymptote, or suchlike. Characterising an *individual* animal for such a parameter would require the repeated measurement of the associated traits on that animal while taking it through a series of nutritional regimes or body weight trajectories and recording its body composition, and this is often prohibitively difficult if not physically impossible. Hence the experimental quantification of within-population *variation* in such traits soon becomes an equally demanding task.

3.1. Inverted modeling

An alternative to the direct measurement of model parameter-related traits may be found in "inverted modelling", or "reverse simulation" as it was called by Bourdon (1998). In animal science, this notion goes back to Baldwin (1976), who suggests that estimates of model parameters can be obtained as follows: "assign initial values to unknown parameters in the model; compute, using these values and diet input data, [...] body energy change estimates; compare these computed estimates with experimental data for each diet input and compute from this comparison, an error of estimate; and allow a computer routine to systematically adjust parameter values in sequential (iterative) solutions until differences (error) between computed and real data are minimized (optimization)". This approach is an iterative one. The model is "inverted" in the sense that (i) phenotypic observations on traits that conventionally would be model output are now used as input to obtain prediction errors that are iteratively minimised by changing the value of some model parameters, and (ii) the final optimised values of these parameters are the output of this iterative process. The procedure is essentially similar to the familiar "fitting of equations to data" that takes place whenever empirical (regression) models are used in data analysis. Indeed, the statistical package that was used in Chapter 6 to estimate the parameters of a straightforward set of simultaneous equations (SAS, 1993) also supports fitting (and hence estimating) internal parameters of more elaborate models such as the ones used in Chapters 1 to 4, with the same least squares and maximum likelihood methods. The use of such software for data analysis would also advantageously prevent the user from over-parameterising the analysis, in terms of the ratio between the number of model parameters to be estimated and the available observations.

A more elegant way of obtaining the same would be to re-work, algebraically, the model's equations, to end up with the fundamental parameters (rather than the observations) on the left-hand side, and coding this inverted model as a new computer program. The most important advantage of this analytical approach is that the resulting program directly produces a unique solution without any need for iteration. As a consequence, "analytical model inver-

sion" has attracted much attention from a wide variety of scientific and engineering disciplines, with literature dating back to the late sixties (recent references are Groetsch, 1993, and Hornung, 1996). The problem with this approach is that many differential equation systems (which is what growth models essentially are) cannot be inverted analytically without serious difficulties. Many of those problems are "ill-posed". Ill-posed mathematical problems commonly have either no solution at all, or a series of non-unique solutions, or solutions that are unstable relative to the delivered input (Tikhonov and Arsenin, 1977).

If an analytically inverted pig growth simulation model were to be provided with input in terms of final body weight and backfat depth and cumulative feed intake, and with a description of the average nutritional and climatic conditions during the growth period, the model is likely to produce a range of possible genotype characterisations that match those phenotypic and environmental specifications. All those genotypes would eventually realise the specified phenotypic performance, but they may differ in the way they arrive there. An iterative (rather than analytical) inversion approach would yield a flat optimum in this case, with the same inconclusive results. What is required in such a case is a more exhaustive description of the phenotype (and of the associated environmental conditions), in order to make the matching process in the inverted model more powerful. This would call for repeated measurement of phenotype and environment along the growth trajectory, providing the system with more animal-intrinsic information to help it focus on animal-intrinsic model parameters.

Of course, all the above holds for model parameter variation as well as for the corresponding mean levels. The values of the coefficients of variation of the growth potential parameters (P_∞, $R_{L\infty/P\infty}$ and B_{Gomp}) used in Chapter 4 were arrived at by a similar iterative process as described above, albeit without formal convergence criteria (Figure 3 of that Chapter). See also section 5.3.

3.2. Variation in maintenance requirements

Residual feed intake (RFI; Koch *et al.*, 1963) can be regarded as an approximation of ME_{maint} (Luiting, 1991): compare the expression $MEI = b_P \times P_{dep} + b_L \times L_{dep} + ME_{maint}$ with the expression $MEI = b_{GR} \times GR + b_{comp} \times BodyComp + RFI$ (where GR is growth rate, BodyComp denotes some measurement of body composition, commonly backfat depth or lean content, and RFI is expressed on an energy basis). The variation of RFI then approximates the variation of ME_{maint}, with a bias that depends on the equivalence of $[b_P \times P_{dep} + b_L \times L_{dep}]$ with $[b_{GR} \times GR + b_{comp} \times BodyComp]$.

Expressing the between-animal standard deviation of RFI as a fraction of the estimated ME_{maint} population mean then yields an (over-)estimate of the coefficient of variation of ME_{maint}. Estimates of such coefficients of variation (CV) of ME_{maint} derived from RFI, together with coefficients of variation of directly measured ME_{maint} in various species are summarised in Figure 4.

Variation was expressed in terms of the CV because the various sources behind this Figure expressed energy intake and ME_{maint} in widely different, and sometimes undocumented, units; calculating CVs was the only way to make them all comparable. Figure 4 also summarises the published heritability estimates for these traits.

It follows from this Figure that (i) ME_{maint} in immature growing mammals shows a phenotypic coefficient of variation within populations of about 0.1, that (ii) the CV of RFI has the tendency to overestimate this parameter, as expected, and that (iii) given the heritability estimates, about 30 % of the variance of ME_{maint} in these animals is of a genetic nature.

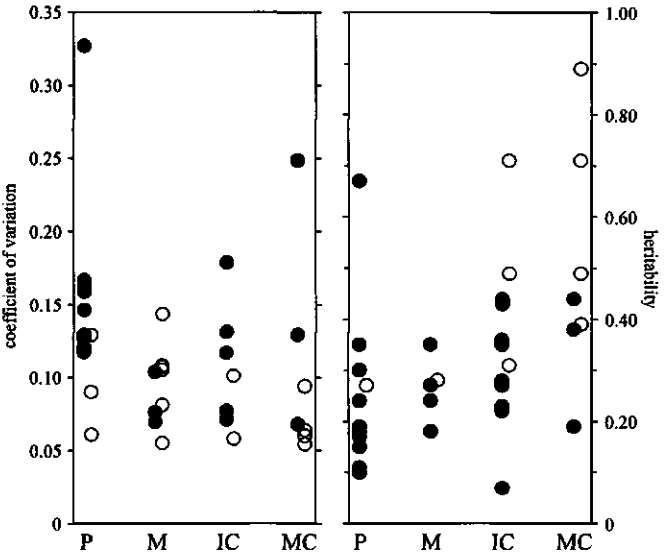


Figure 4 Estimates of phenotypic coefficients of variation (left) and heritabilities (right) of ME_{maint} (○) and residual feed intake (●) in immature pigs (P), mice (M), and immature (IC) and mature (MC) cattle. Data from Archer and Pitchford (1996), Archer *et al.* (1998), Arthur *et al.* (1997), Bech Andersen (1980), Bishop and Hill (1985), Brelin and Brännäng (1982), Brelin and Martinsson (1986), Buttazzoni and Mao (1989), Carstens *et al.* (1989), Cleveland *et al.* (1983), De Haer *et al.* (1993), De Vries *et al.* (1994), Fan *et al.* (1995), Foster *et al.* (1983), Geay (1984), Hastings *et al.* (1997), Hoffmann *et al.* (1993), Jensen *et al.* (1992), Johnson *et al.* (1999), Koch *et al.* (1963), Korver *et al.* (1991), Kownacki and Keller (1978), Labroue *et al.* (1999), Leuthold *et al.* (1994), Mrode and Kennedy (1993), Nielsen *et al.* (1997), Rauw *et al.* (2000), Renand *et al.* (1996), Robinson *et al.* (1997), Saama *et al.* (1993), Stephens (1991), Stephens *et al.* (1988), Taylor and Young (1968), Taylor *et al.* (1981), Thorbek (1975, 1980), Van Arendonk *et al.* (1991), Van Es (1961), Veerkamp *et al.* (1995) and Von Felde *et al.* (1996).

According to the simulation results from Chapters 2 and 4, the variance of thermoneutral ME_{maint} that is related to variation in body (growth) composition, and mediated through protein turnover, is about 55 $[kJ.kg^{-0.75}.d^{-1}]^2$ (see Table 5 of Chapter 4). As we will see in section 6, protein turnover is not the only process that must be expected to vary with varying intensity of protein metabolism; there are several other metabolic functions that must be expected to lead to "associative costs of protein synthesis".

It is argued in section 6 that the mean level of the associated energy requirements is at least as high as our parameter ME_{turn}; the contribution of these functions to the variation in ME_{maint} may then be at least as large as that of protein turnover as well. The variance of ME_{maint} as related to body (growth) composition in healthy growing pigs can then be tentatively estimated as 110 or possibly 150 $[kJ.kg^{-0.75}.d^{-1}]^2$. This corresponds to a standard deviation of 10.5 to 12.2 $kJ.kg^{-0.75}.d^{-1}$; with a mean value of 504 (Table 2 of Chapter 4), the partial CV of ME_{maint} as related to body (growth) composition would then be 0.02 to 0.025.

This leaves a considerable proportion of the total ME_{maint} variation (CV = 0.1 from Figure 4) unaccounted for. In modeling, this variation should rightfully be represented as another stochastic parameter in addition to the ones introduced in Chapter 2 and 4.

Ignoring the protein pool and lipid depot proportioning (which turned out to be minor sources of variation anyway), the only stochastic elements in Chapter 2 are $P_{dep,max}$ and $R_{L/P,min}$ and

in Chapter 4, P_∞, $R_{L\infty/P\infty}$ and B_{Gomp}.
Hence all variation in the simulated system is triggered by body composition traits; variation in ME_{maint} independent of body composition (activity, coping with social stressors, most immune reactions) and variation in the requirements of membrane transport do not play any role. Nevertheless, the simulated variation is compared to "real life" variation, explicitly so in Figure 5 of Chapter 4, in order to parameterise the coefficients of variation of the growth potential parameters (P_∞, $R_{L\infty/P\infty}$ and B_{Gomp}). This should be improved upon in further model development; see section 3.4.

3.3. Partitioning the variation in maintenance requirements

Figure 5 gives a tentative partitioning of ME_{maint} of growing pigs into the processes discussed up to now, based on the simulation results of Chapters 1 and 3 and on data from Beisel (1985), Baracos *et al.* (1987), Kluger (1989) and Demas *et al.* (1997). The four parts on the left of the major plot (service functions, protein turnover, membrane transport and basal activity) are meant to represent thermoneutral, welfare-friendly and healthy individual housing conditions. The fifth part of the major plot contains functions that may come in addition to that first group of four, when the environment becomes less optimal.
Its possible subdivision is in the minor plot, which has the same area as the "additional functions" part of the major plot. Thermoregulation in Figure 5 applies to continuously cold conditions; hot thermoregulation is more difficult to visualise because it reduces maintenance requirements rather than adding to them (see Chapter 3), but the magnitude of its impact is of the same order as of cold thermoregulation. Immune response in Figure 5 applies to conditions of continuous (chronic) subclinical infection. In real life, pigs are rarely cold or subclinically ill *continuously*; so the "additional" fractions in Figure 5 must be seen as upper limits.

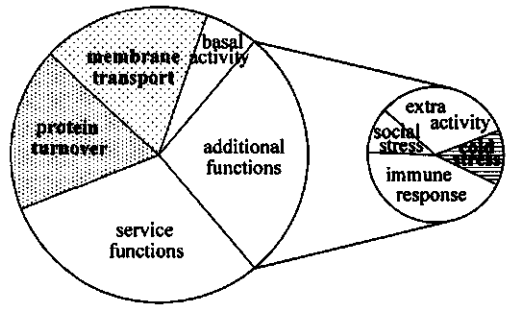


Figure 5 Proposed partitioning of ME_{maint} in growing pigs.

The partitioning of the between-animal variation in ME_{maint} is even more speculative than the partitioning of its mean. But the first parts of the variation-oriented counterpart of Figure 5 can now be filled in, based (i) on Figure 4 in this chapter, which suggests an overall CV for ME_{maint} of 0.1; with a mean value of 504 $kJ.kg^{-0.75}.d^{-1}$ (Table 2 of Chapter 4) this leads to a ME_{maint} variance of $(0.1 \times 504)^2 = 2540$ $[kJ.kg^{-0.75}.d^{-1}]^2$, (ii) on the simulation results from Chapters 2 and 4, which quantify the variation due to protein turnover and thermoregulation as variances of about 55 and 2 to 3 $[kJ.kg^{-0.75}.d^{-1}]^2$, respectively, and (iii) on the surmise in section 3.2 that the membrane transport-related variance has at least the same magnitude as the protein turnover-related portion. The resulting partitioning is in Figure 6, which thus summarises the results that the study behind this thesis intended to produce. Only to facilitate the comparison of the corresponding portions, both plots in this Figure have been drawn the same size as their counterparts in Figure 5.

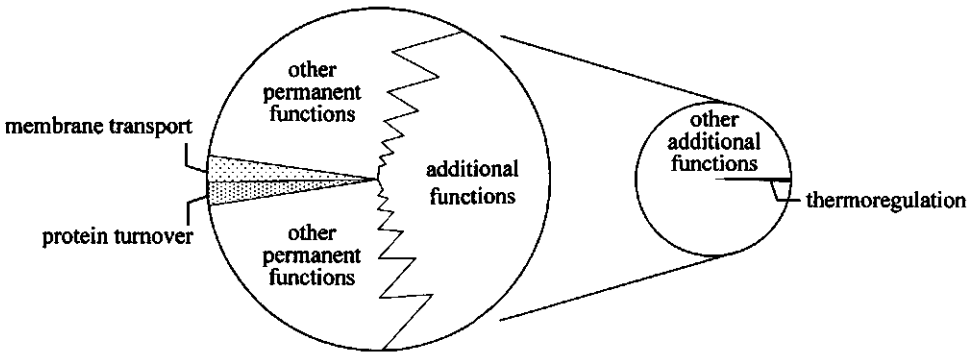


Figure 6 Proposed partitioning of the within-population variance of ME_{maint} in growing pigs. The jagged lines indicate uncertainty about the division among these components. See the text for further details.

The contribution of protein turnover to the variation of ME_{maint} derives from Chapter 4 and is caused by variation in body (growth) composition only. Specifically, between-animal variation in turnover rates is assumed to be absent; hence this portion may be underestimated. The contribution of membrane transport has been set, arbitrarily, just somewhat larger than the protein turnover fraction (as in Figure 5), and may therefore be underestimated as well. The "other permanent functions" in the major plot of Figure 6 comprise the service functions plus basal activity, as in Figure 5. The magnitude of this variance, as compared to the variance due to the additional functions that are triggered by sub-optimal environmental conditions, is very unclear. Hence the jagged division lines in Figure 6; the portion of the "additional functions" may well have to be larger than suggested here. Physical activity is probably the most important single source of variation in healthy thermoneutral pigs (De Haer *et al.*, 1993; see below). The minor plot in Figure 6 shows that the contribution of thermoregulatory metabolism (cold or hot, the relevant variances from Table 5 of Chapter 4 have been averaged) to the variance of ME_{maint} is very small. As was concluded in Chapter 4, the associated variance comprises about 4 % of the variance of the protein turnover requirements (as in the major plot in this Figure).

It follows from Figure 6 that variation in body (growth) composition does not have a major influence on the between-animal variation in ME_{maint}. It was mentioned in section 4 of the Introduction chapter that certain immune functions may have some relation to body composition, but these are not represented in Figure 6. In addition, as mentioned above, the protein turnover and membrane transport variance portions in Figure 6 are probably underestimates. But the conclusion that started this paragraph would not change even when the combined effect of an adjustment for all this would be a two- or threefold increase in the body composition-related portions in Figure 6.

The above findings may explain some incompletely understood aspects of residual feed intake (RFI) measurements in growing animals. Foster *et al.* (1983), De Haer *et al.* (1993), Mrode and Kennedy, (1993), De Vries *et al.* (1994), Von Felde *et al.* (1996), Labroue *et al.* (1999) and Johnson *et al.* (1999) studied RFI in growing pigs, as the residual term of a regression of feed intake on body weight plus production traits. The latter term is commonly a combination of (i) growth rate and (ii) a measure of off-test body composition such as backfat depth or car-

cass lean content. It was argued in section 3.2 that such a combination provides a less-than-perfect representation of the protein and lipid deposition processes of physiological interest. The R^2 values reported by the above mentioned authors range from 0.06 to 0.71, dependent on the design of their regression model and on experimental (housing and feeding) conditions. The average figure of $R^2 = 0.4$ indicates that only about 40 % of the variance in *ad libitum* feed intake of growing pigs is typically due to variation in production traits such as growth rate and fat depth or lean content. This value is much smaller than what is commonly found in productive mature animals such as laying hens or lactating cows (see Luiting, 1999).

The average within-population variance of simulated thermoneutral daily ME intake from Chapter 4 is 4902 $[kJ.kg^{-0.75}.d^{-1}]^2$ (not shown in Chapter 4). The associated variance in RFI would then be 60 % of this, *i.e.* 2941 $[kJ.kg^{-0.75}.d^{-1}]^2$. Following Luiting (1991), variation in RFI in growing immature animals comprises (i) variation in growth efficiency, (ii) variation in ME_{maint} and (iii) measurement error.

With regard to point (i), variation in growth efficiency is largely due to variation in body growth composition because of the different ME requirements of protein and lipid deposition; see equation (1) in Chapter 3. As argued at the start of section 3.2, the associated variation that ends up in estimated RFI is a reflection of the less-than-perfect representation of the physiological processes that are actually taking place ($b_P \times P_{dep} + b_L \times L_{dep}$) by the entities that are commonly used in RFI analyses ($b_{GR} \times GR + b_{comp} \times BodyComp$).

With regard to point (ii) above, it was shown in Chapter 4 that the variation in ME_{maint}, which was derived as 2540 $[kJ.kg^{-0.75}.d^{-1}]^2$ earlier in this section, includes a portion that depends on body (growth) composition as well. In section 3.2 this portion was quantified as 110 to 150 (say, 130) $[kJ.kg^{-0.75}.d^{-1}]^2$. The fraction of this variance that is directly related to the deposition rates would be included in RFI (rather than properly dealt with by the regression analysis) only with the less-than-perfect regression models mentioned above.

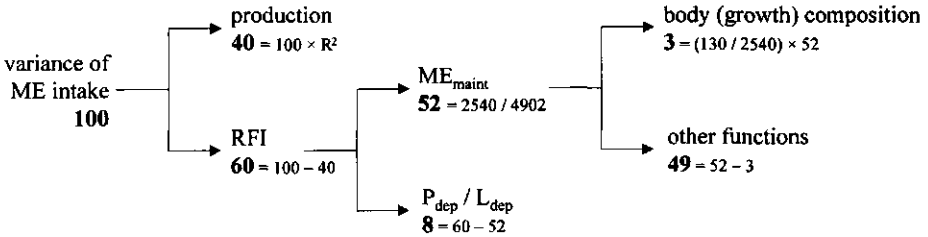


Figure 7 Proposed partitioning of the within-population **variance** of *ad libitum* ME intake (set at 100) of growing pigs. The figures shown for 'production' and 'RFI' are averages of values found in the literature. These differ considerably among experiments, dependent on the design of the regression model to estimate RFI and on environmental conditions; see the text for further details. RFI: residual feed (ME) intake; production: processes represented by growth rate plus backfat depth or body lean content; P_{dep}/L_{dep}: composition of body growth (protein *versus* lipid deposition).

The above reasoning leads to the partitioning of the variance in *ad libitum* ME intake of growing pigs shown in Figure 7. The proportion of the variance of RFI (feed intake adjusted for growth and body composition) that is still associated with body composition and with the composition of body growth is (3 + 8) / 60 = 18 %. This is much more than what is commonly found in productive mature animals (see Luiting, 1991, 1999) that do not show much body

growth and often vary less in body composition than growing animals do. This consequence of a less-than-perfect representation of body (growth) composition in the regression model is one of the reasons why R^2 values for the regression of feed intake on production traits (*e.g.* the value of 40 in Figure 7) are usually much higher in mature animals. With a perfect representation, the RFI regression model would have been based on $[b_P \times P_{dep} + b_L \times L_{dep}]$ and body protein and lipid mass, and the anomalous 3 + 8 = 11 units would have been included in the 'production' term in Figure 7. The values 40 and 60 in that Figure would become 51 and 49, respectively and, apart from measurement errors, RFI variation would be fully equivalent to ME_{maint} variation, the estimate of which would then be fully independent of body (growth) composition.

It follows that about half of the variance in *ad libitum* feed intake of growing pigs must be due to variation in maintenance-related processes that are independent of body (growth) composition. As mentioned above, an important component of these "other functions" in immature pigs is physical activity. De Haer *et al.* (1993) report that 44 % of the variance in RFI of their group-housed pigs was due to variation in feed intake activity alone; in Figure 7 this would be a value of 26 and the corresponding value for total activity would be somewhat higher, perhaps up to 40. Again, the corresponding value for mature animals must be expected to be lower.

3.4. Further developments in stochastic modeling

Another conjecture from Figure 7 is that the variation in ME_{maint} of growing immature animals is at least as important a cause of variation in their *ad libitum* feed intake as variation in their growth-related processes (the emphasis on variation is important). But pig growth modelers seem to have concentrated on the description of the growth potential, as is illustrated by the succession of papers that follow and/or attempt to improve upon the linear-plateau concept of Whittemore and Fawcett (1974): Black *et al.* (1986), De Greef (1992), Walker and Young (1993), Kyriazakis *et al.* (1994), Quiniou *et al.* (1996), Möhn and De Lange (1998) and Van Milgen *et al.* (2000), among others. By contrast, the whole aggregate of maintenance functions is often condensed into a single function of metabolic body weight or body protein mass. Given this imbalance of developmental activities, future dynamic modeling should focus on a more comprehensive description of maintenance processes rather than on an even more detailed description of the growth potential. As was found in Chapter 4 (see the start of its Discussion section), the very different sets of potential growth rules used in Chapters 1 and 3 produced surprisingly similar simulation results, up to and including the within-genotype variation of protein and lipid deposition and implied maintenance requirements. See also the *Adequacy of the model* section of the Discussion in Chapter 2.

It was concluded at the end of section 3.2 that the CV values of the growth potential parameters that were arrived at in connection to Figure 5 of Chapter 4, are inflated with the variation that should rightfully have been attributed to the body composition-independent ME_{maint} aggregate. The next step in the development of this stochastic model would be to add the latter variation as an explicit source of variation; the CVs of the growth potential parameters can then be re-parameterised. Considering the processes visualised in Figures 5 to 7, the steps after that should be the mechanistic modeling of the energetics of membrane

transport, physical activity and immune system activation, and their variation. Membrane transport and immunocompetence are further discussed in sections 6 and 7.

Simulation models require data to be parameterised. At the same time, it must be recognised that existing data becomes less representative over time; Turnpenny *et al.* (2000) write: "The breeds referred to in the [literature] are no longer used, and the breeds used today partition energy [...] differently. Increased growth rates of [pigs] result in higher metabolic heat production and [...] future work should concentrate on collecting comprehensive up-to-date heat loss data from animals rather than on further theoretical modelling". It should be obvious that any model *extension* will increase the information requirement for a proper model parameterisation even further. It follows that models should *not* be extended without a simultaneous proportional increase of evidence, *i.e.* experimental data. Our inclusion of between-animal variation in the partitioning of the body protein pools and lipid depots in Chapters 2 and 4 are good examples of model extensions that did not obey that rule. It may be sufficient for research models such as the ones in this thesis to derive their data from the literature, but as soon as such a model is applied to a specific pig population (which turns it into an *application model*) it may be wiser to remove such routines from the program, and accept an incomplete predictive functionality until that population has been properly characterised for the relevant traits. Failure of model developers to exert such discipline may easily lead model end-users to the type of disappointments that led Conceição (1997) to propose that "the power of modelling techniques in the understanding of biological systems will be undermined by premature attempts to use models to predict the behaviour of the systems". Of course for model developers, such "premature" attempts are precisely the way to find out if their models are yet good enough.

4. Protein and lipid partitioning: Chapter 5

The repeatabilities of body protein and lipid partitioning into various pools and depots were approximated in Chapter 5 by estimating the corresponding degrees of genetic determination (I^2). The association between these two parameters (repeatability and I^2) is not entirely clear, because it depends on the magnitude of maternal effects, other common litter environmental effects, and permanent environmental effects that act during the growth period, and such effects are difficult to quantify. Another cause of doubt about the validity of these I^2 estimates as reflections of repeatability is the poor information infrastructure of the data set analysed in Chapter 5, as discussed there in more detail. Hence it was concluded in Chapter 5 that the estimates need to be confirmed by corresponding ones from other data. The I^2 estimates themselves may be of use as *a priori* parameter values in order to design the trial that would be required to verify them.

Computerised tomography (CT; see Allen, 1990, for a review) can be used for the repeated measurement, within an individual growing pig, of the protein mass and the lipid mass in those tissues that can be easily circumscribed so as to separate them from the surrounding body mass. These would be skeletal muscle, adipose tissue (subcutaneous, abdominal, intermuscular), the liver, and the kidneys. Tissues such as the gastro-intestinal tract, the central nervous system and blood (interesting from a point of view of protein turnover) will be more or less indistinguishable from the digesta, bones, and virtually all other tissue, respectively. All tissue protein will be slightly "contaminated" with blood protein, which will lead to over-

estimates of tissue protein by up to 3.5 % according to the proportions in Table 4 of Chapter 1, assuming an even distribution of blood through the other tissues.
So we focus here on muscle protein and fatty tissue protein, and on subcutaneous lipid and intermuscular lipid. Table 2 of Chapter 5 gives I^2 estimates of about 0.5 for the protein pools, and about 0.3 for the lipid depots. We can substitute these figures into the formula for the standard error of the estimator of repeatability, which is approximated by

$$se_r \approx \sqrt{\frac{2\times(1-r)^2\times[1+(m-1)\times r]^2}{m\times(m-1)\times(n-1)}}$$

for a balanced design (Becker, 1984). In this equation, r denotes the repeatability itself, n denotes the number of animals in the data set, and m denotes the number of observations per animal. This can be rearranged to obtain the required number of observed animals (n_{req}) for given values of r, m, and se_r as

$$n_{req} = 1 + \frac{2\times(1-r)^2\times[1+(m-1)\times r]^2}{m\times(m-1)\times se_r^2}$$

A standard error less than 0.06 (an arbitrarily chosen level of 20 % of the estimate) for a repeatability of 0.3 would then require data on at least 67 animals with five observations each, or 43 animals with ten observations each. For a repeatability of 0.5, a standard error of 0.10 would require 24 and 18 animals, respectively. The total cost of the experiment may well depend more on the total number of observations to be done (m × n) than on the number of animals to be measured (n). The former value is minimised at [m=4, n=83] for r=0.3 and at [m=3, n=35] for r=0.5. The resulting data sets would contain 332 and 105 records, respectively, less than the serial slaughter data sets analysed in Chapter 5. Given access to CT facilities, such a trial would seem feasible although the processing of the images alone would require a large amount of labour.

5. Trends in growth pattern parameters: Chapter 6

The term "meat-type pigs" in the title of the journal article that this Chapter is based on (Knap, 2000) may appear to be a truism. Unlike the situation in poultry and cattle, where the distinction with "egg-type" and "dairy-type" animals is a very serious one, pigs would seem to be essentially "meat-type", and the adjective to be redundant. The point was to stress the focus on what would be called "sire lines" (as opposed to "dam lines") in modern pig breeding but could not be referred to as such because that term suggests much more specialisation than was present in the earlier genotypes studied in Chapter 6. And the concept of "non-meat-type" pigs was certainly a reality in, for example, the USA pig industry of the late seventies which triggered articles with titles like "Long-term backfat versus industry selection in swine" (Dickerson *et al.*, 1977) and "We proved the meat type hog is worth more - a lot more" (Johnston *et al.*, 1980).

5.1. Sigmoid growth functions

It was noted in the section on *Alternative models* in Chapter 6 that "daily protein deposition (the derivative of protein mass with respect to time) in pigs starts at a very low level at birth to increase rapidly towards a maximum and subsequently decline to zero. Emmans (1988) described the course of body protein mass over time with a sigmoid function in order to accom-

modate this pattern of the derivative, and [it was our interpretation that] he chose the Gompertz function because it has its point of inflexion at a relatively early stage (proportionally $1/e \approx 0.368$) of maturity. This allows for the decline of the derivative to take place much more slowly than its initial increase, which is what has been observed in real life".

The Gompertz function has some convenient statistical properties, most notably that it is easily linearised, and that its parameters can easily be standardised and interpreted in terms of Taylor's (1985) genetic size scaling theory. Animal growth studies have generated a large number of arguments in favour of, and against, the Gompertz curve and the other members of the Richards family of sigmoids (logistic, monomolecular, Von Bertalanffy), and we do not intend to discuss these here. A comprehensive treatise of the matter is the one by Fitzhugh (1976). The main disadvantage of all three-parameter Richards functions is the fixed y-coordinate of their point of inflexion. This will result in biased estimates of their other parameters if the data that they are fitted to have their point of inflexion at another level, for genetic or environmental reasons.

The four-parameter Richards function does not have this drawback, but it is notoriously difficult to fit (see Chapter 6 for examples, and also Nelder, 1961).

From that point of view, the Bridges function (Bridges *et al.*, 1986) that was advocated by Schinckel and De Lange (1996) may seem a welcome alternative as a three-parameter sigmoid with a flexible point of inflexion, in the sense that it could be fitted easily to the data analysed in Chapter 6. Figure 8 allows for a more direct comparison of the Gompertz and Bridges curves. Its left-hand plot shows the functions in their conventional form (*i.e.* weight in relation to time). One of the two Bridges curves in this Figure has the same parameters as the Gompertz curve; particularly, the y-coordinate of its point of inflexion has been set at 1/e times the asymptotic value of $W_\infty = 300$ kg. The other Bridges curve differs from the first one only in that y-coordinate, which is at $0.46 \times W_\infty$, the value estimated for the most recent (1993) genotype in Figure 4 of Chapter 6. The right-hand plot shows the same curves, transformed according to Emmans (1997; figure 1): the y-variables are now $-\ln[-\ln(\text{weight} / W_\infty)]$, and the x-variable is days after conception divided by $W_\infty^{0.27}$ according to Taylor (1985). It is clear that the distinction between the Gompertz and Bridges curves with the same parameters will only be possible at low or very high body weights; the two curves differ less than 5 % from each other between 28 and 227 kg BW. But the other Bridges curve shows a very different pattern, and would be readily distinguishable in almost any age or weight trajectory.

Figure 8 shows that the Bridges function as it was

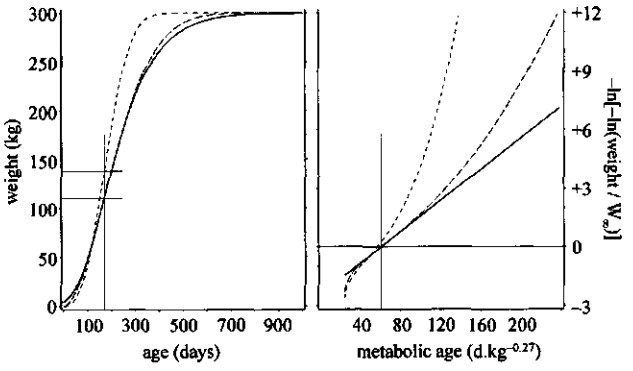


Figure 8 Gompertz (——) and Bridges curves. Parameters: asymptotic weight 300 kg, point of inflexion at 170 days of age. Y-coordinate of points of inflexion of the Bridges curves at 1/e (— —) and 0.46 (– – –) times the asymptote. See the text for further details.

fitted there does not have an intercept: it intersects the vertical axis at zero in the left-hand plot, and falls towards a vertical asymptote at zero metabolic age in the right-hand one. Adding an intercept parameter to the Bridges models for body protein and lipid mass that were fitted in Chapter 6 led to rather disappointing results, in the sense that the analyses did not converge or produced unrealistic estimates for either the intercept or the asymptotic value in more than half the cases. This is probably the reason why Schinckel (1999; figure 2.1) presents the function with a fixed intercept value of 0.22 kg body protein.
Of course, the main question is if it is *necessary* to allow for a flexible point of inflexion when describing potential protein growth. The counter-argument is that observed growth data that require such a function to be properly fitted must suffer from environmental limitations, and hence do not reflect *potential* growth. Emmans (1997; figure 1) used fetal cattle and sheep protein growth data to ascertain unlimited growth, obtained a very nearly linear fit after transformation as in the right-hand plot of Figure 8, and concluded from this that the Gompertz function is the method of choice to describe potential protein growth because it allows for (linear) extrapolation to higher stages of development with little inherent error. Considering the shape of the Bridges curves in Figure 8 in the very early growth period, it seems that this function would produce more problems when extrapolating beyond the range of observations; this would make it less generally applicable. Part of this may be overcome by introducing an intercept to the function but that would remove much of the function's appeal, as argued above.

Whichever function is adopted to describe the growth potential of the genotype of current interest, its parameters will have to be fitted to the population under study. A distinction must then be made between the estimation of (i) the mean population growth curves of body protein and lipid and (ii) the within-population variation of the associated parameters. For the former issue (i), a representative sample of the population can be evaluated by serial slaughter, similar to the trials that produced the data analysed in Chapter 6; see section 5.2. For the latter (ii), we need repeated measurements on the individual animal. Computerised tomography would work well, and isotope (such as D_2O; Susenbeth, 1984) dilution techniques could be employed too, at least for body protein; see section 5.3.

5.2. Body protein and lipid growth curve parameters

Because we must measure growth *potential*, or at least something very close to it, it should be ascertained that an adequately balanced diet is fed throughout the experimental period. This will involve regular changes in diet composition, which will have to be established in feeding trials that run a little ahead of the main experiment. Choice feeding may be a convenient alternative. The environmental temperature will have to be regularly changed as well, as much as to prevent the young pigs from becoming cold as to prevent the older ones from becoming hot (see Figures 3 and 6 in Chapter 3). And clearly, health and other stressors should be minimised, which may well require SPF conditions (see the *Adequacy of the model* section in the Discussion of Chapter 2).
It is important to notice that for the purpose of a comprehensive description, based on a sigmoidal growth function, of growth in pre-pubertal pigs it is not (i) the mature physical protein and lipid weights that must be established, but (ii) the asymptotic value of the sigmoid. As was argued in the *Data* section in the Discussion of Chapter 6, these entities are not equiva-

lent: the latter (ii) is likely to underestimate the former (i). Based on the estimated asymptotic BW of 200 to 220 kg for the more recent genotypes in Figure 3 of Chapter 6, the experiment should probably run to an end weight of at least 175 kg BW. But because the fitted shape of a sigmoid is as much determined by the initial pattern as by the later one, a low start weight (say, 15 kg or less, and preferably birth weight) is as essential as a high end weight.

Non-linear regression techniques such as the ones employed in Chapter 6 often produce poor approximations of the true standard errors of parameter estimates (Schinckel and De Lange, 1996). This was not a large problem for our study in that chapter because most of the data consisted of mean values of subgroups of several animals, which makes the standard errors of the associated estimates difficult to interpret. The emphasis in Chapter 6 is firmly on the estimates themselves, their accuracy can hardly be meaningfully determined. But when a new population is characterised this way, the standard errors of the growth curve parameter estimates will be of more interest, if only to use them for a more efficient design of subsequent trials. A simple and effective way to obtain reliable estimates for standard errors of parameters in non-linear equations is bootstrapping (Efron, 1982). In its most simple form, this involves "model-based resampling" (Davison and Hinkley, 1997; p. 262): run the (non-linear) regression analysis, obtain the predicted y-value and the residual term for each record, scale each residual term by dividing it by $\sqrt{1-h_i}$ (h_i denotes the leverage of the i-th residual term, *cf.* Neter *et al.*, 1985; p. 402), and use these to generate a large number (N) of "new" data sets. Each of these data sets holds the same records as the initial one, replacing the y-variable in each record by the sum of (i) its initially predicted value and (ii) a random drawing (with replacement) from all the scaled residuals. Each of these N data sets is then analysed with the same (non-linear) regression model to obtain N sets of growth curve parameter estimates. The means and standard deviations of these represent the bootstrap estimates of the growth curve parameters and their standard errors, respectively. See Schinckel and De Lange (1996), Thompson *et al.* (1996) and Van Milgen *et al.* (2000) for applications of the method in studying pig growth.

5.3. Variation in growth curve parameters

In Chapter 4, the range of genotype means of the growth potential parameters P_∞, $R_{L\infty/P\infty}$ and B_{Gomp} was derived from the estimates obtained in Chapter 6. For the stochastic aspect of Chapter 4's modeling, the associated coefficients of variation were set according to the outcome of a rather crude type of inverted modeling (section 3.1), comparing the predicted variation in some of the model's output traits to genetic variances of those traits that were obtained from the literature, and adjusting the CV values until a satisfactory "fit" was achieved (Figure 3 of Chapter 4). See section 3.4 for some more remarks on this procedure.
Such CV values can be estimated from similar data as described in section 5.2, given a large enough data set of "longitudinal" observations, *i.e.* repeated observations per individual. The estimation of the between-animal variation in the curve parameters can be carried out most efficiently with random regression methodology (Andersen and Pedersen, 1996; Meyer, 1998), or with Bayesian techniques such as described by Fearn (1975), Varona *et al.* (1999) and Gilg (2000).

5.4. Time trends of feed intake patterns

The *ad libitum* feed intake curves of Figure 1 in the Introduction chapter are repeated in Figure 9, and compared there to the simulated "desired feed intake" (as it was defined in Chapter 3: the intake required to satisfy both the ME and the protein demands of the genotype's growth potential and ME_{maint} at any time) curves of the five genotypes analysed in Chapter 6. Although the data in these two plots represent different sets of genotypes, there is a considerable overlap between the two in the time range covered. Comparison of these plots while ignoring their disconnection would lead to the following surmise.

The reduction of *ad libitum* feed intake in real-life genotypes from the late sixties to the late eighties observed in the left-hand plot (the three upper lines) is accompanied by a reduction of the simulated "desired feed intake" during the same period (albeit in different genotypes) in the right-hand plot. Given the patterns of the growth parameters analysed in Chapter 6 (see Figure 2 of that chapter), this must be due to a reduction of lipid deposition. These early genotypes consume less feed because they "desire" less of it, which is because they grow less fat.

The left-hand plot shows an ongoing reduction of observed *ad libitum* feed intake during the nineties (the lower two lines), but the genotypes from that period in the right-hand plot show an *increase* of simulated "desired feed intake" relative to the 1984 one.

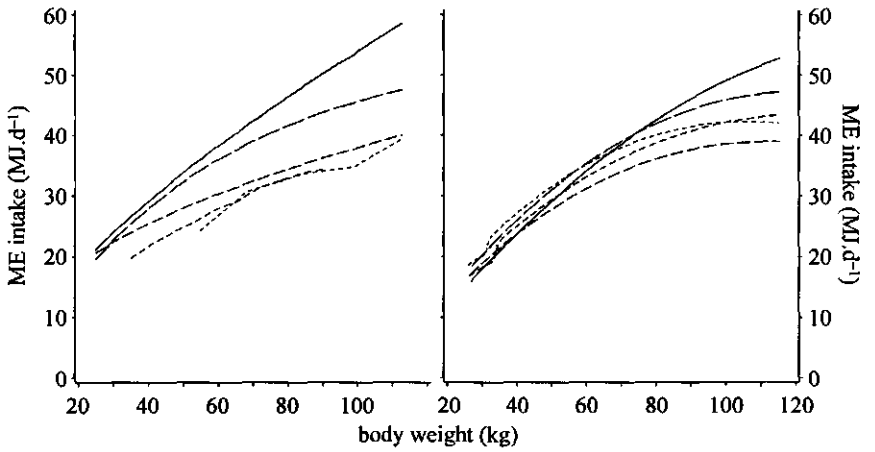


Figure 9 Daily *ad libitum* ME intake of growing pigs of five genotypes. Left: observed data reported by Cole *et al.* (1967; ———), NRC (1987; — — —), Cole and Chadd (1989; — —), Labroue (1996; – – –) and Von Felde (1996; -----); see Figure 1 in the Introduction chapter. Right: simulated "desired ME intake" (*cf.* Chapter 3) of the genotypes in Chapter 6, located in 1969 (———), 1976 (— — —), 1984 (— —), 1990 (– – –) and 1993 (-----).

There are two possible causes for this mismatch: (i) maintenance requirements independent from body composition may have decreased (the body composition-dependent portion is [partly] taken care of by the model in terms of protein turnover and thermoregulation), or (ii) feed intake capacity may have decreased. There is very little concrete evidence for or against either option, the exception being the finding by McCracken *et al.* (1994) that overfed pigs of a modern "high-lean growth" genotype did not increase their protein deposition rate relative to

ad libitum-fed controls (see section 2 of the Introduction chapter), which would count against option (ii). The only plausible reason for a reduction in body composition-independent ME_{maint} during this period with its intensified housing conditions (some of which would *increase* ME_{maint}) would be an indirect effect of selection for feed efficiency. Likewise, Luiting (1991) observed reductions in physical activity in laying hens selected for net feed efficiency (low residual feed intake). However, as was argued in section 1.3, commercial pig breeding has put little emphasis on net feed efficiency up to now.
Given the disconnectedness of the two plots in Figure 9, these surmises require experimental confirmation.

6. Energy costs of membrane transport

6.1. Membrane transport and protein synthesis

The energy cost of active transport of ions such as Na^+, K^+ and Ca^{2+} through cell membranes was recognised as a significant proportion of total energy expenditure in the early eighties (*e.g.* Gregg and Milligan, 1982). The issue was further studied by Milligan and McBride (1985), McBride and Milligan (1987), McBride and Kelly (1990) and Early *et al.* (1990), who presented quantifications ranging from 10 to 62 % of total *in vitro* O_2 consumption in several tissues (muscle, brain, kidney, liver, intestine), dependent on nutrient intake and age, among other factors. The associated contributions to whole-body energy expenditure were estimated at about 25 %.
At the same time, Reeds *et al.* (1985, 1987) commented on the results of an experiment they had carried out: "it seems [likely] that [either] the rate of protein synthesis [or] the inevitable energy costs of this process were grossly underestimated, and we conclude that other aspects of metabolism are activated when protein accretion is increased. One of these processes may be the active transport of sodium and potassium [...] This may be a major contributor to energy expenditure both in basal and metabolically activated states, and our current investigations are directed towards identifying whether a statistical relationship exists between the processes of protein synthesis and mono-cationic pumping". Somewhat later this relationship was quantified by Adeola *et al.* (1989, 1990), who report that "the contribution to total energy expenditure of the energy expended in support of transmembrane movement of Na^+ and K^+" in two separate muscles in growing pigs was 24 and 25 %, and suggest that "this may be part of the auxiliary energy expenditure unaccounted for by Reeds *et al.* (1985), which is connected but not directly associated with protein synthesis. The Na^+,K^+-ATPase dependent respiration was correlated to protein synthesis rate [$0.63 \leq r \leq 0.80$] which [...] would indicate that productive processes are closely linked with auxiliary expenditures of energy".

In accordance with this, the term *associative cost of protein synthesis* was introduced by Kelly *et al.* (1993; pp. 345-346), who write: "The membrane-bound enzyme Na^+,K^+-ATPase plays a key role in both cell mitogenesis [...] and in the co-transport of the substrates (glucose, amino acids) into living cells [...] This enzyme also accounts for a considerable portion (15-60 %) of cellular energy expenditure, and therefore is a good candidate for at least a part of the associative cost of protein synthesis [but] other energy-requiring processes also contribute to [this]. RNA turnover is a likely candidate".
It follows that the metabolic intensity (as the term was used in the Introduction chapter) of

genotypes with a higher protein deposition must be expected to be increased not only because of increased protein turnover (Chapters 1 and 2), but also because of these associative costs due to membrane transport and, possibly, several other metabolic processes.

6.2. Membrane transport and thermoregulation

A (logical) connection to the thermoregulatory functions (Chapters 3 and 4) is provided by Gregg and Milligan (1982) who studied the whole body- and muscle O_2 consumption of thermoneutral and cold-exposed sheep, and concluded that "the Na^+,K^+-ATPase of sheep muscle is a major means of energy expenditure and has an important role in the increased thermogenesis resulting from cold exposure".

6.3. Modeling membrane transport

It follows from the above that the energy requirements of membrane transport form a relevant metabolic feature to be incorporated in animal growth models, but the currently available quantitative information is rather rudimentary. The magnitude of this process seems tissue-dependent (although not to such an extent as the protein turnover rates in Chapter 1). Therefore, modeling its energy requirements could be accomplished along similar lines as for protein turnover in Chapter 1, defining tissue pools with their specific Na^+,K^+-ATPase dependent energy expenditure (*e.g.* following Early *et al.*, 1990, table 2), expanding this with transport of Ca^{2+} and possibly other ions and/or metabolites (*cf.* Summers *et al.*, 1986), and simulating the partial energy requirements in relation to tissue pool size and metabolic intensity. More data is clearly required before that can be meaningfully implemented, especially on other substances than Na^+ and K^+, and specifically on (rapidly) growing pigs.

7. Energy costs of immune system activation

The term *immunocompetence* is used here in a broad sense to indicate the capability of a host organism (*e.g.* a pig) to launch an immune response of sufficient specificity and magnitude against a pathogen, roughly indicating the effective quality of the host's immune system. It is implicitly assumed that animals vary in their genetic potential for immunocompetence.

7.1. Production potential and immunocompetence

Although the actual expression of immunocompetence will depend in the first place on the animal's genetic potential for this trait (see Knap and Bishop, 2000, for more details), there is growing evidence for environment-dependent effects of the genetic potential for production traits on the actual expression of immunocompetence. Genotypes with high production potential (suitably dubbed "metabolic athletes" by Elsasser *et al.*, 1999), when placed in an environment that is inadequate in terms of metabolic resources to support their intrinsically high production levels *and* maintain homeostasis, tend to allocate resources primarily towards production-related processes (see Coop and Kyriazakis, 1999). This will leave other metabolic functions with insufficient resources. When that environment is at the same time challenging in terms of infectiousness, the immune system may become constrained that way, which will lead to inadequate immune response to infection (Luiting, 1999; Knap and Luiting, 1999; Thorp and Luiting, 2000).

Rauw *et al.* (1998) reviewed "undesirable side effects of selection for high production efficiency" in domestic livestock, and write: "Selection for high body weight [in broiler chickens

and turkeys] has resulted in a correlated negative immune performance. Broilers selected for high growth rate showed lower antibody responses when challenged with sheep erythrocytes than a low BW line [...] and a randombred control line [...] Little or no differences were found [...] in the non-adaptive components of the immune system. Nestor *et al.* (1996ab) found a significantly higher mortality in turkeys selected for high BW compared to a randombred control line in a natural outbreak of erysipelas (11.8 and 1.6 % respectively), and when challenged with either *Pasteurella multocida* (72.1 and 43.6 %) or Newcastle disease virus (32.5 and 15.8 %)". Nir (1998) refers to selection studies for high and low antibody response to sheep red blood cells in poultry that show a negative relationship between antibody production and growth rate (Van der Zijpp, 1983; Siegel and Gross, 1980; Martin *et al.*, 1990; Kreukniet *et al.*, 1994; Praharaj *et al.*, 1995).

At present, the formal literature provides very little quantitative information on this issue in other livestock species, but tendencies towards unfavourable relations between immunocompetence and lean growth capacity in growing pigs have been reported by Stahly *et al.* (1994), Frank *et al.* (1997), McComb *et al.* (1997) and Schinckel *et al.* (1998). The results of these authors are difficult to interpret, not only because there is little information about standard errors in the reports, but also because the described trends are not always consistent between traits. The most striking result is perhaps the mortality of the pigs of Frank *et al.* (1997). Their "lean" and "fat" genotypes (with on average 57.1 and 50.7 % carcass lean, respectively) showed 3.6 and 2.8 % mortality, respectively, in a "low immunostimulation" environment (segregated early weaning, disinfected finishing facilities, etc.) and 18.5 and 5.6 % mortality, respectively, in a "high immunostimulation" environment (conventional weaning, "continuous flow finisher", etc.).
Sinclair *et al.* (1999) studied the immune response against bovine herpes virus vaccine in dairy cows of high versus average genetic merit for milk production. They concluded provisionally that the high merit cows, when given a diet low in concentrates, "redirect resources from the maintenance of an adequate immune system to milk production in order to maintain advantages in milk yield". The average merit cows did not show any dependence of immune response on diet.

7.2. Immune system activation and production

The immune system has at least three ways to actively regulate the various components of nutrient metabolism. These are, according to Klasing *et al.* (1991): (i) by direct neural connections to the central nervous system, which may trigger behavioural adaptations and/or release of hypothalamic and pituitary hormones; (ii) by release of hormones such as ACTH and thyrotropin by immune cells; (iii) most importantly, through leucocytic cytokines which trigger not only anorexia and fever but also processes like the upregulation of gluconeogenesis from glycogen, fatty acids and amino acids, accompanied by the downregulation of muscle protein deposition (and/or muscle proteolysis) to support this gluconeogenesis and to support acute-phase glycoprotein synthesis (Jepson *et al.*, 1986).
As a result, infection and the associated activation of the immune system will lead to a cascade of resource-reallocating processes in the host, most notably the following: (i) the production of acute-phase glycoproteins, immune cells and immunoglobulins, which requires extra protein synthesis; (ii) repair of damaged tissue, which may cause a strong increase in

protein turnover rates and hence increase metabolism considerably (see section 1.4); (iii) fever, with the same metabolic effect as subcritical ambient temperature; (iv) depression of voluntary feed intake (anorexia), the process with the most dramatic effects on energy metabolism.

Apart from fever (see below), the quantitative impact of these processes on nutrient metabolism is poorly documented. Demas *et al.* (1997) immunised mice with a non-pathogenic mollusc hemocyanin. Compared to non-immunised controls, immunised mice showed significantly increased specific antibody serum levels. On days 10 and 15 after immunisation they had developed a mild fever: their rectal temperatures were increased by 1.6 and 1.0 °C, and their metabolic rate (O_2 consumption) by 30 %. This increase in body temperature would by itself cause a 7 to 20 % increase in metabolic rate (see below); the remaining 10 to 23 % increase in metabolic rate would then be due to immune system activation *per se* (mainly the protein synthesis processes (i) above).

Fever constitutes an important "metabolic cost": a raise of body temperature by 1 °C causes an increase of metabolic rate by 5 to 13 % (Baracos *et al.*, 1987; Van Dam *et al.*, 1996ab; Demas *et al.*, 1997). Furthermore, fever is usually accompanied by body protein catabolism. "Only after fever begins does the nitrogen balance become abruptly negative [...] Daily losses [in humans] may then range from 2 to 23 g per day, depending on the presence of anorexia [...] and the magnitude of hypermetabolism [...] Early losses of nitrogen come chiefly from the so-called labile nitrogen pool, [...] primarily the protein in skeletal muscle and other somatic tissues. If an infection enters a subacute or chronic phase, and if the body supplies of labile nitrogen become exhausted, daily losses of nitrogen begin to lessen, and the body enters a new relatively steady state [...] Rapidly growing normal children typically exhibit a strongly positive nitrogen balance. If they become ill with an infection of mild to moderate severity, they may not revert to a negative nitrogen balance, but show a less positive balance instead" (Beisel, 1985).

But overall energy partitioning may be only little affected in the absence of fever. Schrama *et al.* (1997) reviewed trials in which the energy metabolism of growing pigs was studied in relation to immunisation with *Pasteurella multocida* toxin and various non-pathogenic substances, none of which resulted in fever. The authors conclude: "In general, the presented data [...] suggest that mild stress and/or disease first result in the reallocation of energy between different maintenance processes [*i.e.* the immunity-related maintenance processes are increased, and activity is reduced by a similar amount]. More severe stress and/or disease will result in a decreased feed intake, which often coincides with an increase in the total energy required for maintenance processes [...] The reallocation induced by exposure to stressors can conflict with the reallocation of nutrients required for the animal to maintain its health status". Anorexia during immune system activation seems counterproductive, as the body would need *more* resources to deal with its elevated metabolism and with the nutrient requirements of the pathogen. Indeed, studies have been directed to the required "upgrade" of diet composition to keep production at the economically desired levels (Klasing *et al.*, 1991; Stahly, 1996; Van Houtert, 1997), and attempts have been made to reduce the anorexia response by genetic selection (*i.e.* selection for increased resilience; Albers *et al.*, 1987; Albers and Gray, 1989; Bisset *et al.*, 1994, 1996). Kyriazakis *et al.* (1998) attempted to explain the paradox, which is especially significant from a production point of view, by suggesting that anorexia promotes an

effective immune response in the host by removing possible immunotoxic effects of micro-nutrient excesses.
All the above was summarised in terms of production traits by Stahly (1996), who discussed trials with young growing pigs subjected to high and low immune system activation (achieved by conventional weaning and medicated early weaning, respectively; Williams *et al.*, 1997ab). He concludes that "minimizing the activation of the pig's immune system" in those trials resulted in higher *ad libitum* feed intake, growth rate, muscle development, and feed efficiency.

Most current simulation models of animal growth metabolism deal with the resource requirements of the animal, given its genetic potential, in addition to the resource demands of various environmental "load" factors (most notably, nutrition and climate, see Chapter 3). Immunological costs have not been included in any of these models yet, although Black *et al.* (1999) hint at its possibilities. Modeling the resource demands of an immune system activation in a similar manner would then require a quantitative description of the ways the immune system interacts with nutrient metabolism, in terms of the four resource-reallocating processes listed above (protein synthesis, tissue repair, fever, anorexia). This interaction is likely to depend on environmental factors (type of pathogen, other load factors, pathogen density in the case of macroparasites) and on animal-intrinsic factors (immunocompetence potential, nutritional status).
Modeling these processes is of interest in an animal breeding context because the genetic evaluation of breeding animals is based evermore often on performance data that have been created in various environmental settings. The system to be described in such evaluations (*e.g.* a lactating cow, a growing pig, a laying hen) is non-linear in terms of its metabolic processes and their genetic background. When health-related factors are included in the description, the system also becomes strongly environment-variant. This makes the use of linear additive models to describe such systems questionable, and calls for the development of stochastic, dynamic, mechanistic simulation models. Similar arguments hold for the prediction of performance of a particular genotype in novel conditions. Ultimately, what needs to be modeled is genotype by health environment interaction.
In modeling, the distinction between clinical disease (with its extreme but usually short-lasting consequences) and subclinical disease (with its less extreme but chronic consequences) is important. Severe fever is a common aspect of clinical disease, and possibly the most important one in terms of resource demands. Because fever is usually not severe in subclinical disease conditions, the metabolically most important consequence of subclinical disease is likely to be anorexia.
An interesting question is if there are interactive effects around resource reallocation in case of infection: do specific production-related processes become suppressed selectively? If so, the environment-variant aspects of the infectious scenario become even more relevant. Beisel (1985) writes: "...the function of [...] host defensive mechanisms [...] requires an ongoing capacity of body cells to synthesize new proteins. For this reason, any nutritional deficit or imbalance that influences protein synthetic functions can lead in turn to [...] a weakening of host resistance", and Coop and Kyriazakis (1999) propose that resources are preferentially (not absolutely) allocated towards (i) maintenance of body protein, including repair, replacement and reaction to tissue damaged or lost due to infection; (ii) the processes of acquisition of immunity that precede the actual immune response; and (iii) growth and reproduction, in

that order of preference. They conclude that "the ability to maintain relatively undepressed functions of growth and reproduction" (*i.e.* resilience) will depend on the extent of tissue damage due to infection, and also that this implies a strong influence of nutrition on the expression of immunocompetence. The suggested tendency of modern highly productive genotypes to allocate resources preferentially towards production-related processes (see section 7.1) would not accord with Coop and Kyriazakis's (1999) order of preference. There may be a genotype-dependent trend here, which would make mechanistic modelling all the more attractive (and difficult).

7.3. Modeling immune system activation

Empirical modeling of the resource demands of an immune system activation would require measurement of the degree of immune system activation in animals in some infectious setting, and of the associated nutrient intakes and/or production performance. The latter measurements can then be regressed on the former ones, and the resulting prediction equation can be used to predict resource requirements in future conditions. Such analyses have been performed by Leathwick *et al.* (1992) and Bishop and Stear (1999) for nematode infections in sheep. The former authors developed a model to simulate the epidemiology of nematodiasis that distinguishes between gut tissue damage (in terms of "worm burden", the number of established parasites in the gut) and immune response (in terms of the number of infective larvae ingested daily) as the components responsible for the parasite's effect on the host's growth performance. The relevant equation in this model empirically predicts host weight loss (WL) as a function of cumulative daily worm burden (WB) and cumulative daily larvae intake (LI) as $WL = a \times WB + b \times LI + c \times WB \times LI$. The constants a, b and c are derived from regression analysis of field data.

By contrast, the mechanistic modeling approach would attempt to describe the host genotype in terms of (i) its resource requirements for acquisition of immunity to infection; (ii) its resource requirements for the actual expression of the immune response, including the effects of fever and anorexia; (iii) its increased protein turnover rates due to infection, all in relation to infection with a specific pathogen. For a particular host genotype in a particular environmental setting (nutrition, climate) and infected with a particular pathogen, a mechanistic model would then predict the extra resource requirements to maintain full expression of its production potential. Failing the fulfilment of these resource requirements, it would predict the realised sub-optimal production level. Such an approach would require the description of the infectious environment in terms of a few parameters that make it possible to quantify its metabolic load. It would also require much more quantitative information about the resource demands of the various components of immune system activation than is currently available.

8. Measurement errors on independent variables in regression analysis

The large numbers of observations that would be required to verify/falsify the predicted regression coefficients between the model's metabolic variables in Chapters 2 and 4 do not encourage the set-up of an experiment with that particular goal in mind. But a first, statistically much less demanding, step would be to test if the implied effects differ between genotypes at all, which for some traits could be conveniently done by high-low sampling. An example is the experiments carried out by Kyriazakis *et al.* (1994) and Kyriazakis and Emmans (1995), who (i) measured the material efficiency of protein deposition in young growing pigs of two

extreme genotypes (Meishan and Large White-based crossbreds), (ii) found no significant differences in their parameter of interest between these genotypes, and (iii) extended this finding to the conclusion that the parameter is likely to have a constant value among most pig genotypes. Thus the need to estimate it with great precision in various genotypes is clearly absent.

Nevertheless, in the section on *Verification* in the Discussion of Chapter 2 it was noticed that "erroneous data on the X variable [in a regression model] cause it to become correlated with the error terms [...], and the resulting regression coefficients are biased; this is a statistical problem in itself that goes far beyond the scope" of that Chapter. This issue is of importance for the purpose of this thesis because due to X-errors, least squares regression coefficients estimated on real-life data carry a negative bias and may become so much deflated that they appear to be significantly lower than the simulated value to be verified. As a consequence, that simulated value would erroneously be considered to be falsified. Hence an alternative to least squares estimation that overcomes these "false negatives" may be crucial to model validation. What we attempt to do in section 8 is to illustrate such alternatives.
The issue of X-errors has been a long-standing subject of statistical study, especially in fields with notoriously large uncertainties of observations such as the social and economical sciences. A recent and comprehensive treatise is the one by Johnston (1991; pp. 428-435), who draws heavily on more formal texts by Durbin (1954) and Kendall and Stuart (1961; pp. 375-415). We present here a simplified summary of the parts from these texts that are relevant for our present purposes (sections 8.1 to 8.3), and a small example to illustrate the options for the kind of analysis that would have to be dealt with (section 8.4). Dhanoa *et al.* (1997) give a similar example for different traits.

8.1. Least squares estimators

Define b_0 and b_1 as the familiar intercept and regression coefficient, associated with X variables that are measured without error. When dealing with an independent variable *with* X-errors, the simple regression model is $y_i = b_0 + b_1 \tilde{x}_i + e_i$, where $\tilde{x}_i$ denotes the i-th true (but unobserved) value of the independent variable and e_i is the familiar residual term of the regression analysis. The associated *observed* value is $x_i = \tilde{x}_i + u_i$, where u_i denotes the measurement error. Hence $\tilde{x}_i = x_i - u_i$, and the "structural relation" between the observed variables X and Y becomes $y_i = b_0 + b_1 x_i + (e_i - b_1 u_i)$. This is not a straightforward regression, because X is now a random variable that is correlated with the extended residual term $(e - b_1 u)$. This follows from the covariance between the two: $\text{cov}(x, e - b_1 u) = \text{cov}(x, e) - b_1 \text{cov}(x, u) = -b_1 \text{cov}(\tilde{x} + u, u) = -b_1 \sigma_u^2$. Here, σ_u^2 denotes the variance due to X-errors, which will be larger than zero when measurements are imprecise.

As a consequence of this covariance, the LS estimator of b_1 is $\hat{b}_1 = \frac{b_1}{1 + \sigma_u^2 / \sigma_X^2}$, which makes b_1 "unidentifiable". The negative bias due to σ_u^2 as a proportion of the total variance of the X variable (σ_X^2) is evident. It follows that knowledge of the magnitude of σ_u^2 is necessary to obtain an unbiased estimate of the regression coefficient. An estimate of the X-error variance ($\hat{\sigma}_u^2$), together with the sample variance of the X variable, could be used to obtain an

adjusted LS estimator:

$$\hat{b}_1 = \frac{\text{cov}(x,y)}{\text{var}(x)} \times \left(1 + \frac{\hat{\sigma}_u^2}{\text{var}(x)}\right) \quad (1)$$

where var(x) and cov(x,y) denote the sample (co-)variances

8.2. Maximum likelihood estimators

Such an estimate of σ_u^2 can also be used to obtain a maximum likelihood (ML) estimator of the regression coefficient:

$$\hat{b}_1 = \frac{\text{cov}(x,y)}{\text{var}(x) - \hat{\sigma}_u^2} \quad (2)$$

Another option is to express σ_u^2 as a multiple of the corresponding error variance of the Y variable (σ_v^2). When we define the ratio between the two as $\lambda = \sigma_u^2/\sigma_v^2$, an alternative ML estimator is

$$\hat{b}_1 = \frac{\text{var}(y) - \lambda\,\text{var}(x) + \sqrt{[\text{var}(y) - \lambda\,\text{var}(x)]^2 + 4\lambda\,\text{cov}^2(x,y)}}{2\,\text{cov}(x,y)} \quad (3)$$

again a simple function of the sample variances and covariances, which would be used when it is more feasible to obtain an estimate of λ than of σ_u^2. Large-sample confidence intervals of this estimator, which are asymmetrical, have been described as well.

8.3. Instrumental variable estimators

"Instrumental variables" serve as an instrument in the estimation of the relationship between X and Y. For this purpose they should be correlated with the true values $\tilde{x}_i$, but uncorrelated with the X-error terms u_i and the Y-errors v_i, and hence provide supplementary information on X that can be incorporated in the regression analysis. Referring to our instrumental variable as Z, the regression coefficient of Y on X can be estimated as $\hat{b}_1 = \frac{\text{cov}(y,z)}{\text{cov}(x,z)}$, where cov(x,z) and cov(y,z) are the sample covariances again. Substituting the true X values, this can be re-written as $\hat{b}_1 = \frac{\text{cov}(b_0 + b_1\tilde{x} + v, z)}{\text{cov}(\tilde{x} + u, z)}$ and because Z is uncorrelated with u and v, this collapses to $\hat{b}_1 = \frac{\text{cov}(b_1\tilde{x}, z)}{\text{cov}(\tilde{x}, z)} = b_1$, so this estimator is unbiased. Within the framework of the relationship between ME_{maint} and P_{dep}, a possibly convenient candidate for an instrumental variable would be the rate of body lipid deposition (L_{dep}). The condition of zero correlation between L_{dep} and the errors in P_{dep} measurement would require these traits to be measured independently, *i.e.* lipid should not be estimated as some deficit of P_{dep} and overall energy retention. Of course, this approach would only make sense when L_{dep} could be measured with considerably lower error than P_{dep}, and it is difficult to think of *any* comparable trait that would fit that requirement.

In general, it will also be difficult to ensure that the above condition of zero correlation between Z and u is fulfilled, and a more generally applicable approach has been developed that

makes use of a numerical transformation of the observed x_i values as the instrumental variable. The most sophisticated of these transformations requires the X-errors to be small enough to ensure that the series of observed x_i values is in the same numerical order as the series of true $\tilde{x}_i$ values, in other words that their rank correlation is unity. This condition can be met, in practice, by arranging for the data points to be sufficiently widely dispersed, which would require a deliberate sampling scheme of the experimental animals.

When the x_i values are ordered in ascending order the instrumental variable can be based on their rank values (1, 2, ..., n). The extended incidence matrix of the instrumental variable is then $\mathbf{Z}' = \begin{bmatrix} 1 & 1 & \cdots & 1 \\ 1 & 2 & \cdots & n \end{bmatrix}$, and estimates for b_0 and b_1 follow as the elements of the vector $\mathbf{b} = (\mathbf{Z}'\mathbf{X})^{-1}\mathbf{Z}'\mathbf{y}$, which works out as

$$\hat{b}_0 = \frac{\bar{y}\sum i x_i - \bar{x}\sum i y_i}{\sum i x_i} \text{ and } \hat{b}_1 = \frac{\sum i(y_i - \bar{y})}{\sum i(x_i - \bar{x})} \qquad (4)$$

In these equations, $\bar{x}$ and $\bar{y}$ denote the sample means. Standard errors for these estimators follow from the asymptotic $\text{var}(\mathbf{b}) = (\mathbf{Z}'\mathbf{X})^{-1}\mathbf{Z}'\mathbf{Z}(\mathbf{X}'\mathbf{Z})^{-1}\sigma_u^2$, which requires information on the magnitude of the X-errors again.

8.4. Example: heat production versus protein deposition rate

To illustrate the above techniques we make use of data on heat production (HP) in young growing pigs, in relation to their rate of protein synthesis (not: deposition), as published by Reeds *et al.* (1980; figure 5). In this experiment, protein synthesis was measured from the disappearance rate from the blood of infused radio-actively labeled leucine and HP from indirect calorimetry.

The left-hand plot in Figure 10 shows the observations and the LS regression line. The authors do not give any information on the precision of their measurements; for simplicity we assume here coefficients of variation due to measurement error of 10 % for both traits. With n = 20 and sample means of $\bar{x}$ = 405.3 g.d^{-1} and $\bar{y}$ = 9.70 MJ.d^{-1}, we then get σ_u^2 = 1643 and σ_v^2 = 0.942 so that λ = 0.00573.

This transforms the data points into the elliptical 68 % confidence regions depicted in the right-hand plot of Figure 10 (each ellipse in this plot is only one σ_u unit wide and one σ_v unit high for the sake of clarity). At the same time, it transforms the regression analysis into the problem of finding a straight line that intersects as many of these ellipses as possible (Kendall and Stuart, 1961; p. 385). The regression lines in this plot represent the LS regression and the estimates from models (1) to (4).

The lines from models (1) to (3) pass through the point $(\bar{x}, \bar{y})$, as does the LS regression. The regression coefficient estimates from the four approaches are in Table 4. Adjusting for the bias on the LS estimate according to model (1) gives a value of $\hat{b}_1 = 0.0224 \times 1.158 = 0.0259$.

Table 4 Estimated regression coefficients (in MJ.g^{-1}) for the data in Figure 8.

method (model)	$\hat{b}_1$
LS	0.0224
adjusted LS (1)	0.0259
ML (2)	0.0266
ML (3)	0.0247
instrumental variable (4)	0.0229

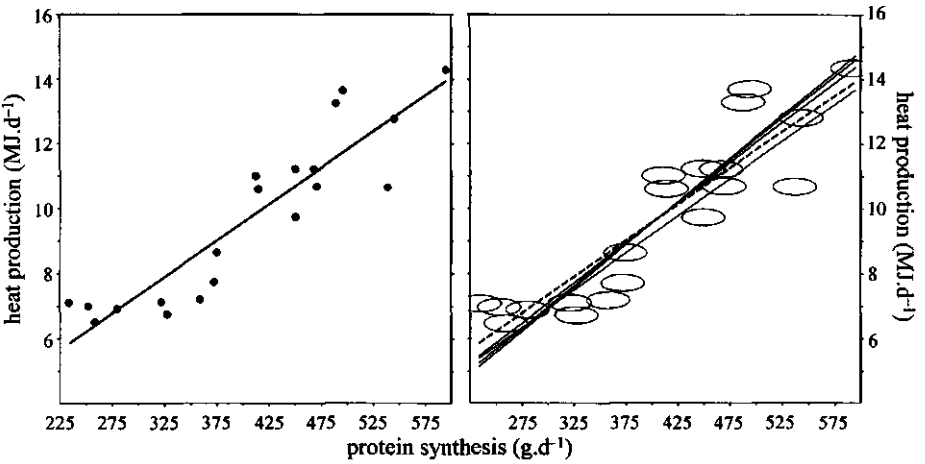


Figure 10 Heat production in relation to protein synthesis of young growing pigs. Left: values observed by Reeds *et al.* (1980), with the least squares regression line. Right: 68 % confidence regions for each observation (ellipses), assuming a measurement error CV of 10 % for both traits. The dotted line is the least squares regression line, the solid lines represent its adjusted version according to model (1), the maximum likelihood regressions according to models (2) and (3), and the instrumental variable regression according to model (4). See the text for details.

The two ML estimates encompass this value to within 5 %. The instrumental variable regression coefficient estimate differs little from the LS estimate (the difference is in the intercept, see Figure 10), perhaps because model (4) is strictly inappropriate for this particular data set with its considerable overlap of the data point confidence regions.

Replacing the LS estimate by any of the results from models (1) to (3) increases our perception of the regression of HP on protein synthesis in these particular data by Reeds *et al.* (1980) by 10 to 19 %, but this does not substantially alter the interpretation of these results. In other data this may be different. The main point of this whole exercise was made in the *Verification* section of Chapter 2: when experimental data are used to judge the realism of a value predicted by simulation, and the independent variable in that simulation was set without measurement error, the statistical analysis of the experimental results should accommodate that feature. Otherwise, the bias on the parameter estimates may introduce false negatives.

9. The use of growth models in pig breeding

Kinghorn (1998) discussed the use of selection indices in animal breeding, and identified two weaknesses of this linear regression approach to describing the breeding objective for a meat production system.

First, many "traits of true importance, such as mature size, shape of the growth and feeding curves, and the patterns of tissue deposition" (which correspond closely to the growth potential parameters of the model used in Chapters 3 and 4) cannot be reliably measured and hence their genetic (co-)variances cannot be established. As a consequence, "such traits are often ignored when developing breeding objectives and yet their direct or indirect effect on profit can be large".

Second, the biological interactions among traits in meat production systems are often not lin-

ear. As was also noticed by Bourdon (1998), describing an essentially non-linear system with a linear model may result in a satisfactory fit within a narrow parameter space but will require frequent re-fitting when the system moves through a wider space: linear genetic (co-)variances for any population undergoing genetic change must regularly be re-estimated. The same phenomenon results in genotype by environmental interactions.
Kinghorn (1998) then suggests that "biological modeling of production systems can be used to predict such changes" in genetic patterns and relationships, and "can be used to set breeding objectives" or "might play a quality control role, to predict deleterious effects of breeding objectives set through use of" selection index theory. For example, Fowler *et al.* (1976) reasoned that "the advantage of attempting to identify pigs with a low maintenance requirement in a breeding programme would be reduced" in the case of "a genetic correlation between high rates of lean tissue synthesis and higher maintenance requirements"; this is an optimisation problem that may be resolved by biological modeling. Ball and Thompson (1995) and Ball *et al.* (1998b) attempted just that and concluded from their simulation studies that "any adverse effects of increased maintenance costs by selection for leaner sheep was more than outweighed by the increase in biological efficiency".

Much earlier, economic values for traits of linear breeding objectives had been estimated from the predictions of the bio-economic models initiated by Dickerson (1982) and further developed by Tess *et al.* (1982). These models embed a growth (and reproduction and lactation) model similar to the one used in Chapters 1 and 2 into a model of a closed breeding-finishing herd, and evaluate the effect on overall unit profitability of a change in a trait represented in the biological part of the model. A similar approach was followed by De Vries (1989).

The proper specification and parameterisation of breeding objectives may be regarded as one of the most crucial features of animal breeding, but there are at least four other aspects of a breeding program that may benefit from the use of simulation models such as the ones described in this thesis.

First, selection criteria must be based on some sort of estimated breeding value, usually a weighted average of phenotypic production traits as observed on the selection candidate and on its relatives. As was argued in section 3.1, dynamic growth models may be "inverted" and used to predict the value of the internal parameters based on input of observed growth-related traits (the phenotypic production traits mentioned above) plus a characterisation of the environment in which those traits were created. If sufficient information can be produced to support such modeling on the individual animal level, the resulting growth potential parameter estimates can be used as selection criteria. The combination with information from relatives could be accomplished after the inverted model runs or, preferably, be integrated with it. The latter would require a considerable developmental effort but might result in a non-linear alternative to the linear BLUP procedures currently used for breeding value estimation.
Second, the current search for quantitative trait loci (QTL; see Visscher and Haley, 1998) is largely based on association of these genes with production traits such as growth rate. This is largely a reflection of the current availability of data sets of satisfactory size and structure to support QTL detection. However, the real benefit of molecular genetics for livestock breeding is not in intensifying genetic change in traits that can easily be measured phenotypically and hence can easily be selected for by conventional means, but in effecting genetic change in

traits that cannot (see also Albers, 1998). Fitness-related (particularly immunocompetence-related) traits provide a very typical example of this (Knap and Luiting, 1999), but the search for QTL for growth-related traits could be made much more efficient when associations were made with the same growth potential parameter estimates as mentioned in the previous paragraph. Since the simulation model adjusts for environmental "noise", and these parameters are supposed to reflect the growth *potential* (a fully genetic characteristic), their estimates should be much closer associated to the gene level than phenotypic observations can be.
Interestingly, the combination of the above two points suggests that dynamic simulation models may provide a convenient tool to quantify the much-needed biological connection between quantitative and molecular genetics on the breeding company level.

Third, the environmental conditions in which the above mentioned observations of growth-related traits are created can have a profound effect on the degree of expression of the associated genetic potentials. Hence performance testing regimes have been the subject of regular changes since the sixties, often with the intent to increase that degree of expression (see Knap and Van der Steen, 1994) as much as to keep up with commercial management conditions. Stochastic growth models such as the ones described in Chapters 2 and 4 can be used to evaluate the effect of changes (intentional or not) in environmental conditions such as diet composition, feeding level, climate, health, group size, and body weight ranges on the degree of expression of the potential for any growth-related trait of interest. This would require a proper parameterisation of the model for the genotype under concern, which would call for inverted modeling (section 3.1) first.
Fourth, when the production environment can be properly characterised, and the model is truly mechanistic, it would be possible to evaluate various genotypes and their suitability for a given set of market conditions, and *vice versa* to specify the optimum management conditions for a given genotype. Closely linked to this application, field performance data of a specific genotype as collected in different environmental conditions could be mechanistically adjusted towards a standardised assessment of the genotype's environmental sensitivity.

The above options seem important enough to warrant further model development directed towards such applications. This would specifically require a focus on stochastic simulation.

10. Concluding remarks

This study was set up with the intention to quantify the impact of body (growth) composition on maintenance requirements, by collecting the relevant information available in the literature and putting it together into a quantitative framework. The resulting stochastic simulation models allow one to work one's way through the known body of relationships and parameters, and add a quantitative dimension to the qualitative insights that can be built up from the literature without much effort.
The direct output of this exercise (*i.e.* the results of Chapters 2 and 4) has been summarised in Figure 6 of this chapter. For convenience, the values represented in that Figure are briefly repeated here: a total between-animal variance of ME_{maint} of about 2540 $[kJ.kg^{-0.75}.d^{-1}]^2$, a partial variance related to body (growth) composition due to protein turnover plus membrane transport of 110 to 150 $[kJ.kg^{-0.75}.d^{-1}]^2$, or 4 to 6 % of the total, and a partial variance due to thermoregulatory processes of 2 to 3 $[kJ.kg^{-0.75}.d^{-1}]^2$, or about 0.1 % of the total. Although leaner pigs do have higher maintenance requirements as a direct result of that virtue, this phe-

nomenon does not contribute much to the within-population variation in maintenance requirements.

Apart from its concrete predictions, the simulation methods used here provide a convenient tool for prioritising R&D effort; when used with care, they may also produce quite explicit formulations of experimental work to be carried out. In each of the previous six chapters of this thesis it is concluded that the quantitative information that would be required to properly parameterise the evaluated models, does not seem to be available, and in each case it becomes quite clear which information would be needed to fill in the gap. It might be argued in general that experiments are most needed to satisfy two goals: (i) to create information that was identified by literature studies (of which simulation is perhaps the most structured form) as being both crucial and missing, and (ii) to test hypotheses that result from such studies. After integrating the experimental results into the model that triggered their creation, new information gaps will be identified and new hypotheses generated, and so the cycle of exploration continues. Examples of point (i) are Chapters 1 and 3, and in a different sense Chapters 5 and 6; of point (ii), Chapters 2 and 4.

As was argued in section 3.4, in many fields of interest there is a much greater need for well-structured data than for further model extensions or reformulations. But other areas would benefit from model development prior to experimental effort, simply because such development would (cheaply) identify the most pressing research needs according to the previous paragraph. This point is illustrated by the text accompanying Figures 5 and 6.
Most pig growth models deal with conversion of available nutrients in a very similar way (considering the similar outcomes of Chapters 2 and 4). This study has focused on the sinks of ME *before* it becomes available for production, which may be conveniently grouped together as maintenance requirements. As was argued in connection with Figure 7 (section 3.4), more research effort should be devoted to this issue rather than to even more elaborate descriptions of the growth potential, also because the latter are hardly verifiable.

Stochastic simulation may turn out to be a powerful tool for animal breeding applications, as argued in section 9. It would benefit this kind of application very much if experimental data were created, and published, with its dimension of within-population variation in mind.

Acknowledgements

Valuable contributions were made by Florence Labroue who supplied data for Figure 3, by Erhard Kornblum and Brian Vernon who checked some data for Figure 4, by Joan Tibau i Font who triggered the set-up of Figure 5, and by Martin Verstegen, Gerry Emmans and Ella Luiting who supplied their usual constructive comments.

Summary

In the **Introduction** chapter, it is argued that the energy requirements for body maintenance (ME_{maint}) in growing pigs must be expected to show variation between animals, part of which is related to variation in body composition. This is because the maintenance processes of protein turnover, active transport of ions through cell membranes ("membrane transport"), thermoregulation and possibly also some immune functions, are functionally dependent on the composition of the body, and/or on the composition of body growth. The question that led to the study carried out in this thesis is from Webster (1988): "to what extent [can] differences in maintenance requirements be attributed to differing proportions of the different organs and tissues of the body, each having different metabolic rates" ? We attempt to provide part of the answer by stochastic simulation, putting together the available information on variation in body composition between growing pigs, and on the way protein turnover and thermoregulation depend on body composition.

The stochastic models that are used in Chapters 2 and 4 assume certain amounts of animal-intrinsic variation in the partitioning of body protein into protein pools and of body lipid into lipid depots. In **Chapter 5** we check for the existence of such variation in real life. Results from serial slaughter trials on Danish Landrace and Danish Yorkshire pigs were used to estimate additive genetic and litter-associated variance components for several traits. These traits were total body protein and lipid mass (TOTPROT and TOTLIPD), the proportions of total body protein that are present in the muscles (PROTMUS) or in the (sub-)cutaneous tissue plus bones (connective tissue protein, PROTCON), and the proportions of total body lipid that are present in the (sub)-cutaneous tissue (LIPDSUB), in the muscles (inter- and intramuscular fat, LIPDMUS), or in the bones (LIPDBON). TOTPROT and TOTLIPD were adjusted by regression for body weight; PROTMUS and PROTCON were adjusted for TOTPROT; and LIPDSUB, LIPDMUS and LIPDBON were adjusted for TOTLIPD. The pooled estimates of the degree of genetic determination (the sum of the additive genetic and litter-associated variance components, which approximates the repeatability) of these traits were 0.48 for TOTPROT, 0.56 for TOTLIPD, 0.56 for PROTMUS, 0.57 for PROTCON, 0.32 for LIPDMUS, 0.33 for LIPDSUB, and 0.22 for LIPDBON. It is concluded that there is animal-intrinsic variation in partitioning of body protein and lipid.

The simulations in Chapters 3 and 4 distinguish between several hypothetic pig genotypes, in terms of the mean values of their growth potential parameters. In **Chapter 6** we re-analyse previously published data from serial slaughter trials on growing pigs of five genotypes to provide realistic values for those simulations. Gompertz curves were fitted to body protein and lipid mass in order to estimate mature protein and lipid mass (P_∞, L_∞) and the specific growth rate parameter (B_{Gomp}) that was presumed to be equal for the protein and lipid curves. L_∞ was expressed as its ratio to P_∞, $R_{L\infty/P\infty}$. The maximum rate of protein deposition was derived as $P_{dep,max} = P_\infty \times B_{Gomp} / e$. The analysed data encompass body weights of 10 to 133 kg, 13 to 217 kg, 18 to 106 kg, 20 to 110 kg, and 11 to 145 kg. The Gompertz function fitted these data sets well, as judged by the standard deviations and distribution patterns of the residual terms. Autocorrelations among the residuals were non-significant.
Averaged over sexes (females and entire and castrated males), the P_∞ estimates were all close to 31 kg; the $R_{L\infty/P\infty}$ estimates ranged from 1.4 to 4.7 $kg.kg^{-1}$, the B_{Gomp} estimates from 0.009 to 0.017 $kg.d^{-1}.kg^{-1}$, and the resulting $P_{dep,max}$ estimates from 110 to 193 $g.d^{-1}$. The genotypes were placed in 1969, 1976, 1984, 1990 and 1993. Plotting the estimates against

time (year) showed distinct time trends for all parameters except P_{∞}. $R_{L\infty/P\infty}$ seems to gradually decline towards a plateau around unity, whereas B_{Gomp} and $P_{dep,max}$ increase linearly. These trends were confirmed by an analysis of body weight based on the same data plus data on three other genotypes that spanned the same time period. Analyses of the same protein and lipid data to fit a sigmoid growth function with a flexible point of inflexion did not change the apparently absent time trend of P_{∞}. The estimates of the inflexion points of the fitted protein accretion curves, expressed as proportions of P_{∞}, were indistinguishable from the fixed 0.368 value of the Gompertz function for the earliest three genotypes and then showed a tendency to increase, up to 0.46 for the 1993 population.
These time trends must be the consequence of a combination of changes in nutritional and other environmental factors and genetic changes. They cannot be the sole result of within-line selection for growth and body composition traits, since this should increase P_{∞}. It seems as if pig breeders have repeatedly initiated their sire lines from genetic resources with small mature size, to subsequently increase this trait as an indirect result of within-line selection.

The actual simulations were carried out in Chapters 1 to 4.

A dynamic model for simulation of growth in pigs was extended in **Chapter 1** by a module to describe protein turnover in six body protein pools (muscle, connective tissue, liver, blood plasma, gastro-intestinal, and "other" proteins). The model describes protein deposition in these pools following different growth curves and different rates of turnover. Growth curve parameters and turnover rates were obtained from the literature.
In growing animals, experimentally measured turnover rates represent a combination of turnover of already present body protein and repeated synthesis of newly deposited protein. We attempt here to distinguish between these processes by varying the values of the fractional rate of synthesis of newly deposited protein (FRS_{dep}) and of the proportion of maintenance energy requirements not related to protein turnover ($FrcME_{maint}$), and to compare the simulated output to the output from the original model without the protein turnover module.
The turnover rate (TR_{pres}) of already present connective tissue protein reached unrealistic values for $FRS_{dep} > 2.5\ d^{-1}$, which puts an upper limit to FRS_{dep}.
The output from the extended and the original models showed similar patterns for certain combinations of FRS_{dep} and $FrcME_{maint}$, dependent on the levels of model input variables. For $FRS_{dep} \leq 2.5\ d^{-1}$ (the upper limit mentioned above), these patterns have their maximum similarity at $FrcME_{maint} = 0.65$, coinciding with $FRS_{dep} = 2.0\ d^{-1}$. The corresponding TR_{pres} values were 0.060, 0.019, 0.585, 1.492, 0.582, and 0.016 d^{-1} for the above mentioned pools.

The dynamic model extended in Chapter 1 was made stochastic, in **Chapter 2**, in order to simulate groups of pigs with between-animal variation in $P_{dep,max}$, in the minimum lipid to protein deposition rate ($R_{L/P,min}$), and in the distribution of body protein over protein pools (muscle, connective tissue, and other proteins). As a result, these simulated pigs show between-animal variation in body and body protein composition. This in turn leads to between-animal variation in energy requirements for protein turnover (ME_{turn}), which causes between-animal variation in ME_{maint} as a result of variation in body composition.
Simulated population means for $P_{dep,max}$ were varied in seven steps from 100 to 250 $g.d^{-1}$, with a within-population variation coefficient of 10 %; the feeding level was also varied in

seven steps. Dependent on the levels of $P_{dep,max}$ and the feeding level, 100 kg pigs showed within-population standard deviations in body protein and lipid content of 0.31 to 0.54 kg and 1.22 to 2.17 kg, respectively. ME_{maint} showed a protein-turnover-related within-population coefficient of variation (CV) of 1.4 to 2.0 %. Comparison over populations suggests that a 150 % increase in $P_{dep,max}$ (from 100 to 250 $g.d^{-1}$) would increase protein-turnover-related ME_{maint} by 11 to 15 %, from between 470 and 486 $kJ.kg^{-0.75}.d^{-1}$ to 541 $kJ.kg^{-0.75}.d^{-1}$.

The inferences that can be made from this with regard to experimental design are discussed. The simulated (co-)variances are used to derive the number of experimental observations that would be required to obtain an estimate for the linear regression of ME_{turn} on the rate of muscle protein deposition ($P_{dep,mus}$) significantly different from our simulated value of 21.24 $kJ.kg^{-0.75}.d^{-1}$ per kg. Because in practice neither trait can be measured without error, the influence of X-errors (*i.e.* measurement errors on $P_{dep,mus}$) must be taken into account. Such errors cause a negative bias on the least squares estimate of a regression coefficient. It was found that a coefficient of variation due to measurement errors in $P_{dep,mus}$ of more than 10 % would make it effectively impossible to experimentally verify our simulation results: as a consequence of the X-errors, the observed regression coefficient becomes so much deflated (entirely due to bias) that it will appear to be significantly lower than the value to be verified. Because the X-errors of protein deposition traits tend to be of just that order of magnitude, such experimental results require more sophisticated statistical treatment; this will receive more attention in Chapter 7.

The dynamic model of Chapter 1 was extended, in **Chapter 3**, by a module to assess maximum and minimum heat loss (HL_{hot}, HL_{cold}) for a given pig, to compare these figures to heat production (HP), and to take thermoregulatory action when $HP < HL_{cold}$ (cold conditions) or $HP > HL_{hot}$ (hot conditions). At the same time, the growth algorithm of this model was adapted to allow for the simulation of *ad libitum* feed intake and to make the $P_{dep,max}$ parameter variable in relation to age, the latter by re-formulating the algorithm in terms of the parameters studied in Chapter 6.

HL_{cold} and HL_{hot} were largely determined according to algorithms obtained from the literature, but HL_{cold} was made dependent on body fat depth through tissue insulation. Data to establish the relation ($y = 0.05 + 0.002\ x$) between cold tissue insulation (y, in $°C.m^2.W^{-1}$) and P2 backfat depth (x, in mm) independent of body weight were obtained from the literature. The same data showed that HL_{hot} is not related to backfat depth in pigs.

Cold thermoregulatory action included an increase of *ad libitum* feed intake. Hot thermoregulatory action included reduction of physical activity, increase of body temperature, wetting of a proportion of the skin, and reduction of *ad libitum* feed intake.

A sensitivity analysis showed that the model's output in terms of *ad libitum* feed intake, heat production, protein deposition and lipid deposition is strongly sensitive to the characterisation of the genotype being simulated.

The model was used to simulate trials from the literature. Although the model does not explicitly calculate the lower and upper critical temperatures, these could be adequately predicted from its output. Comparison of model output with experimental data revealed an adequate prediction of *ad libitum* feed intake and of the partitioning of *ad libitum* ingested ME into heat production, protein deposition and lipid deposition in cold, thermoneutral and hot conditions.

At restricted ME intake, and especially in cold conditions, the model tends to overestimate heat production and underestimate lipid deposition, probably because it does not take account of long-term acclimatisation.

The model extended in Chapter 3 was made stochastic, in **Chapter 4**, to simulate groups of pigs with between-animal variation in P_∞, L_∞ and B_{Gomp}, and in the distribution of body protein and lipid over pools and depots. The resulting variation in body composition leads to variation in energy requirements for protein turnover and thermoregulation, causing between-animal variation in ME_{maint}.

Simulated population means for P_∞, $R_{L\infty/P\infty}$ and B_{Gomp} were varied in 3 steps each. Excluding six unrealistic parameter combinations, this led to $3^3 - 6 = 21$ simulated genotypes. The within-population CV values of the above three parameters were se at 7, 15 and 3 %. Random replicates of each genotype were simulated five times, in climatic conditions that were subsequently severely cold, mildly cold (about 5 and 1 °C below lower critical temperature), thermoneutral, mildly hot and severely hot (about 1 and 5 °C above upper critical temperature), during the entire growth period of 23 to 100 kg liveweight. Simulated feed intake was *ad libitum*.

Simulated thermoneutral within-population standard deviations of body protein and lipid content at 100 kg body weight were 0.21 to 0.46 kg, and 0.78 to 2.14 kg, respectively. On average, the corresponding values in cold and hot conditions were slightly higher.

ME_{maint} showed a protein-turnover-related within-population CV of 1.5 % at thermoneutrality, as in Chapter 2. Thermoregulatory action contributed about 4 % extra variance in cold and hot conditions, but CV values were not affected. A genetic increase in $P_{dep,max}$ from 100 to 250 $g.d^{-1}$ would increase ME_{maint} as related to protein turnover and thermoregulation by 11 % at thermoneutrality (as in Chapter 2), and by 6–11 % in cold or hot conditions.

Two relevant groups of genotypes could be distinguished based on the within-population regression coefficients of ME_{maint} on daily or cumulative protein deposition ($b_{dailyPdep}$, $b_{cumPdep}$). These ranged from 0.250 to 0.428 $kJ.kg^{-0.75}.d^{-1}$ per $g.d^{-1}$ and from 2.77 to 5.45 $kJ.kg^{-0.75}.d^{-1}$ per kg, respectively, in 12 "conventional" genotypes at thermoneutrality. On average, $b_{dailyPdep}$ was increased by 48, 20, –11 and –36 % in the other climatic conditions mentioned above, respectively. The corresponding increase of $b_{cumPdep}$ was 32, 14, 8 and 48 %. Three fast-growing lean genotypes showed similar $b_{dailyPdep}$ and $b_{cumPdep}$ at thermoneutrality, but much more pronounced increases in cold and hot conditions.

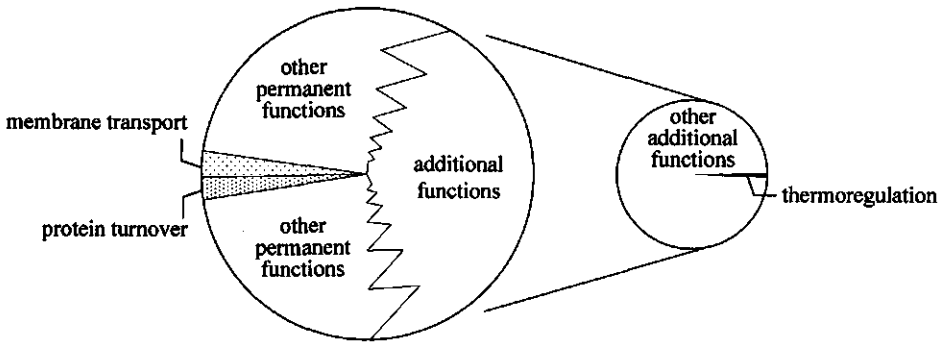
It is concluded that differences in body composition traits between pig genotypes do not cause important between-genotype differences in thermoregulatory ME_{maint}, and that thermoregulatory processes contribute little body-composition-related variation to hot or cold ME_{maint} within most genotypes.

The inferences to be made from this with regard to experimental design are discussed, as in Chapter 2. The verification of the above predictions will require a very elaborate experiment, involving many hundreds of pigs. It seems highly unlikely that such a verification will be carried out.

In **Chapter 7**, the General Discussion, aspects of protein turnover and thermoregulation are discussed that could not be included in Chapters 1 or 3, either because there was insufficient space in the associated journal articles, or because this information was discovered after these

had been published. Likewise, some issues of body protein and lipid partitioning (Chapter 5) and protein and lipid growth curves (Chapter 6) that were not discussed in those chapters, including ideas about the verification of the results obtained in those chapters.
We then describe some quantitative aspects of membrane transport and immunocompetence, two important maintenance functions that have not been explicitly dealt with in this study due to insufficient information, but would be strong candidates for mechanistic modeling.
The statistical problem of measurement errors on the independent variables of a regression analysis, which was encountered but not further elaborated in Chapters 2 and 4, is dealt with in more detail, evaluating alternatives to least squares regression and working through a small example that relates heat production of growing pigs to protein synthesis.
Finally, we discuss the use of growth models, such as the ones evaluated in Chapters 1 and 3, in animal breeding, and make some concluding remarks.

The question that led to the study carried out in this thesis, as quoted in the first paragraph of this Summary, has been partly answered in Chapters 2 and 4. The relevant findings are summarised in Chapter 7, in terms of (i) the magnitude of variation in maintenance requirements and (ii) the way this variation is partitioned. The latter is visualised in the Figure below.



Proposed partitioning of the within-population **variance** of ME_{maint} in growing pigs. The jagged lines indicate uncertainty about the division among these components. See Chapter 7 for details.

The "other permanent functions" comprise service functions such as circulation, coordination, respiration and excretion, plus basal activity. The "additional functions" are triggered by suboptimal environmental conditions, and include not only thermoregulation but also immune reactions, reactions to cope with social stress, and extra physical activity. The overall conclusion is that the maintenance functions in growing pigs that are related to body (growth) composition explain only a limited proportion of the total variance in ME_{maint}.

Samenvatting

Groeiende varkens verschillen in hun energiebehoefte voor lichaamsonderhoud, en een deel van die variatie houdt verband met verschillen in lichaamssamenstelling. Dit komt doordat sommige onderhoudsprocessen (eiwitturnover, actief transport van ionen over de membraan van lichaamscellen, warmtehuishouding en ook sommige afweerreacties) functioneel samenhangen met de samenstelling van het lichaam en/of met de samenstelling van lichaamsgroei. Het onderzoek achter dit proefschrift probeert een deel van het antwoord te geven op een vraag geformuleerd door Webster (1988): "in hoeverre kunnen verschillen in de onderhoudsbehoefte worden toegeschreven aan verschillende proporties van de organen en lichaamsweefsels, elk met hun eigen stofwisselings-intensiteit ?" We zoeken naar dat antwoord met behulp van stochastische simulatie, waarmee we de informatie proberen samen te vatten die in de wetenschap beschikbaar is over de variatie van lichaamssamenstelling bij groeiende varkens, en over de manier waarop eiwitturnover en warmtehuishouding samenhangen met de lichaamssamenstelling.

De stochastische modellen die in hoofdstukken 2 en 4 van dit proefschrift worden gebruikt, gaan uit van een bepaalde dier-eigen variatie in de manier waarop lichaamseiwit en lichaamsvet over eiwit- en vetdepots zijn verdeeld. In **hoofdstuk 5** gaan we na of er inderdaad sprake is van zulke variatie tussen individuele varkens. We gebruiken daarvoor eerder gepubliceerde uitsnijgegevens om de genetische en toom-afhankelijke variatie van zeven kenmerken te berekenen: de totale hoeveelheid eiwit en vet in het lichaam (in het Engels afgekort als TOTPROT en TOTLIPD), het aandeel van het lichaamseiwit dat aanwezig is in de spieren (PROTMUS) of in de huid, het onderhuidse vetweefsel en de botten (bindweefseleiwit, PROTCON), en het aandeel van het lichaamsvet dat aanwezig is in de huid en het onderhuidse vetweefsel (LIPDSUB), in de spieren (inter- en intramusculair, LIPDMUS), of in de botten (LIPDBON). We hebben TOTPROT en TOTLIPD daarbij gecorrigeerd voor verschillen in lichaamsgewicht, PROTMUS en PROTCON voor verschillen in TOTPROT, en LIPDSUB, LIPDMUS en LIPDBON voor verschillen in TOTLIPD. In deze analyse ging het in feite om de herhaalbaarheid van deze kenmerken, die benaderd kon worden via de "erfelijkheidsgraad in ruime zin"; de uiteindelijke schattingen zijn 48 % voor TOTPROT, 56 % voor TOTLIPD, 56 % voor PROTMUS, 57 % voor PROTCON, 32 % voor LIPDMUS, 33 % voor LIPDSUB, en 22 % voor LIPDBON. Deze resultaten leiden tot de conclusie dat er inderdaad sprake is van dier-eigen variatie in de verdeling van lichaamseiwit en lichaamsvet.

De simulaties in de hoofdstukken 3 en 4 werken met een aantal hypothetische varkensrassen, die we omschrijven in termen van de parameters die hun potentiële groei bepalen. In **hoofdstuk 6** proberen we zinnige waarden voor die groeiparameters af te leiden uit eerder gepubliceerde uitsnijgegevens van vijf varkensrassen. We hebben daarvoor de eiwit- en vetgroei van die varkens beschreven met Gompertz-curves, wat leidt tot schattingen van het volwassen eiwit- en vetgewicht (P_∞, L_∞) en van de parameter die de specifieke groeisnelheid van zowel eiwit als vet beschrijft (B_{Gomp}). L_∞ wordt verder uitgedrukt als fractie van P_∞, wat leidt tot de parameter $R_{L\infty/P\infty}$. De maximale eiwitgroei volgt uit $P_{dep,max} = P_\infty \times B_{Gomp} / e$. Het gaat hier om gegevens die zijn gemeten tussen 10 en 133 kg, 13 en 217 kg, 18 en 106 kg, 20 en 110 kg, of tussen 11 en 145 kg levend gewicht. De Gompertz-functie bleek goed bij de gegevens aan te sluiten, voorzover dat kon worden bepaald uit de variatie en de verdelingspatronen van de resttermen, en de niet-significante autocorrelaties.

Gemiddeld over gelten, beren en borgen vonden we schattingen voor P_∞ die geen van alle ver van 31 kg afwijken. De schattingen voor $R_{L\infty/P\infty}$ lopen van 1.4 tot 4.7 $kg.kg^{-1}$, en die voor B_{Gomp} van 0.009 tot 0.017 $kg.d^{-1}.kg^{1}$. Daaruit volgen dan schattingen voor $P_{dep,max}$ van 110 tot 193 $g.d^{-1}$. We hebben deze rassen in 1969, 1976, 1984, 1990 and 1993 geplaatst, en de schattingen uitgezet tegen de tijd. Dit leverde duidelijke patronen op voor alle parameters behalve P_∞. Het lijkt erop dat $R_{L\infty/P\infty}$ geleidelijk aan afneemt tot een plateau-waarde van ongeveer 1, terwijl B_{Gomp} en $P_{dep,max}$ lineair toenemen. Dezelfde trends kwamen naar voren in een analyse van lichaamsgewicht, zoals gemeten aan deze varkens plus aan drie andere rassen uit dezelfde periode.
Verder hebben we deze eiwit- en vetgegevens met een andere sigmoïde functie beschreven, die van de Gompertz-functie verschilt doordat hij een flexibel buigpunt heeft. Dit veranderde niets aan het ontbreken van trend in de tijd voor de parameter P_∞. De schattingen voor het buigpunt van de eiwitgroeicurves van de rassen uit 1969, 1976 en 1984 (uitgedrukt als fractie van P_∞) zijn statistisch niet te onderscheiden van de vaste waarde van 36.8 % van de Gompertz-functie. Daarna gaan ze omhoog, tot aan 46 % voor de populatie uit 1993.
Deze ontwikkelingen moeten het gevolg zijn van een combinatie van veranderingen in voedings- en andere milieu-factoren, en genetische veranderingen. Ze kunnen niet volledig worden verklaard als het resultaat van selectie op mest- en slacht-eigenschappen binnen rassen, want daardoor zou P_∞ moeten zijn toegenomen. Het lijkt erop dat varkensfokkers hun berenlijnen herhaaldelijk hebben opgestart op basis van dieren met een laag volwassen gewicht, en dat volwassen gewicht vervolgens hebben verhoogd d.m.v. selectie binnen die lijnen.

Hoofdstukken 1 t/m 4 beschrijven de eigenlijke simulatiestudies.

In **hoofdstuk 1** hebben we een bestaand simulatiemodel voor groei bij varkens uitgebreid met een module die de eiwitturnover in zes eiwitdepots beschrijft: spiereiwit, bindweefseleiwit, levereiwit, bloedplasma-eiwit, het eiwit in het maagdarmstelsel, en de overige lichaamseiwitten. Deze depots verschillen van elkaar in de snelheid waarmee ze hun eiwit vernieuwen (hun "turnover rates", in het Engels), en in hun groeipatronen in de tijd; gegevens om deze verschillen te kwantificeren kwamen uit de literatuur.
Bij groeiende dieren vertegenwoordigen de turnover rates zoals die experimenteel kunnen worden gemeten twee gelijktijdig verlopende processen: de turnover van bestaand lichaamseiwit en de herhaaldelijke opbouw-en-afbraak van nieuw gevormd (groeiend) eiwit. We proberen onderscheid tussen deze processen te maken door variatie aan te brengen in de fractionele synthese van nieuw gevormd eiwit (FRS_{dep}) en in het aandeel van de onderhoudsbehoefte dat niet met eiwitturnover samenhangt ($FrcME_{maint}$). De resultaten van de simulaties met dat model worden vergeleken met de resultaten van een model waarin eiwitturnover geen expliciete rol speelt.
De turnover rate van bestaand lichaamseiwit (TR_{pres}) bereikte daarbij onzinnige waarden wanneer FRS_{dep} boven 2.5 d^{-1} uitkwam; dat maakt die waarde tot een bovengrens voor FRS_{dep}.
De simulatieresultaten van de beide modellen (met en zonder eiwitturnover als expliciete functie) vertonen vergelijkbare patronen voor bepaalde combinaties van FRS_{dep} en $FrcME_{maint}$, afhankelijk van de waarden van een aantal inputvariabelen. Voor waarden voor FRS_{dep} beneden 2.5 d^{-1} (de zojuist genoemde bovengrens) komen deze patronen het sterkst

overeen bij de combinatie van ME_{maint} = 0.65 en FRS_{dep} = 2.0 d^{-1}. De bijbehorende waarden voor TR_{pres} van de bovengenoemde eiwitdepots zijn 0.060, 0.019, 0.585, 1.492, 0.582 en 0.016 d^{-1}.

Het model dat in hoofdstuk 1 werd uitgebreid met een eiwitturnover-module is in **hoofdstuk 2** stochastisch gemaakt. Het doel hiervan is om groepen varkens te kunnen simuleren, met variatie tussen dieren in de maximale eiwitgroei ($P_{dep,max}$), in de minimale verhouding tussen vet- en eiwitaanzet ($R_{L/P,min}$), en in de verdeling van lichaamseiwit over de depots spiereiwit, bindweefseleiwit en overig eiwit. Dit leidt tot variatie tussen dieren in de energiebehoefte voor eiwitturnover, en dat leidt uiteindelijk tot variatie tussen dieren in de onderhoudsbehoefte als gevolg van variatie in lichaamssamenstelling.
We hebben zeven populaties varkens gesimuleerd, die van elkaar verschillen in termen van hun gemiddelde $P_{dep,max}$; deze parameter varieerde van 100 tot 250 $g.d^{-1}$, met een variatiecoëfficiënt binnen populaties van 10 %. Het voerniveau is ook in zeven stappen gevarieerd. Afhankelijk van $P_{dep,max}$ en voerniveau vonden we standaardafwijkingen (per populatie) van lichaamseiwitgewicht (bij 100 kg levend gewicht) tussen 0.31 en 0.54 kg, en van lichaamsvetgewicht tussen 1.22 en 2.17 kg. De variatiecoëfficiënt van de onderhoudsbehoefte (per populatie) loopt van 1.4 tot 2.0 %. Als we de resultaten over populaties heen vergelijken vinden we een samenhang tussen het deel van de onderhoudsbehoefte dat samenhangt met eiwitturnover en het populatiegemiddelde van $P_{dep,max}$. Een toename van 150 % (van 100 naar 250 $g.d^{-1}$) in laatstgenoemde parameter leidt tot een toename tussen 11 en 15 % (van 470–486 naar 541 $kJ.kg^{-0.75}.d^{-1}$) in de eerstgenoemde.
De resultaten kunnen worden benut ter ondersteuning van het ontwerp van proeven, bijvoorbeeld experimenten om na te gaan of de simulatieresultaten met de werkelijkheid overeenkomen. De simulatie voorspelt bijvoorbeeld een waarde van 21.24 $kJ.kg^{-0.75}.d^{-1}$ per kg voor de regressiecoëfficiënt van de energiebehoefte voor eiwitturnover op de spiereiwitgroei (in het Engels afgekort als $P_{dep,mus}$) tussen 23 en 100 kg lichaamsgewicht. We gebruiken de (co)-varianties van de gesimuleerde kenmerken om af te leiden hoeveel waarnemingen nodig zouden zijn om die waarde experimenteel te bevestigen. We hebben daarbij rekening te houden met de invloed van de meetfouten van de x-variabele ($P_{dep,mus}$) op de berekende regressiecoëfficiënten; zulke "X-errors" veroorzaken een systematische onderschatting van de regressiecoëfficiënt in een normale kleinste kwadraten-analyse. Op basis van de simulatieresultaten kon worden afgeleid dat hun experimentele toetsing praktisch onmogelijk wordt wanneer de variatiecoëfficiënt t.g.v. meetfouten van $P_{dep,mus}$ groter wordt dan 10 %. In dat geval wordt de regressiecoëfficiënt zo sterk onderschat dat de schatting significant lager lijkt uit te komen dan de gesimuleerde waarde. Omdat de meetfout van eiwitaanzet in de praktijk net die orde van grootte heeft, vereisen dergelijke experimentele resultaten een aangepaste statistische techniek; daarop wordt in hoofdstuk 7 teruggekomen.

Het model van hoofdstuk 1 is in **hoofdstuk 3** uitgebreid met een module die de warmtehuishouding simuleert. Om dat te bereiken worden de maximale en de minimale warmte-afgifte van een gegeven varken ingeschat; deze waarden worden vervolgens vergeleken met de warmteproduktie van datzelfde varken, en er wordt thermoregulatoire actie gesimuleerd als de warmteproduktie lager uitvalt dan de minimale warmte-afgifte (dan heeft het dier het koud), of hoger dan de maximale (dan is het dier te warm). De rekenregels van het model die de

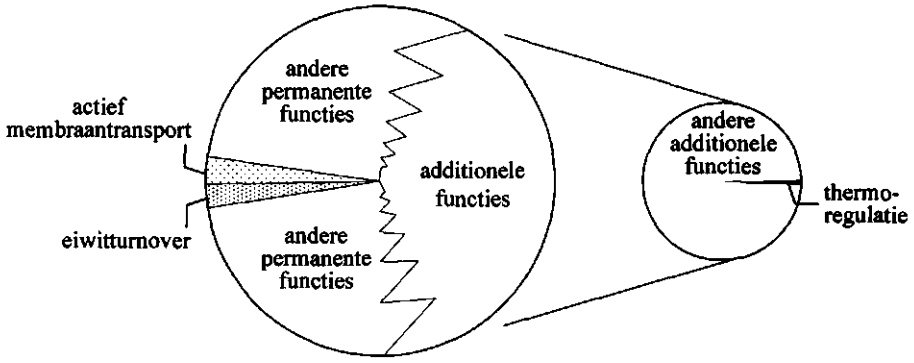
groei bepalen zijn tegelijkertijd aangepast om *ad libitum* voeropname te kunnen simuleren en om het verloop van $P_{dep,max}$ in de tijd variabel te kunnen maken. Dat laatste is gedaan m.b.v. de parameters die in hoofdstuk 6 werden bestudeerd.
De warmte-afgifte is voornamelijk benaderd met een al eerder gepubliceerd model, maar de minimale warmte-afgifte is afhankelijk gemaakt van de isolatiewaarde van het onderhuidse vetweefsel. We hebben gegevens uit de literatuur gebruikt om de vergelijking $y = 0.05 + 0.002 x$ af te leiden, waarbij y staat voor die isolatiewaarde (in $°C.m^2.W^{-1}$) en x voor de dikte van het rugspek (in mm). Diezelfde gegevens wezen uit dat de maximale warmte-afgifte van varkens niet met de spekdikte samenhangt.
Thermoregulatoire actie om koude omstandigheden het hoofd te bieden komt neer op verhoging van de *ad libitum* voeropname. De overeenkomstige actie in warme omstandigheden bestaat uit een verlaging van de activiteit, verhoging van de lichaamstemperatuur, het nat houden van een deel van de huid, en een verlaging van de *ad libitum* voeropname.
Een gevoeligheidsanalyse wees uit dat de gesimuleerde *ad libitum* voeropname, warmteproduktie, eiwitaanzet en vetaanzet sterk afhangen van de eigenschappen van het gesimuleerde genotype, in termen van de groeiparameters P_∞, L_∞ en B_{Gomp}.
We hebben het model gebruikt om eerder gepubliceerde experimenten te simuleren. De onderste en bovenste kritieke temperatuur konden betrouwbaar uit de simulatieresultaten worden afgeleid, hoewel het model deze parameters niet expliciet berekent. Een vergelijking van de overige simulatieresultaten met de proefgegevens wees uit dat de *ad libitum* voeropname betrouwbaar wordt voorspeld. Hetzelfde geldt voor de verdeling van *ad libitum* opgenomen voerenergie over warmteproduktie en eiwit- en vetaanzet in koude, thermoneutrale en warme omstandigheden. Bij een beperkte voeropname, met name in de kou, heeft het model de neiging om de warmteproduktie te overschatten en de vetaanzet te onderschatten, waarschijnlijk doordat het geen rekening houdt met lange-termijn aanpassingen van het dier aan de kou.

Het model dat in hoofdstuk 3 werd uitgebreid met een thermoregulatie-module is in **hoofdstuk 4** stochastisch gemaakt. Het doel hiervan is om groepen varkens te kunnen simuleren, met variatie tussen dieren in de groeiparameters P_∞, L_∞ en B_{Gomp}, in de verdeling van lichaamseiwit over de depots spiereiwit, bindweefseleiwit en overig eiwit, en in de verdeling van lichaamsvet over het onderhuidse depot en overige depots. Dit leidt tot variatie tussen dieren in de energiebehoefte voor eiwitturnover en warmtehuishouding, en dat leidt uiteindelijk weer tot variatie tussen dieren in de onderhoudsbehoefte als gevolg van variatie in lichaamssamenstelling.
We hebben 21 populaties varkens gesimuleerd, die van elkaar verschillen in termen van hun gemiddelde waarde voor de bovengenoemde drie parameters. Elk van deze is in drie stappen gevarieerd, en van de 27 genotypes die zo ontstonden zijn er zes weggelaten omdat hun parametercombinaties in de praktijk niet vóórkomen. Binnen elke populatie werden variatiecoëfficiënten voor de groeiparameters aangelegd van 7, 15 en 3 %. Elke populatie is vijfmaal gesimuleerd, in klimaatsomstandigheden die achtereenvolgens erg koud, tamelijk koud (ca 5 en 1 °C beneden de onderste kritieke temperatuur), thermoneutraal, tamelijk warm en erg warm waren (ca 1 en 5 °C boven de bovenste kritieke temperatuur), steeds over de hele groeiperiode van 23 tot 100 kg levend gewicht. De gesimuleerde voeropname was steeds *ad*

libitum.
Afhankelijk van de groeiparameters vonden we standaardafwijkingen (per populatie) van lichaamseiwitgewicht (bij 100 kg levend gewicht) tussen 0.21 en 0.46 kg, en van lichaamsvetgewicht tussen 0.78 en 2.14 kg. De overeenkomstige variatie in koude en warme omstandigheden is wat groter. Net als in hoofdstuk 2 is de gesimuleerde variatiecoëfficiënt van de onderhoudsbehoefte (per populatie) ongeveer 1.5 %. Deze variatie wordt bijna geheel veroorzaakt door verschillen in eiwitturnover; thermoregulatoire acties leiden tot ongeveer 4 % extra variantie in koude en warme omstandigheden, maar de variatiecoëfficiënten worden er niet door beïnvloed. Als we de resultaten over populaties heen vergelijken vinden we weer een samenhang tussen de onderhoudsbehoefte en het populatiegemiddelde van $P_{dep,max}$. Een toename van 150 % (van 100 naar 250 $g.d^{-1}$) in laatstgenoemde parameter leidt tot een toename van 11 % in de eerstgenoemde, net als in hoofdstuk 2. Dat geldt voor thermoneutrale omstandigheden; in de kou en in de warmte wordt dat 6 tot 11 %.
Op basis van de gesimuleerde regressiecoëfficiënten (per populatie) van de onderhoudsbehoefte op de dagelijkse eiwitaanzet (in het Engels afgekort als $b_{dailyPdep}$) resp. op de cumulatieve eiwitaanzet ($b_{cumPdep}$) konden we twee interessante groepen genotypes onderscheiden. Onder thermoneutrale omstandigheden vonden we bij 12 genotypes waarden voor $b_{dailyPdep}$ tussen 0.250 en 0.428 $kJ.kg^{-0.75}.d^{-1}$ per $g.d^{-1}$ en voor $b_{cumPdep}$ tussen 2.77 en 5.45 $kJ.kg^{-0.75}.d^{-1}$ per kg; deze genotypes zouden kunnen worden omschreven als "conventioneel". In tamelijk koude en erg koude omstandigheden (zie boven) vonden we waarden voor $b_{dailyPdep}$ die 20 resp. 48 % waren verhoogd; in de tamelijk warme en erg warme omstandigheden vonden we waarden die 11 resp. 36 % waren verlaagd. De overeenkomstige resultaten voor $b_{cumPdep}$ waren verhogingen van 14 resp. 32 %, en 8 resp. 48 %. Bij drie snel en mager groeiende genotypes vonden we vergelijkbare waarden voor $b_{dailyPdep}$ en $b_{cumPdep}$ in thermoneutrale omstandigheden, maar veel heftiger reacties in de kou en de warmte.
De belangrijkste conclusies van hoofdstuk 4 zijn (i) dat verschillen in lichaamssamenstelling bij groeiende varkens niet leiden tot belangrijke verschillen tussen rassen in de energiebehoefte voor warmtehuishouding, en (ii) dat de thermoregulatoire functies weinig bijdragen aan variatie in de onderhoudsbehoeften binnen rassen.
Net als in hoofdstuk 2 bespreken we de gevolgen van onze simulatieresultaten voor het ontwerp van experimenten. Voor de bevestiging van de bovengenoemde voorspellingen zullen proeven nodig zijn met vele honderden varkens, en met ingewikkelde procedures om de kritieke temperaturen van tijd tot tijd vast te stellen. Het lijkt niet erg waarschijnlijk dat dat soort proeven in de nabije toekomst zullen kunnen worden uitgevoerd.

Hoofdstuk 7 is het algemene discussie-hoofdstuk van dit proefschrift. We bespreken hier details m.b.t. eiwitturnover en warmtehuishouding die wegens ruimtegebrek in de wetenschappelijke tijdschriften niet in hoofdstukken 1 en 3 konden worden opgenomen, of die pas aan de orde kwamen toen die hoofdstukken al waren gepubliceerd. Hetzelfde geldt voor een aantal details m.b.t. eiwit- en vetverdeling (hoofdstuk 5) en groeicurves (hoofdstuk 6).
Vervolgens gaan we in op de kwantitatieve aspecten van actief membraan-transport en afweerreacties, twee belangrijke onderhoudsprocessen die belangrijke bijdragen zouden kunnen leveren aan de voorspellende kracht van simulatiemodellen maar niet expliciet in dit proefschrift aan de orde zijn gekomen t.g.v. een gebrek aan harde gegevens.

Het statistische probleem van "X-errors" bij regressie-analyse dat we tegenkwamen aan het eind van hoofdstukken 2 en 4, wordt hier in meer detail behandeld. We bespreken alternatieven voor kleinste kwadraten-analyse, en we presenteren een voorbeeld waarin de warmteproductie van groeiende varkens wordt gerelateerd aan hun eiwitsynthese.
Tenslotte bespreken we het nut van dit soort simulatiemodellen voor veefokkerij-toepassingen, en maken we wat algemene concluderende opmerkingen.



Een eerste aanzet voor de verdeling van de **variantie** van de onderhoudsbehoefte bij groeiende varkens. De zigzag-lijnen geven onzekerheid aan m.b.t. de omvang van deze componenten. Details in hoofdstuk 7.

In de eerste alinea van deze samenvatting staat de vraagstelling achter dit promotie-onderzoek verwoord. Deze vraag wordt gedeeltelijk beantwoord in hoofdstukken 2 en 4. De meest relevante resultaten worden in hoofdstuk 7 samengevat, in termen van (i) de omvang van de tussen-dieren-variatie van de onderhoudsbehoefte, en (ii) de manier waarop die variatie is verdeeld over variatie in de onderliggende fysiologische processen. Dat laatste wordt weergegeven in de bovenstaande figuur. De "andere permanente functies" omvatten fysiologische processen zoals bloedsomloop, ademhaling, hersen- en zenuwfuncties, en lever- en nierfuncties, en bovendien een basaal niveau aan activiteit. De "additionele functies" worden ingeschakeld in geval van sub-optimale milieu-omstandigheden; het gaat hier niet alleen om thermoregulatie maar ook om afweerreacties, reacties om om te gaan met sociale stress, en extra activiteit. De uiteindelijke conclusie is dat de onderhoudsprocessen van groeiende varkens die samenhangen met de samenstelling van het lichaam en/of de lichaamsgroei slechts een beperkt deel van de totale variatie van de onderhoudsbehoefte verklaren.

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About the author...

Pieter Willem Knap was born on 16 January 1957 in Wormerveer, somewhat north of Amsterdam in the Netherlands. After classical grammar school and a very short career with Citibank, he enlisted at Wageningen Agricultural University in 1975 to study Animal Science. In 1981 he obtained his MSc *cum laude*, with subjects in Animal Breeding (carried out at the IVO-Schoonoord institute in Zeist), Aquaculture (partly carried out at the University of Utrecht), and Theoretical Production Ecology. In 1979 he spent his trainee period at Suez Canal University in Ismailia, Egypt, culturing grass carp for ILACO-Euroconsult.

Just after returning from Egypt he met Ella Luiting, with whom he still lives happily together.

From 1981 to 1993 he was responsible for research and development with the Dutch Pig Herdbook Societies (now known as Stamboek Pigs), which included the implementation of a controlled four-way crossbreeding program, the initiation of specialised sire and dam line breeding, a re-design of performance testing methods, and the setup of a relational-database-*cum*-BLUP-evaluation computer system.
In 1993 and 1994 he was technical projects manager with the UK branch of PIC (the Pig Improvement Company), focusing on the formalisation of European breeding objectives and on performance testing technology.
From 1994 to 1997 he was technical director of Norsvin International, the business branch of the Norwegian Pig Breeders' Association. This involved the initiation and further support of daughter nucleus breeding programs and their business in Minnesota (USA), Slovenia, Thailand and Queensland (Australia).
Since 1997 he works as quantitative genetics research manager for PIC International Group, based at the Roslin Institute in Edinburgh (UK). He is responsible for the development and implementation of technology to support the design of pig breeding programs and performance testing systems.

The study reported in this thesis was carried out as a spare-time activity between 1989 and 2000, making use of idle computer capacity at the above mentioned companies.