PREDICTION OF PRODUCTION

Nutrition induced tissue partitioning in growing pigs

K.H. de Greef

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Proefschrift
ter verkrijging van de graad van
doctor in de landbouw- en milieuwetenschappen,
op gezag van de rector magnificus,
dr H.C. van der Plas,
in het openbaar te verdedigen
op woensdag 16 september 1992
des namiddags te vier uur in de aula
van de Landbouwuniversiteit te Wageningen.

De vrees des HEEREN is het beginsel der wetenschap (*Spr. 1:7*)

STELLINGEN

I

Het concept van konstante samenstelling van de aanzet van lichaamsweefsel bij vleesvarkens bij niet-maximale eiwitaanzet is een oversimplificatie.

DIT PROEFSCHRIFT

II

De lineair-plateau relatie tussen eiwitaanzet en energieopname impliceert een lineaire relatie tussen vetaanzet en energieopname. Dit betekent echter niet dat deze vetaanzet in vaste verhouding staat tot de eiwitaanzet.

DIT PROEFSCHRIFT

III

Vetaanzet bij vleesvarkens verdringt geen lichaamswater.

DIT PROEFSCHRIFT

IV

Fysiologische karakterisering van de produktiekapaciteit van vleesvarkens is van cruciaal belang voor een doelmatig gebruik van groeisimulatiemodellen.

DIT PROEFSCHRIFT

V

Gebrek aan kennis van de gekombineerde effekten van huisvesting en gezondheid op vleesvarkens bemoeilijkt het gebruik van onderzoeksresultaten voor de praktijk.

VI

In toekomstig wetenschappelijk onderzoek omtrent voeding van vleesvarkens zal meer aandacht moeten worden besteed aan zowel de principes als aan de effekten van voederstrategieën.

VII

De methodieken voor chemische analyse van lichaamsweefsels verdienen meer aandacht.

VIII

Simulatiemodellen zijn niet geschikt om kennis te genereren.

IX

Ondanks nuanceringen en waarschuwingen in de wetenschappelijke literatuur wordt de waarde van de N-balans als maat voor de eiwitaanzet in veel gevallen overschat.

X

Het feit dat de evolutietheorie even zo goed op 'geloof' gebaseerd is als het creationisme duidt aan dat de wetenschap en het onderwijs in deze forse oogkleppen op hebben.

XI

Voor mensen die termen als *Deo Volente, D.V.* en termen van gelijke strekking niet van waarde achten bij aankondigingen, is het de moeite waard de bijbelboeken Lukas (hoofdstuk 12, vers 13 - 21) en Jakobus (hoofdstuk 4, vers 13 - 17) hier eens op na te slaan.

XII

Het voortdurend gebruik van waterstraalpompen in laboratoria getuigt niet van een verantwoorde houding op milieugebied.

XIII

Het via de Europese Gemeenschap pogen te financieren van onderzoek begint eigenschappen van kapitaalvernietiging te krijgen.

XIV

Het corrigeren met behulp van lineaire covariabelen getuigt meestal van een (te) rechtlijnig denken.

XV

Het verschil tussen een onderzoeksgroep en een groep onderzoekers heeft niets met de lokatie te maken.

K.H. de Greef
PREDICTION OF PRODUCTION. Nutrition induced tissue partitioning in growing pigs

Wageningen, 16 september 1992

Voorwoord

Het in dit proefschrift beschreven onderzoek is uitgevoerd bij de vakgroep Veevoeding van de Landbouwuniversiteit te Wageningen als een gezamenlijk onderzoeksprojekt van Mengvoeder UT-Delfia bv, Varkens onderzoek centrum Nieuw-Dalland bv en de Landbouwuniversiteit.

Prof. dr ir M.W.A. Verstegen en dr ir B. Kemp ben ik niet alleen zeer erkentelijk voor de stimulerende begeleiding, maar ook voor de geboden vrijheid. Ook dr ir L.A. den Hartog had een wezenlijke bijdrage. Hij tilde het projekt van de grond, en begeleidde het tot aan zijn vertrek naar het Proefstation voor de Varkenshouderij. Begeleid worden door Martin, Leo en Bas is een belevenis. Een zeer wezenlijke bijdrage is geleverd door ir E.F. Knol en ir J.A.H.M. Smulders. Vanuit respectievelijk Dalland en UTD waren zij inhoudelijk nauw betrokken, en hebben daarbij een forse vinger in de pap gehad in de ontwikkeling van ondergetekende. De driewegkruising Fokkerijorganisatie - Mengvoederbedrijf - Onderzoeksinstelling laat zien dat heterosis een groot iets is. Egbert en Jan, bedankt!

Tijdens de experimenten was Peter van der Togt de steun en toeverlaat. Hij heeft zich hierbij ontwikkeld tot dè specialist in het 'tot moes' vermalen van varkens. Ook Tamme Zandstra stond altijd klaar om te helpen, waar dan ook gebruik van gemaakt werd. Anderen die tijdens de experimenten onmisbaar waren, zijn de medewerkers van proefaccomodatie 'De Haar', waarbij met name André Jansen en Ries Verkerk veel werk op een perfecte manier hebben verzet. Een speciaal woordje van dank wil ik richten tot dhr Van Ginkel en zijn medewerkers van Slachthuis Veenendaal. Dankzij hun enthousiasme en flexibiliteit was er meer mogelijk dan men redelijkerwijs op een slachthuis kon verwachten. De mensen van de afdeling T.A.S.K. van IVO-DLO in Zeist hebben zorggedragen voor het uitvoeren van de IVO-standaardmethode. Alleen op deze manier was het mogelijk een karkas om te zetten in voor ons meer waardevolle delen. Zonder de mensen van het laboratorium van Veevoeding waren de varkens nooit omgezet in zoveel cijfertjes. Hierbij moet vooral de expertise van Marianne van 't End met betrekking tot het verwerken van de 'vleesjes' genoemd worden.

The contacts with dr P.J. Moughan (New Zealand) and dr R.G. Campbell (Australia) have been of great value to the present thesis. Especially the generosity of Paul Moughan and his university for providing the opportunity to use the 1984 version of the Massey Pig growth model is greatly appreciated.

Vanzelfsprekend zijn er velen die wel een bijdrage geleverd hebben, maar niet met name genoemd zijn. Enkelen hiervan zijn de overige kollegas en medewerkers van Veevoeding, Dalland en UTD, studenten en stagiaires. Uiteraard gaat mijn dank ook naar hen uit. Als laatste zou ik willen vermelden dat de persoonlijke kontakten met bovengenoemde personen het geheel extra plezierig gemaakt heeft.

Wageningen, juni 1992

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Greef, K.H. de. 1992. **PREDICTION OF PRODUCTION. Nutrition induced tissue partitioning in growing pigs.** In several swine growth models, a constant ratio is assumed between lipid deposition rate and protein deposition rate when pigs are fed below their protein deposition capacity. In the present thesis, it was studied whether there is an effect of energy intake, live weight and nutritional history on composition of growth in restrictedly fed pigs. Results showed that all three mentioned factors affected the composition of growth. Fatness of deposited tissue increased both with an increase in energy intake and with live weight. It was concluded that the composition of total tissue is not constant. It was derived that the composition of extra tissue, deposited due to extra energy intake is independent of energy intake. This extra tissue is consistently fatter than total deposited tissue. This explains the increase in fatness of pigs with each increase in energy intake. It was studied which factor (energy intake or body weight) is of major importance for the partitioning of tissue. Composition of growth was affected by energy intake to a considerably larger extent than by live weight. Furthermore, data on the partitioning of deposited protein and lipid within the body showed that, even at low energy intakes, the major part of extra tissue (due to extra energy intake) was deposited in non-lean carcass parts. **PhD Thesis, Department of Animal Nutrition, Wageningen Agricultural University, Haagsteeg 4, 6708 PM Wageningen, The Netherlands.**

Contents

GENERAL INTRODUCTION

GENERAL INTRODUCTION

In general, pigs with a high feed intake have fatter bodies as compared to pigs with lower feed intakes. The optimal production strategy can be described as producing pigs with a high meat content with acceptable growth rate and acceptable feed consumption. This is equivalent with optimization between fat deposition and lean deposition. Much research effort has been made to find the optimum amount of feed for pigs. However, this optimum depends on more factors than just feed intake. It also is influenced by gender, genotype, weight range, feed composition, housing conditions, economy etc. Furthermore, several interactions between these factors exist. The multifactorial aspect makes description of effects difficult and endless. Combining knowledge of mechanisms into a simulation model will help in ordering information. Parts of the growth process can be described separately. These processes can again be described in subprocesses or, when information of the driving factors is unknown, they can be described empirically. The complete set of aspects (or processes) can be combined into an algorithm. This is the simulation model, and will allow integral calculation with all incorporated aspects. In literature, several growth models for pigs have been described. They vary from very empirical up to a high level of integration of biochemical processes. Structure and level of aggregation highly depend on available information and on the purpose for which the model is developed (see Moughan and Verstegen, 1988).

The Linear-Plateau concept

For growth prediction, especially description of the response in tissue deposition to nutrient intake is important. An important step forward in this respect is the concept as proposed by Whittemore and Fawcett (1976). They described the response of pigs to increasing energy intake in terms of protein deposition and lipid deposition. From the experimental observation that, within certain limits, protein deposition is always accompanied by lipid deposition and that at high levels of energy intake lipid deposition is vastly increasing, they described a mechanism. It was suggested that protein deposition increases linearly with increasing energy intake. This relation is limited, pigs are thought to have an intrinsic maximum of protein deposition capacity (further referenced to as *PDmax*). Furthermore, it is assumed that there is a minimal amount of lipid deposition (LD) accompanying each unit of protein deposition (PD). Thus, there is a minimal ratio between LD and PD. Below the intrinsic maximum for protein deposition, production energy is partitioned between PD and LD according to this minimal ratio (r). Above the protein deposition capacity, all remaining energy is used for lipid deposition. This concept will bereferred to as the Linear-Plateau concept. Thus, the Linear-Plateau concept deals both with the linear increase in protein deposition with increasing energy intake up to the plateau and with the minimal ratio between lipid and protein deposition. The mentioned maximal protein deposition rate (*PDmax*) and minimal ratio between lipid and protein deposition (r) are assumed to be constant for pigs during the growing-fattening range (Whittemore, 1983). In figure 1, the general principle of the Linear-Plateau concept is illustrated.

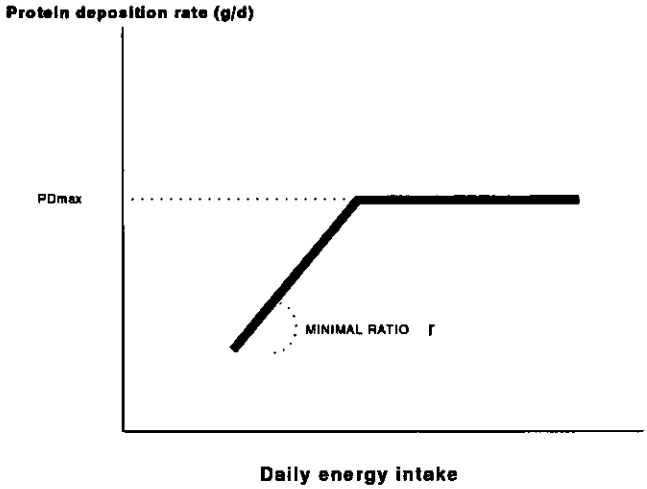

Figure 1. The relation between energy intake and protein deposition rate as proposed by Whittemore and Fawcett (1976).

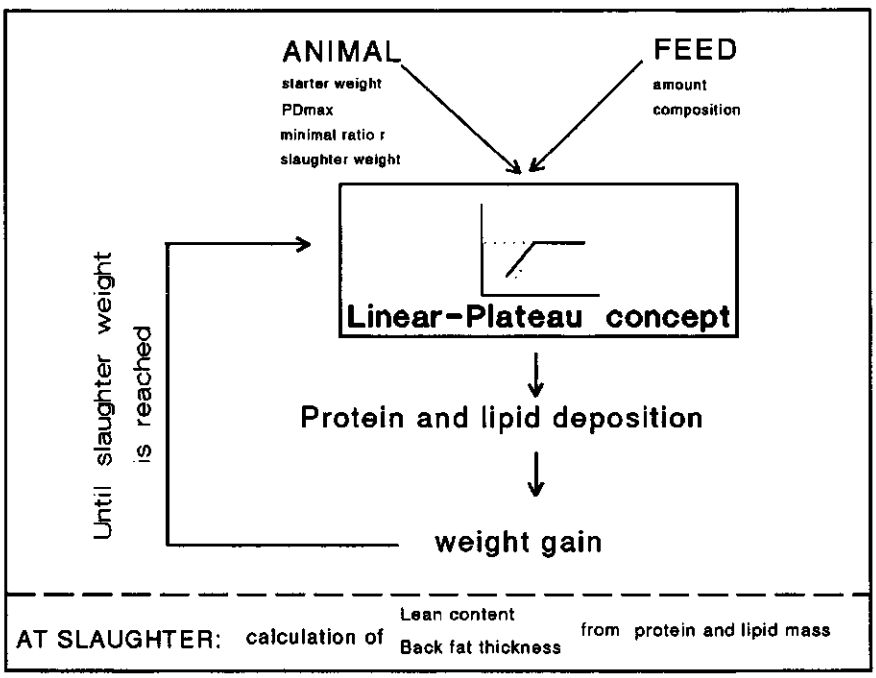

Figure 2. Simplified example of the general structure of a growth model (adapted from Moughan et al., 1987).

The concept of nutrient partitioning as proposed by Whittemore and Fawcett is incorporated in several growth models (Whittemore, 1976; Moughan et al., 1987; Watt et al., 1987; Pomar et al., 1991; TMV, 1991). Figure 2 presents the general structure of a typical growth model incorporating the Linear-Plateau concept. Input for the model are aspects of nutrition and aspects of the pig. Aspects of the nutrition are feed intake and feed composition (represented by amino acids and energy). Aspects of the pig are starter weight, final weight, and characterization parameters (describing how the pig responds to nutrition: *PDmax* and *r*). The model calculates on a daily basis. For one day, intake of nutrients is calculated from feed intake and feed composition. Combining this with the characterization of the pigs (protein deposition capacity (*PDmax*) and minimal ratio of lipid to protein deposition (*r*)) allows calculation of protein and lipid deposition. From the protein deposition rate, water and ash deposition can be calculated. Summation of the four body components (protein, lipid, water and ash deposition) results in empty body gain. Assuming a certain amount of gut fill allows calculation of live weight gain. The model adds up these deposition rates to the masses of body components already present, and calculation can be performed for the next day. This process is repeated until the final weight is reached. At that stage, physical parameters like lean content and back fat thickness can be derived from the chemical components like protein and lipid mass. Structure of growth models incorporating the linear plateau concept have been described in more detail by Whittemore (1976 and 1983) and Moughan et al. (1987).

Application of the Linear-Plateau concept

The Linear-Plateau concept[1] is very attractive in explaining and demonstrating nutritional principles. This is illustrated by figure 1. It clearly shows that there is an upper limit in relevant response (protein deposition), and that nutrition is a major tool in optimizing production (Whittemore, 1983 and 1987). It also illustrates that the capacity of pigs (*PDmax*) influences the optimal feed allowance to a substantial degree (Whittemore, 1987). Explaining and demonstrating nutritional principles requires a qualitative approval of the concept. For education and extension purposes, it is not highly important that the concepts describes the relation between tissue deposition and performance very accurately in quantitative terms. However, when the concept is integrated in growth models, thus quantifying the relation between intake and tissue deposition, it has to be evaluated whether the concept describes a true mechanism.

> An example may illustrate consequences of qualitative and quantitative use of the concept. The concept implicates that there is a discrete optimal feeding level. This is the feed intake at which the protein deposition rate just equals *PDmax*. At this point, protein deposition is maximized with a minimal lipid deposition rate. In qualitative terms, this demonstrates that pigs have some maximal capacity, and have to be fed accordingly. In a quantitative approach, when pigs are characterized in terms of a protein deposition capacity (*PDmax*) and a minimal ratio of lipid to protein deposition (*r*), the optimal feed allowance can be calculated. Animals have to be fed an amount of energy sufficient to (*1*) allow for their maintenance expenditure, (*2*) allow for the *PDmax* and (*3*) allow for the minimal lipid deposition. Due to the assumed constancy of *PDmax* and *r*, with

[1] Mentioning the Linear-Plateau concept references to both the linear-plateau relation between energy intake and protein deposition and to the additional assumption of a constant ratio between lipid deposition and protein deposition (i.e. the integrated use of these two assumptions in growth models)

increasing live weight, the amount of feed only has to be increased for the increase in maintenance expenditure. In fattening pigs, this means an increase of only 7.6 MJ ME /day (about 0.6 kg of feed per day) between 25 and 100 kg live weight. Assuming a constant daily gain of 800 g/d, a weekly increase of only 45 g/d would supply enough energy to meet the increase in maintenance requirement. However, when the minimal ratio of lipid to protein deposition is not constant over the live weight range, calculations will be erroneous. When r increases with live weight, the calculated optimal feed allowance as calculated using the Linear-Plateau concept will result in a too low feed allowance at higher body weights, and protein deposition rate will be below maximum. This example demonstrates that when a qualitative approach (which succeeds well in demonstrating a principle) is extended to a quantitative approach for the whole growing period, and applied in terms of optimal feed allowance, one can come to a conclusion which will not be supported by experimental or practical observations.

Thus, if the concept is to be used for quantitative purposes, it has to be validated. This is relevant as the concept is used widely in the world in swine growth models (Whittemore, 1980; Moughan et al., 1987; Watt et al., 1987; Pomar et al., 1991; TMV, 1991), most of which are used for optimizing feeding strategies in practice. A good way to study the concept is validating a growth model in which the concept is incorporated. There are several specific demands in validating a growth model. A validation requires a specific experimental setup, one which takes the aspects of the concept into account. Besides this, a model also takes other assumptions into account and therefore, a discrepancy between observed and predicted is not readily attributable to certain assumptions. Furthermore, the response to nutrition is calculated in terms of changes of chemical body composition. Thus, a data set at least has to consist of several nutritional treatments and has to quantify effects on chemical body composition. Because of these specific demands, it is quite difficult to use literature data for validation. In literature, no data sets are available which meet all of these criteria. For this reason, in the present study, a series of experiments was setup and conducted taking into consideration that particular parameters related to the Linear-Plateau concept and other assumptions in models could be evaluated.

In the following chapters, a series of studies will evaluate and discuss the tissue partitioning in growing pigs. As nutrition is regarded the main factor influencing tissue partitioning, this receives a major part of the emphasis. Other assumptions made in models (like amount of gut fill, energetic efficiency of synthesis of protein and lipid, maintenance energy requirement etc.) will not be discussed in the present thesis. Material to be used originates from literature (chapter II) and from experiments which were designed for this purpose (chapters I, III, IV and V). The experiments presented in chapters I, III, IV and V have been performed on entire male pigs of synthetic strains of high genetic merit, or on a cross between such strains. Reason for this is the assumption that these pigs represent pigs of the forthcoming years and the fact that any relevant level of performance can be studied in this type of pigs. Variation between animals is important both in breeding and in practice. In the present studies, however, relatively homogenous batches of pigs were used to reduce between animal variation. Effects of between animal variation on performance parameters have not been subjected to study.

Chapter I describes a validation of the Massey Pig Model. This model is based on the described concept of nutrient partitioning as proposed by Whittemore and Fawcett (1976).

Validation allows analysis of several assumptions in the model. So far, most validations of swine growth models have not taken effects on chemical body composition or chemical composition of gain into account. As model calculations are based on chemical tissue deposition, discrepancies between observed and predicted chemical tissue deposition will help in studying which assumptions in the model are weakest. Therefore, the validation emphasised on effects of nutrition on chemical body composition.

The Linear-Plateau concept has two major assumptions. First is the linear-plateau relation between energy intake and protein deposition. Second major assumption in the Linear-Plateau concept is the constant minimal ratio between lipid deposition and protein deposition below maximal protein deposition rate. The linear-plateau relation between protein deposition and energy intake is supported by a series of experiments performed in Australia (Campbell et al., 1983 and 1985; Campbell and Taverner, 1988). In chapter II, the publications reporting these experiments are studied whether the second assumption (constant r) is also supported by these experiments. Thus, this chapter presents a further validation of the Linear-Plateau concept.

Aspects of partitioning of production energy into protein and lipid deposition are further studied in chapter III. In this chapter, the effects of live weight and energy intake on the ratio between lipid and protein deposition are studied. Special effort is made to estimate the effect of energy intake and of body weight independently.

A major aspect of models, and of present studies, is the response of animals to nutritional input. In this respect, especially the response of pigs to energy intake is studied in the present thesis. Therefore, the diets used in the studies presented in this thesis were formulated to be sufficient in nutrients other than energy. Effects of limited amino acid supply are studied only in chapter IV. Limited protein supply is a method to create fat pigs at low levels of live weight gain. By means of a severe protein restriction, a group of pigs was created which were considerably older and fatter at 65 kg live weight as compared to control animals. Beyond 65 kg live weight, both groups were fed adequate diets. Between 65 and 105 kg live weight, it was studied whether partitioning of production energy into tissue deposition (i.e. protein and lipid, lean tissue and fatty tissue) is affected by the previous nutritional treatment.

The Linear-Plateau concept deals with whole body chemical tissue deposition. Relevant in pig production is lean production. Chapter V presents a study on the partitioning of deposited tissue between tissue groups (lean, other carcass parts and organs) in the body. Nutritional and body weight effects on this partitioning help in evaluating to what extent an increase in body chemical tissue deposition will result in an increase in lean deposition. From data of the same experiment as presented in chapter III, it is studied into which tissue groups new tissue is deposited. Especially the contrast between two nutritional treatments is studied in order to derive into which tissue groups additional units of tissue are deposited.

In the general discussion, the evaluation of the Linear-Plateau Concept (chapters I - IV) will be discussed. Conclusions from the studies will be compared to other concepts in literature on tissue deposition in pigs. An attempt will be made to propose an improved

characterization of pigs. The general discussion will also further study partitioning of deposited tissue into tissue groups. Finally, relations between the Linear-Plateau concept and tissue partitioning will be discussed concisely.

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Chapter I

VALIDATION OF A PORCINE GROWTH MODEL WITH EMPHASIS ON CHEMICAL BODY COMPOSITION

K.H. DE GREEF, M.W.A. VERSTEGEN AND B. KEMP

SUMMARY

A growth model for pigs was validated. The model developed at the Massey University (New Zealand) was chosen because its assumptions and structure have been published completely. Besides this, its structure is representative for most other swine growth models. In an experiment, two strains of pigs received per strain: a protein sufficient ration at either (i) 33 MJ DE/day on average or (ii) 27 MJ DE/day on average or (iii) a protein deficient (50% of ARC recommendation) ration fed *ad libitum*. Rations were composed by mixing two basal feeds, containing 25% and 7% crude protein respectively. At 28, 65 and 105 kg live weight, 4 pigs in every strainxtreatment combination were slaughtered. Observed average daily gain, feed conversion ratio, chemical body composition at slaughter and deposition rates of protein and lipid were compared to values calculated by the model. In general, the model predicted the effects of the nutritional treatments adequately. Especially protein deposition rate and live weight gain were predicted quite accurately. However, above 65 kg live weight, the model underestimated lipid deposition rate in the protein sufficiently fed pigs by 20-30%. An increase in the ratio between lipid and protein deposition rate at the higher live weights was found. The model assumed no effect of live weight on this ratio. In contrast to the protein sufficient rations, in the protein deficient treatment, lipid deposition was overestimated considerably while protein deposition rate was predicted adequately. Results also indicated that the amount of water deposited for each unit of deposited protein is higher than predicted by the model. Main conclusion from the validation is that the parameter which accounts for the minimal ratio of lipid to protein is dependent on live weight.

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VALIDATION OF A PORCINE GROWTH MODEL WITH EMPHASIS ON CHEMICAL BODY COMPOSITION

INTRODUCTION

Several models which predict performance of growing pigs as a function of nutritional input and animal characteristics have been described in literature (Whittemore and Fawcett, 1974 and 1976; Whittemore, 1983; Moughan and Smith, 1984; Moughan et al., 1987; Black et al., 1986).

Mentioned models combine knowledge of growth response to input of nutrients in order to predict effects of nutritional manipulation. Predicted parameters are primarily rate of gain and body composition. Other parameters like backfat thickness and carcass grading can be predicted as well. Part of swine growth models consists of mechanistic predictions. Where information is lacking, empirical relations are used. Because of lack of full knowledge about mechanisms that determine growth, the combination of all assumptions (the model) has to be evaluated. This validation, comparing observed to predicted performance, should be performed from a source independent from those used for building the model.

The mentioned models predict performance by calculating the response of the animal to nutritional input in terms of daily protein and lipid deposition. Parameters like water deposition and live weight growth are derived from the predictions of lipid and protein deposition. Therefore, validation has to take chemical body composition into account.

An experiment was performed with two different types of animals and three feeding strategies. The experiment was designed with two aims. The first aim was to obtain a data set to validate a growth model, the Massey Model as described by Moughan et al. (1987). The responses in protein and lipid deposition to different nutritional inputs as calculated were compared to the experimental results. The second aim was obtain predictions of anatomical parameters like backfat thickness and lean content. These anatomical data are needed to extend the model to slaughter quality parameters. In the present paper, the validation of model will be described.

MATERIAL AND METHODS

Description of the model

The Massey Model (Moughan et al., 1987) is studied in this respect. Assumptions and structure of this model have been published completely (Moughan and Smith, 1984; Moughan et al., 1987). Prediction of growth in the validated model is based on the concept of nutrient partitioning as described by Whittemore and Fawcett (1976) and Whittemore (1976 and 1983). In this concept, the animal responds to an increase of a limiting nutrient in a linear way, up to a certain limit. At this point, another factor (e.g. another nutrient or the capacity of the animal) becomes limiting. Protein deposition responds linearly to an increase in energy intake. There is a maximum in protein deposition capacity. Therefore,

with increasing energy intake, protein deposition will reach a plateau. This linear-plateau mechanism has been proposed by Whittemore and Fawcett (1974), and has been shown in parts of the growing period by Campbell et al. (1983 and 1985) and by Campbell and Taverner (1988). At energy intakes where protein deposition is below the plateau, there is a certain minimal amount of fat deposited together with each unit of deposited protein (Whittemore and Fawcett, 1976). This amount (grams) of lipid per gram of deposited protein will be referenced as the minimal ratio of lipid to protein (r). Above the energy intake supporting maximal protein deposition, energy which is not used for the protein and minimal lipid deposition is all deposited as lipid. In short, above maintenance, animals give priority to protein deposition with a certain minimal amount of fat (r). Above protein deposition capacity (*PDmax*) all remaining energy is directed towards lipid deposition. In the validated version of the Massey Model, r was set to 1 (Moughan et al., 1987). This means that at least one gram of lipid deposition for each gram of protein deposition is assumed. The animal is characterized by its protein deposition capacity (*PDmax*).

Experiment

The experiment was performed in a 2×3 factorial design; entire males of two strains and three feeding strategies. The two strains were a boar line (S1) and a commercial (S2) line. Three nutritional treatments were imposed. One treatment was a protein sufficient (ARC, 1981) ration, at a level which was expected to be nearly *ad libitum* (treatment HIGH). The second was a ration comparable to that of treatment HIGH, but restricted by about 20% in amount of feed as compared to treatment HIGH (treatment RESTR). The third treatment was a protein restriction, 50% of ARC requirement, offered *ad libitum* (treatment PROTDEF).

The rations were composed on daily basis by mixing two maize based, high density basal feeds, a high protein feed (25% CP and 1.48% lysin) and a low protein feed (7% CP and 0.36% lysin). The amino acid profile in the rations was designed to have lysin be the first limiting amino acid and no excess imbalances of other amino acids. The rations were adjusted weekly. Table 1 shows for each week feed intake (standardized to 15 MJ DE/kg ration), percentage of lysin and methionine&cystine in the ration and number of animals still in the experiment.

Slaughter weights were 28 kg live weight (4 animals per strain), 65 kg LW (4 animals of each treatment×strain combination) and 105 kg LW (5 animals of each treatment×strain combination). The animals were housed individually. Basal feeds, animals, housing, slaughter procedure and method of body chemical analysis have been described in more detail elsewhere (De Greef et al., 1992).

Validation

Feed composition data and weekly feed intake figures for each individual animal were used as input to the model. Validation results (average daily gain, feed conversion ratio, protein deposition, lipid deposition, ratio of lipid to protein deposition, %protein in the empty body, %lipid in the empty body) will be presented as means for treatment groups, although validation is performed on individual pigs. The animals of both strains in the model were defined as having a protein deposition capacity of 200 grams per day. This protein deposition capacity was measured on the same strains of animals in another part of the

experiment (De Greef et al., 1992). Predicted deposition rates of protein and lipid were compared with observed data for two weight ranges: 28-65 kg live weight and 65 to 105 kg live weight.

Table 1. Average live weight (LW, kg), feed intake (FI, g/d), lysin content (%lys) and content of methionine and cystine (%m&c) in the ration and number of pigs still in the experiment (#) for each week of the experiment[1].

	HIGH					RESTR					PROTDEF				
week	LW	FI	%lys	%m&c	#	LW	FI	%lys	%m&c	#	LW	FI	%lys	%m&c	#
1	32	1464	1.41	0.90	18	31	1373	1.38	0.89	17	29	1279	0.75	0.51	18
2	39	1725	1.23	0.80	18	37	1510	1.20	0.78	17	34	1547	0.63	0.44	18
3	47	2033	1.16	0.75	18	43	1626	1.11	0.72	17	39	1790	0.62	0.43	18
4	54	2172	1.13	0.73	18	49	1654	1.08	0.70	17	44	1851	0.56	0.40	18
5	61	2286	1.11	0.72	17	55	1696	1.03	0.68	17	48	1919	0.52	0.37	18
6	69	2368	1.07	0.70	10	61	1739	1.00	0.66	17	52	1929	0.50	0.36	18
7	77	2417	1.04	0.68	10	67	1803	0.98	0.65	12	57	1983	0.49	0.35	18
8	85	2485	1.02	0.67	10	74	1877	0.96	0.63	9	60	1922	0.47	0.34	17
9	93	2548	1.00	0.66	10	80	1952	0.93	0.62	9	64	1801	0.47	0.34	14
10	100	2656	0.98	0.65	8	88	2032	0.91	0.61	9	67	1904	0.46	0.34	11
11	103	2669	0.98	0.64	2	95	2107	0.89	0.59	9	71	1843	0.46	0.33	11
12						98	1939	0.89	0.59	6	74	1763	0.46	0.33	11
13						101	2142	0.88	0.59	4	78	1890	0.46	0.33	10
14						103	2179	0.88	0.58	1	82	1855	0.46	0.33	10
15											86	1771	0.46	0.33	10
16											87	1780	0.46	0.33	9
17+18											88	1701	0.46	0.33	7
19+20											87	1426	0.46	0.33	5
21+22											88	1370	0.45	0.33	4
23+24											93	1444	0.45	0.33	4
25+26											96	1545	0.45	0.33	3
27+28											100	1724	0.46	0.33	3
29+30											101	1493	0.46	0.33	1

[1] One animal (treatment RESTR, strain S2, slaughter weight 105 kg) was excluded in the calculations due to serious illness during the experiment.

Statistics

Observed values were analyzed for the effect of Treatment and Strain of pig according to the model (SAS, 1990)

$$Y_{ijkl} = \mu + T_i + S_j + W_k + T{\times}S_{ij} + T{\times}W_{ik} + e_{ijkl}$$

in which:

- Y: dependent parameter
- μ: overall mean
- T: effect of nutritional Treatment i (i = HIGH, RESTR, PROTDEF)
- S: effect of Strain j (j = S1, S2)
- W: effect of Weight range k (k = 28-65kg, 65-105kg)
- e: residual error.

Values predicted by the model were compared to the values observed in the experiment by testing whether their difference was different from 0 using Student's t-test. Differences between predicted and observed values are presented as percentage deviation between these values.

RESULTS

In table 2, the effects of the three treatments on the observed parameters are tested. Dietary treatment had a significant influence on all parameters tested. Weight range also influenced all parameters, except for average daily gain and rate of protein deposition. Dietary treatments resulted in groups which differed distinctly with regard to performance. Effects of strain were mainly on body composition data. No effects of strain were detected on observed *in vivo* parameters like average daily gain and feed conversion ratio.

Table 2. Effects of the various experimental factors on performance and body composition data and residual standard deviation (rsd).

	Treatment	Strain	Weight range	T×S	T×W	rsd
FI (g)	***		***		t	147
ADG (g)	***				t	83
FCR	***		***		*	0.19
PD (g)	***			*		13
LD (g)	***	t	**			40
LD/PD	***	t	**			0.35
%PROTEIN	***	t	**	*	***	0.52
%LIPID	***	*	***		**	2.1

***: P<0.001 **: P<0.01 *: P<0.05 t: P<0.10

Treatment HIGH

In table 3, average values of observed and predicted average daily gain (ADG) and feed conversion ratio (FCR) are presented. Treatment HIGH resulted in an average daily gain of 1072 g/d in weight range I, and 1139 g/d in weight range II. Observed feed conversion ratio was 1.83 in weight range I and 2.20 in weight range II. The strains S1 and S2 showed similar ADG and FCR in both weight ranges. Predictions of ADG and FCR by the model were not significantly different from observed values in both weight ranges (table 3). For each observation (each animal per week), predicted live weight is plotted versus observed live weight in figure 1. Predicted live weight (LW) fitted observed values of LW well.

Table 3. Observed and predicted daily gain (ADG, g/day) and feed conversion ratio (FCR) for each weight range, and %difference between predicted and observed values.

		HIGH S1	Δ	HIGH S2	Δ	RESTR S1	Δ	RESTR S2	Δ	PROTDEF S1	Δ	PROTDEF S2	Δ
25-65kg WEIGHT RANGE I													
ADG	obs	1096		1049		857		858		617		641	
	pred	1083	-1	1093	+4	812	-5*	803	-6	630	+2	679	+7^t
FCR	obs	1.79		1.87		1.87		1.86		2.81		2.89	
	pred	1.80	+1	1.77	-5	1.99	+6*	1.99	+8	2.74	-2	2.71	-6^t
65-105kg WEIGHT RANGE II													
ADG	obs	1123		1155		927		929		445		569	
	pred	1131	+1	1123	-3	794	-14**	741	-20*	391	-13**	539	-6^t
FCR	obs	2.23		2.17		2.17		2.12		3.87		3.69	
	pred	2.22	-1	2.23	+3	2.57	+19**	2.74	+30**	4.50	+16*	3.96	+7

Δ: %difference between predicted and observed; **: P<0.01 *: P<0.05 t: P<0.10

Table 4. Observed and predicted deposition rates of protein and lipid (PD, LD, g/d) and difference between predicted and observed values for each weight range.

		HIGH S1	Δ	HIGH S2	Δ	RESTR S1	Δ	RESTR S2	Δ	PROTDEF S1	Δ	PROTDEF S2	Δ
25-65kg WEIGHT RANGE I													
PD	obs	187		153		139		146		70		74	
	pred	171	-8	173	+14	130	-6	128	-12^{t}	66	-5	71	-4
LD	obs	193		198		134		144		195		227	
	pred	177	-8	179	-9	130	+2	128	-9	255	+31***	278	+24**
65-105kg WEIGHT RANGE II													
PD	obs	171		192		145		148		51		57	
	pred	194	+13**	195	+3	137	-5^{t}	132	-9	37	-28**	50	-11
LD	obs	304		299		198		180		195		277	
	pred	200	-32*	201	-31*	137	-29*	132	-22	206	+11	283	+5
25-105kg BOTH WEIGHT RANGES													
PD	obs	174		175		145		149		57		64	
	pred	181	+4*	183	+5	135	-7^{t}	132	-11*	47	-18**	58	-10^{t}
LD	obs	244		254		169		163		189		250	
	pred	185	-23*	188	-25*	135	-20***	132	-18^{t}	223	+21**	277	+12**

Δ: %difference between predicted and observed, ***: P<0.001 **: P<0.01 *: P<0.05 t: P<0.10

Table 4 shows deposition rates of protein and lipid. Strain S1 showed the highest protein deposition rate (PD) in the first weight range (187 g/d), whereas strain S2 showed the highest level of protein deposition in the second weight range (192 g/d). On average over both weight ranges, both strains deposited similar amounts of protein (174 and 175 g/d). Protein deposition rate of strain S1 was 13% overestimated by the model in weight range II (P<0.01). Other predictions of PD were not significantly different from observed values. HIGH fed pigs deposited 196 g lipid per day on average in weight range I, and 301 g/d in weight range II. Predicted lipid deposition rates were not significantly different from observed values in the first weight range, although most predicted values were lower (on average 8%) than the observed values. Lipid depositions in the second weight range were underestimated by the model by 31% (P<0.05). In weight range I, the ratio of lipid to protein deposition (LD/PD) was 1.05 and 1.30 for Strain S1 and S2 respectively. In weight range II, respective values were 1.77 and 1.60. Prediction of this ratio was 1.04 in weight range I and 1.03 in weight range II. Thus, LD/PD was clearly (P<0.01) underestimated for both strains in the second weight range (figure 2).

Treatment RESTR

Restriction of feed intake as compared to treatment HIGH (treatment RESTR) resulted in a reduced rate of live weight gain in both weight ranges, on average 858 and 928 g/day respectively (table 3). The model underestimated this ADG in weight range I by 6% (P<0.05). In weight range II, the underestimation was 17 % (P<0.01). Feed conversion ratio was 1.86 in weight range I and 2.15 in weight range II. FCR was overestimated in both weight ranges, 7% (P<0.05) and 24% (P<0.01) respectively. With increasing live weight, the RESTR treatment shows an increasing lag between predicted and observed live weight (figure 1).

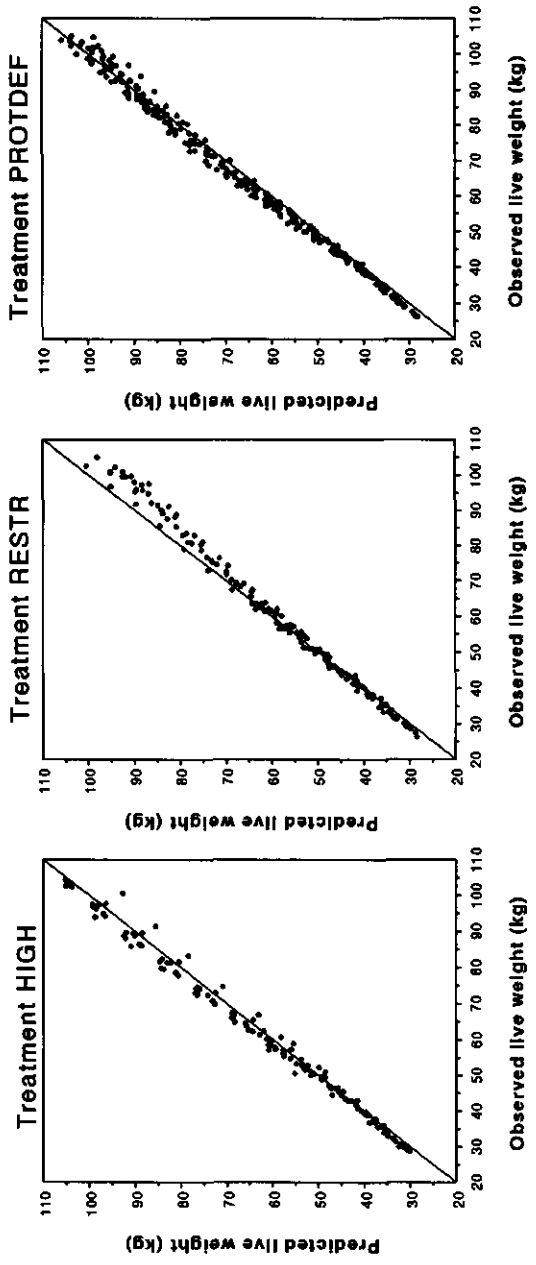

Figure 1. Predicted versus observed live weight (kg) for each treatment group.

The restrictedly fed pigs deposited about 145 g protein per day in both weight ranges. Most predictions of PD were similar to observed values. In weight range I, the protein deposition in the S2-animals was underestimated by 12% ($P<0.10$).

Lipid deposition was dependent on weight range ($P<0.01$). In weight range I, LD amounted 139 g/d on average, whereas LD amounted 190 g/d on average in weight range II. Although most predicted values were lower than the observed values, in the first weight range, predicted lipid deposition rates were not significantly different from observed values ($P>0.10$). Lipid depositions in the second weight range were underestimated by about 26% ($P<0.05$).

Below 65 kg live weight, the ratio between LD and PD was 0.98. Between 65 and 105 kg LW, this ratio was 1.30 on average. The model, however, assumes LD/PD to be constant (1.00) in both weight ranges. Therefore, LD/PD was predicted satisfactorily in weight range I, and LD/PD was underestimated ($P<0.01$) in weight range II (figure 2).

Treatment PROTDEF
In the protein deficient pigs, observed average daily gain was 629 g/d and 507 g/d in weight range I and II respectively. Respective feed conversion ratios averaged 2.85 and 3.78 (table 3). Both strains had similar ADG and FCR below 65 kg live weight. ADG and FCR were predicted well for S1 pigs in this range. On the other hand, ADG and FCR were somewhat over- and underestimated, respectively, for S2 pigs ($P<0.10$). In weight range II, ADG and FCR were dependent on strain. S1 pigs grew slower and had higher FCR as compared to S2 pigs. The predicted difference between the strains in this range was larger than the observed difference. ADG of the slower growing S1 pigs was underestimated to a larger extent then ADG of S2 pigs. Predicted and observed live weight agreed rather well in treatment PROTDEF.

Deposition rates of protein were 72 and 54 g/d for the two respective weight ranges. Protein deposition rate was predicted well in weight range I. PD was underestimated in range II, for S1 pigs to a larger extent than for S2 pigs. Lipid deposition was dependent on strain ($P<0.10$) and weight range ($P<0.01$). In range I, LD was 211 g/d, LD at the higher weight range averaged 236 g/d. Pigs of strain S2 consistently deposited more lipid than pigs of strain S1 did (table 4). Predicted deposition rates of lipid were higher than observed deposition rates. In the weight range I, the overestimation was 24-31% ($P<0.01$). In range II, it amounted 5 - 11%, but was not significantly different from 0.

The protein restriction resulted in lipid to protein deposition ratios of 2.9 and 4.3 in weight ranges I and II respectively. Lipid to protein deposition ratio was overestimated by 30-38% in weight range I ($P<0.01$). In weight range II, overestimation of the LD/PD of strain S1 was 60% ($P<0.05$). For strain S2, the difference between predicted and observed LD/PD in weight range II was 26% (figure 2).

Results - all treatments
In the model, water mass in the pig is calculated from protein mass (Water mass (kg) = $4.9 \times$Protein mass $(kg)^{0.855}$, Moughan et al., 1987). Water deposition is calculated from the increase in water mass. Discrepancies between observed and predicted protein mass

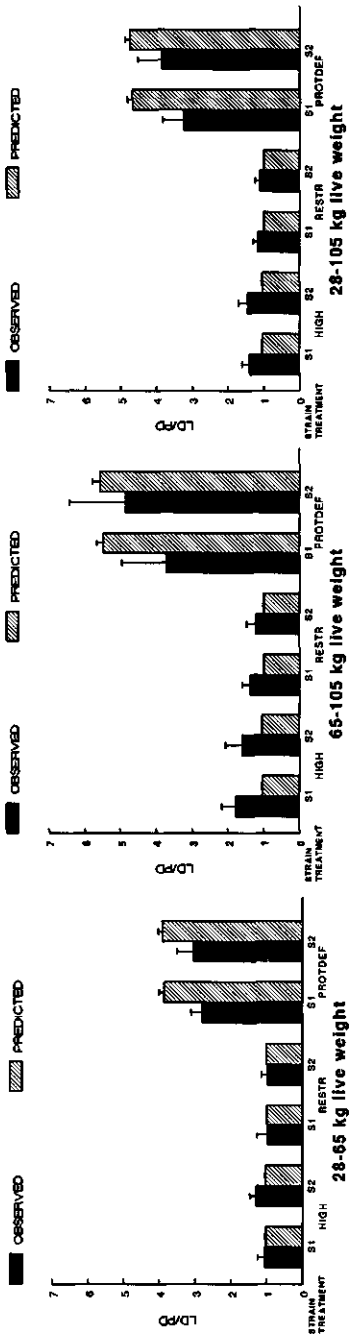

Figure 2. Observed and predicted lipid to protein deposition ratio

result in an incorrect prediction of water mass. Therefore, water deposition rate will also be predicted incorrectly. This interferes with the aim to validate whether calculation of water mass as dependent on protein mass is correct. Therefore, in figure 3, for all observations, the observed water mass is plotted against the observed protein mass. The line used by the model is also presented. Figure 3 shows a higher observed amount of water per unit of protein mass as compared to the line describing the prediction. Comparing the line with the observed data shows that observed water mass is on average 10% higher than water mass calculated by the model. The formula Water mass (kg) = 5.4×Protein mass $(kg)^{0.855}$) fits the observed data very well.

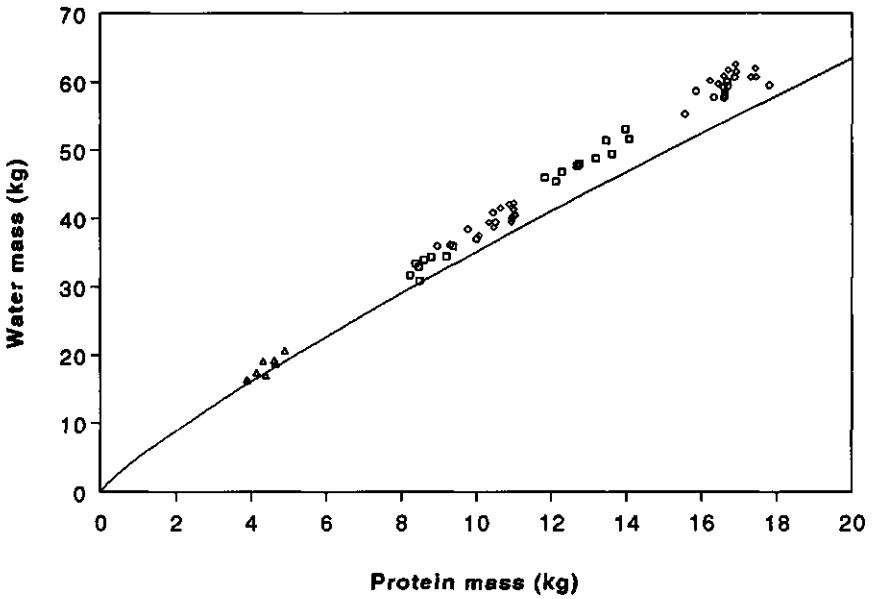

Figure 3. Observed and predicted (———) water mass as a function of protein mass for all treatment groups.

In order to evaluate the model in its ability to predict performance parameters for the whole fattening period, average daily gain and feed conversion ratio for the whole live weight range (28-105kg) and end-body composition are studied. These are shown in table 5.

Combining both weight ranges, average daily gain and feed conversion ratio are predicted with a deviation less than 2% from observed values for treatment HIGH. For treatment RESTR, ADG is underestimated between 10.3% (strain S1, $P<0.01$) and 13.7% (strain S2, $P<0.001$). Respective overestimations by the model of FCR for treatment RESTR are 13.7% ($P<0.01$) and 19.4% ($P<0.001$). Prediction of ADG and FCR for treatment PROTDEF were not significantly different from observed values (table 5).

Table 5. Overall performance parameters and body composition at 105 kg live weight, and %difference between predicted and observed values.

		HIGH S1	Δ	HIGH S2	Δ	RESTR S1	Δ	RESTR S2	Δ	PROTDEF S1	Δ	PROTDEF S2	Δ
25-105kg LIVE WEIGHT													
ADG	obs	1082		1107		892		909		503		593	
	pred	1078	0	1092	-1	798	-10**	784	-14***	476	-6^{t}	587	-1
FCR	obs	2.05		2.01		2.04		1.97		3.44		3.30	
	pred	2.05	0	2.04	+2	2.31	+14**	2.35	+19***	3.66	+6^{t}	3.35	+1
AT SLAUGHTER (105kg LIVE WEIGHT)													
%PR[a]	obs	16.8		16.5		17.1		17.3		13.5		12.9	
	pred	17.6	+5*	17.6	+7**	17.7	+3*	17.7	+2^{t}	12.3	-9*	12.3	-5
%LIP[b]	obs	20.0		20.4		17.4		16.9		31.4		35.2	
	pred	16.2	-19**	16.2	-20*	16.0	-8^{t}	16.0	-5	39.2	+26**	39.4	+13*

Δ: %difference between predicted and observed, ***: P<0.001 **: P<0.01 *: P<0.05 t: P<0.10
[a] %PR = %protein in the empty body [b] %LIP = %lipid in the empty body

Body compositions at 105 kg (table 5) show for treatments HIGH and RESTR that protein contents are overestimated and lipid contents are underestimated. In treatment HIGH, observed body lipid content was 20% for both strains. This was predicted to be 16.2% for both strains. After feed restriction (treatment RESTR), strain S1 had 17.4% lipid, and strain S2 had a body lipid content of 16.9%. Both strains were predicted by the model to have a body lipid content of 16.0%.

At 105 kg, PROTDEF-treated pigs were considerably fatter and had lower protein contents than HIGH and RESTR treated pigs. In contrast to treatments HIGH and RESTR, body protein content of treatment PROTDEF was underestimated ($P<0.05$) and lipid content was overestimated ($P<0.05$). The lipid content of strain S2 at treatment PROTDEF was 4% higher than that of strain S1. These two contents were predicted by the model to be the same (39%).

DISCUSSION

The model evaluated here was developed for pigs up to about 90 kg live weight (Moughan et al., 1987). For the present validation, it was extended for pigs up to 105 kg. This may be permitted as the sensitive assumptions are not very dependent on live weight e.g. protein deposition capacity (Whittemore et al., 1988), energetic efficiency of protein and lipid deposition (Kielanowski, 1972) and energy costs of maintenance (ARC, 1981). In general, the model predicted the effects of nutritional manipulation on rate of gain adequately. It is realized that the Massey model has been developed further since 1987. For present evaluation, the latest version which was published in a scientific journal in full detail was used (Moughan et al., 1987).

Prediction of the effects of treatment RESTR *versus prediction of effects of treatment* HIGH
Deposition rates for the whole period (28-105 kg LW) showed that protein deposition rate is overestimated at the high intake feed intake level (HIGH), and underestimated at the low feed intake level (RESTR). Daily gain was underestimated for treatment RESTR and

not for treatment HIGH. Validating their own model, Moughan et al. (1987) found a good fit between experimental values and model predictions. In their validation, predicted live weight fitted observed live weight very well at the higher level of feed intake. Average daily gain was underestimated somewhat at the lower feed intakes. Therefore, in Moughan's validation, the same trends occur with regards to average daily gain as compared to the present validation: the effect of restricted feed intake on daily gain is underestimated.

At the low feed intake level (RESTR), rate of gain was underestimated. A too low estimation of the minimal ratio of lipid to protein deposition (r) may be related to this. A low r will increase calculated live weight gain by changing deposition from lipid to protein. In weight range I, the predicted ratio of lipid to protein deposition fitted the observed ratio very well. In weight range II, the ratio was underestimated. Decreasing the r will enlarge this underestimation of ratio of lipid to protein. Thus, a lower assumed r would increase the accuracy of calculation of rate of liveweight gain, but would deteriorate the predicted ratio of lipid to protein deposition. As the model calculations are based on protein and lipid gain, the composition of the gain is more important than the rate of gain. Thus, for both weight ranges, changing the value of the parameter r will not improve predictions of ratio of lipid to protein deposition.

Underestimation of daily gain by the model for the restricted fed pigs may be related to the energy cost for maintenance. In the validated version of the Massey model, energy costs for maintenance are assumed to be a function of protein mass. This function (MJ) is 1.85×protein mass $(kg)^{0.78}$ (Moughan et al., 1987). For the RESTR-animals, this corresponds to 520 kJ/kg metabolic weight ($W^{0.75}$) at 25 kg; 512 kJ/kg metabolic weight at 65 kg and 510 kJ/kg metabolic weight at 105 kg. These values are high as compared to estimates of maintenance requirements derived from literature by Van Es (1972) and ARC (1981). A high maintenance requirements results in a low amount of energy available for production. Increase in energy available for production in treatment RESTR would have increased the calculated level of protein and lipid deposition. Because these animals were fed below their protein deposition capacity, this means (Moughan et al., 1987) that they will deposit the minimum ratio of lipid to protein deposition. Therefore, a lower estimation of maintenance energy would have increased average daily gain and not have influenced the ratio of lipid to protein deposition for treatment RESTR. Thus, overestimation of maintenance may cause the underestimation of daily gain in treatment RESTR. Moughan (1985) already mentioned the sensitivity of the model for maintenance requirements estimates. Overestimation of maintenance costs will be smaller for treatments HIGH and especially PROTDEF, as their protein masses were lower than those of RESTR.

Under- and overestimation of lipid deposition dependent on type of restriction

The estimate of maintenance requirements or the energy cost of tissue deposition may be related to the underestimation of lipid deposition in protein sufficient fed pigs. As mentioned in the previous paragraph, maintenance requirements in the validated version of the Massey Model are a function of protein mass. For the PROTDEF-animals, the model calculated maintenance as 520 kJ/kg metabolic weight at 25 kg; 436 kJ/kg metabolic weight at 65 kg and 417 kJ/kg metabolic weight at 105 kg. These values are comparable to literature (Van Es, 1972; ARC, 1981). Thus, overestimation of lipid deposition in PROTDEF animals is not caused by a too low assumed maintenance requirement. Animals

which deposit high amounts of body fat, like the protein deficient pigs, can directly incorporate feed-lipid into body lipid. This is energetically much more efficient than accretion of lipid from other nutrients (Millward et al., 1976). In the present experiment, the trend was the opposite, the animals were even more inefficient than expected. There is no obvious explanation for such a reduced energetic efficiency induced by protein deficiency.

The effect of live weight on the ratio of lipid to protein deposition
At all treatments, body lipid contents increased with slaughter weight. The ratio of lipid to protein deposition was higher above 65 kg live weight than before 65 kg. Both for treatments HIGH and RESTR, the model calculated the same lipid to protein ratios for the two weight ranges. Whittemore (1986) also suggested such an independency on live weight of composition of deposited tissue below *PDmax*. There are no clear experimental data about *r* in literature. Present results suggest that the minimal fat to protein deposition ratio increases with weight. Results of Black and Griffits (1975) in lambs also suggest such a weight-dependent amount of lipid deposited per unit of deposited protein (Black et al., 1986).

Strain effects
Both strains are hybrid lines. Strain S1 is a sire line. Strain S2 is an end cross product, aimed at using as a commercial fattening pig. Strain S1 is used to sire the father line of strain S2, 25% of the genetic background of strain S2 is provided by strain S1. Results of treatment HIGH suggest a difference between the strains in partitioning of their energy towards protein and lipid deposition. Below 65 kg LW, composition of growth of strain S1 is leaner than that of strain S2 and above 65 kg LW, it is fatter than S2. At 105 kg body weight, body compositions were similar. Therefore, the two strains, which are genetically related, are different in their response to nutrition. The model only accounts for variation in the input parameters Feed intake, Feed composition and Capacity of the animals (*PDmax*). These parameters were comparable for both strains. Therefore, certain differences between strains are not characterized by taking *PDmax* as the only characterization of the animal. The present study shows that strains were similar with regard to performance parameters like ADG and FCR. Differences in deposition rates and associated chemical body composition were apparent. This emphasises the fact that in studying small differences between animals or treatments, chemical body composition has to be taken into account.

Prediction of deposition rate of water
Results show that the predicted level of water deposition as dependent on protein mass is lower than the observed value. The formula which is used by the model originates from over 20 years ago (Kotarbinska, 1969). The selection towards leaner pigs in the last decades may have resulted in higher amounts of water relative to protein. Literature reports leaner types of pigs to have higher amounts of water for each unit of protein (Henderson et al., 1983; Campbell and Taverner, 1988). Increasing the amount of calculated water with 10% fitted the observed values very well. It is interesting to note that there is no effect of the amount of lipid in the body of pigs on the relation between protein mass and water. The decrease in water content coinciding the increase in lipid content is merely a result of the decrease in lean body mass content.

CONCLUSIONS

The validated growth model calculates protein deposition rate well for our range of nutritional manipulations. Results from the present experiment show that the ratio of lipid to protein deposition rate increases with increasing live weight. More estimates of this parameter are needed.

For pigs with relatively lean body compositions, the model estimated maintenance requirement of pigs about 20% higher than advised by ARC (1981). This influences the prediction of energy available for production. The present work suggests that maintenance requirements are lower than the values based on body protein contents. Using the ARC (1981) maintenance requirements would have improved predictions of the model in leaner animals.

Overestimation of lipid deposition at protein deficient rations suggests that efficiency of energy deposition at protein deficient diets for pigs is lower than assumed in literature.

Prediction of live weight gain is very dependent on the prediction of the protein and lipid deposition rates. When protein and lipid deposition are predicted well, prediction of water deposition rate determines the quality of predicting average daily gain. When the amount of water calculated by the validate model is increased by 10%, predicted average daily gain is close to observed daily gain.

ACKNOWLEDGEMENT

The provision of a Basic-program of the 1984 version of the Massey model by dr P.J. Moughan of Massey University, Palmerston North, New Zealand is greatly acknowledged. This version provided us the opportunity to verify our Fortran-version of the model as described in Moughan et al. (1987).

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Chapter II

A NOTE ON THE RELATION BETWEEN ENERGY INTAKE AND LIPID AND PROTEIN DEPOSITION IN GROWING PIGS

K.H. DE GREEF, M.W.A. VERSTEGEN AND R.G. CAMPBELL

SUMMARY

Data from three experiments presented in literature which support the linear relation between energy intake and protein deposition were studied. Aim of this was to check whether these data also support the assumption of the Linear-Plateau concept that there is a constant ratio between lipid and protein deposition at energy intakes lower than needed for maximal protein deposition. The study revealed that not only protein deposition, but also lipid deposition was related linearly to energy intake. However, the ratio between lipid deposition and protein deposition increased with each increase in energy intake. Therefore, results of this study questioned one of the major assumptions of the Linear-Plateau concept, the constancy of the minimal ratio of lipid to protein deposition. It was derived that, on theoretical grounds, one can indeed expect a constancy in the relation between protein deposition and lipid deposition. However, there is not a constant ratio between *total* lipid deposition and *total* protein deposition, but a constant ratio between *extra* lipid deposition and *extra* protein deposition. This extra deposition is the deposition of tissue caused by an extra unit of energy intake. Every increase in energy intake results in fatter composition of growth because the ratio between *extra* lipid and protein deposition is larger than the ratio between *total* lipid and protein deposition.

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A NOTE ON THE RELATION BETWEEN ENERGY INTAKE AND LIPID AND PROTEIN DEPOSITION IN GROWING PIGS

INTRODUCTION

Tissue deposition in growing pigs is dependent on many animal and environmental factors. The most important of these are the animals inherent capacity for protein accretion and nutrient intake. Several concepts of influences on protein deposition have been proposed in literature (Whittemore and Fawcett, 1976; Carr, Boorman and Cole, 1977; Fowler, 1978; Fowler, Fuller, Close and Whittemore, 1980; ARC, 1981). The concept of a linear-plateau response, as proposed by Whittemore and Fawcett (1976), is commonly used in pig growth models to describe the relationship between energy intake and protein deposition in a protein adequate situation. This concept was demonstrated experimentally for several types (genders and strains) of pigs by Campbell, Taverner and Curic (1983 and 1985) and by Campbell and Taverner (1988). However, models using the Linear-Plateau concept generally incorporate the additional assumption that the ratio of lipid to protein deposition at protein balanced energy intakes below maximal protein deposition (plateau value) is constant (Whittemore and Fawcett, 1976), but may vary amongst types of pigs. The studies on the effects of protein balanced energy intake on tissue deposition were focused on protein deposition. Lipid deposition was measured accordingly, but not studied extensively. Further knowledge of the lipid deposition accompanying that protein deposition will improve the understanding of the partitioning of energy between protein and lipid deposition. In the present paper, the results of the experiments supporting the Linear-Plateau concept are used to investigate whether, below maximal protein deposition, the ratio between lipid deposition and protein deposition is a constant within a type of pig.

MATERIAL

In a series of experiments protein deposition was studied in pigs as a function of protein intake and energy intake (see Campbell, 1988). The authors found that the response of protein deposition to increasing intakes of either nutrient could be described by a linear-plateau function. The present paper uses the results of experiments where protein deposition rate was studied as a function of increasing energy intake in the presence of sufficient protein intake (Campbell et al., 1983 and 1985; Campbell and Taverner, 1988) to investigate the consequent effects on lipid deposition and the lipid to protein deposition ratio. The effect of energy intake was studied in male and female pigs between 20 and 45 kg live weight (Campbell et al., 1983). This was studied between 48 and 90 kg live weight by the same authors (Campbell et al., 1985). Between 45 and 90 kg live weight, the effect of energy intake on tissue deposition was studied in boars and castrates of one strain, and in boars of another, improved strain (Campbell and Taverner, 1988). Published averages of treatment groups were used for the present evaluation. When referencing to energy intake, the situation of ample protein supply is meant.

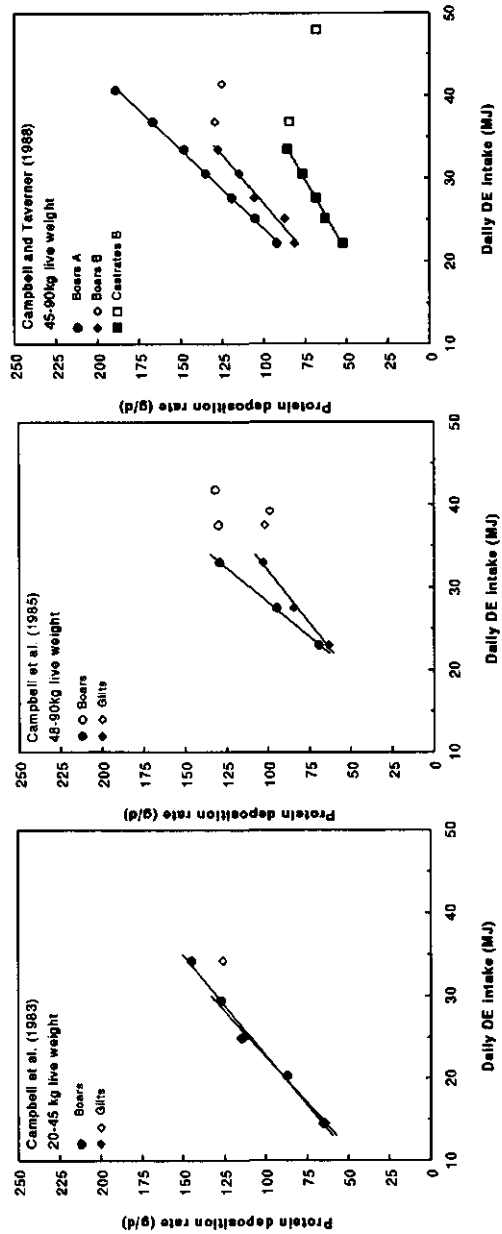

Figure 1. Protein deposition rate as a function of daily DE intake. Open symbols indicate that maximum protein deposition has been reached.

RESULTS

In figure 1, rate of protein deposition is plotted against daily digestible energy intake for the three experiments studied. These plots clearly show the linear response of protein deposition to increasing energy intake, with a plateau level being reached in most cases (Campbell, 1988). Both the slope of the linear part and the maximal protein growth (plateau level) varied across and within experiments. Linear estimates of the relation between energy intake and protein deposition below the plateau in protein deposition are also presented.

The results also show that lipid deposition rate increases with increasing energy intake. The effects of energy intake on lipid to protein ratio are shown in figure 2. This figure is important to test the hypothesis whether at protein deposition rates below the plateau rate, the ratio of lipid to protein deposition for any given type of pigs is relatively constant. These data show that at energy intakes below that required to support maximal protein deposition (filled symbols), increasing energy intake has a larger effect on lipid deposition than on protein deposition. Thus, below the plateau, the ratio between lipid and protein deposition rate increases with increasing energy intake. Despite some variation between successive groups of pigs, this effect is clear in all three experiments studied (figure 2). Above the plateau of protein deposition, increasing energy intake further increases the lipid to protein deposition ratio.

DISCUSSION

The results of the studied experiments (Campbell et al., 1983 and 1985; Campbell and Taverner, 1988) clearly show a linear response of protein deposition to increasing energy intake. In some experimental groups, within the range of energy intakes studied, protein deposition plateaued. These results strongly support the concept of linear-plateau response as proposed by Whittemore and Fawcett (1976). Therefore, the assumption of a linear-plateau response of protein deposition to increasing energy intake in several growth models (Whittemore and Fawcett, 1976; Whittemore, 1983; Moughan, Smith and Pearson 1987; Watt, DeShazer, Ewan, Harold, Mahan and Schwab, 1987) seems valid. Furthermore, the data sets show that the amount of lipid deposited is also a function of energy intake. An important aspect is that over the linear component of the relationship between energy intake and protein deposition the lipid to protein deposition ratio increases with increasing energy intake. This belies and seriously questions the frequent assumption in growth models that lipid to protein ratio below protein deposition capacity is constant within a given pig type (Whittemore and Fawcett, 1976; Whittemore, 1983; Moughan et al., 1987; Watt et al., 1987).

The linearity of the increase in lipid deposition over the linear relationship between energy intake and protein deposition is clearly demonstrated in table 1, which presents linear regressions of protein and lipid deposition on DE intakes for the experiments studied. Only observation below the plateau for protein deposition are included. According to the r^2 values, the regressions fit well. This implies that both protein deposition and lipid deposition are linearly related to energy intake. The r^2 values are relatively high due to the relatively low number of observations (3-7 group averages per regression line).

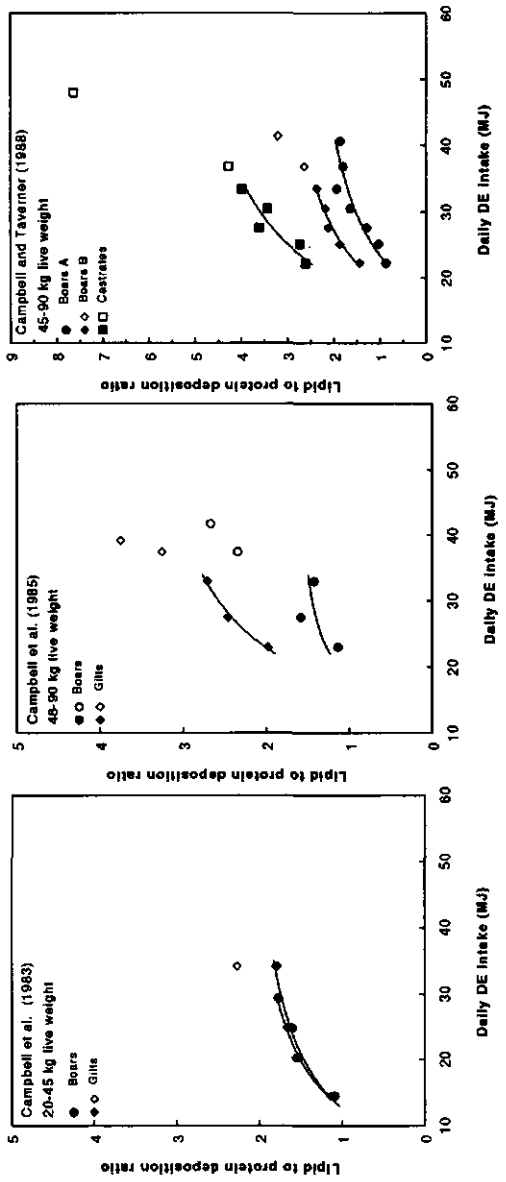

Figure 2. Ratio between lipid and protein deposition rate as a function of digestible energy intake. Filled symbols indicate that protein deposition rate is below its maximum. Open symbols indicate that protein deposition capacity has been reached.

Table 1. Quantification of the relation between DE intake (DEI, MJ/d) and deposition rate of protein (PD, g/d) and lipid (LD, g/d). Data from published means in Campbell et al. (1983 and 1985) and Campbell and Taverner (1988).

Source	sexe	#	Protein deposition	r^2	Lipid deposition	r^2	Marginal ratio
			PD = a + b × DEI		LD = c + d × DEI		d/b
1	M	5	PD = 5.84 + 4.13 × DEI	(0.990)	LD = - 64.42 + 9.69 × DEI	(0.994)	2.35
1	F	4	PD = - 0.90 + 4.46 × DEI	(0.986)	LD = - 78.69 + 10.59 × DEI	(0.995)	2.37
2	M	3	PD = -69.68 + 6.02 × DEI	(0.999)	LD = -150.19 + 10.34 × DEI	(0.935)	1.72
2	F	3	PD = -25.98 + 3.94 × DEI	(0.991)	LD = -222.10 + 15.31 × DEI	(0.990)	3.89
3	M	7	PD = -25.40 + 5.23 × DEI	(1.000)	LD = -265.55 + 15.52 × DEI	(0.973)	2.97
3	M	5	PD = -15.03 + 4.26 × DEI	(0.976)	LD = -238.55 + 16.17 × DEI	(0.986)	3.80
3	C	5	PD = -10.55 + 2.87 × DEI	(0.995)	LD = -261.87 + 17.77 × DEI	(0.966)	6.19

Source: 1 = Campbell et al. (1983); 2 = Campbell et al. (1985); 3 = Campbell and Taverner (1988).
Sexe: M = male; F = female; C = castrate
#: number of treatment groups used in the regressions

The slope of the line describing protein deposition quantifies the amount of *extra* of protein which is deposited from each *extra* unit of energy intake. This leaves a constant amount of the *extra* energy for deposition of *extra* lipid. This implies the linear relation between lipid deposition and energy intake. Thus, the observed linear relation between energy intake and lipid deposition could be expected from the linear relation between energy intake and protein deposition.

The ratio between lipid and protein deposition can be assessed by dividing the line describing lipid deposition by the line describing protein deposition. In figure 2, these lines are presented together with the observed data. The ratio between the two linear lines is a non linear relation, due to the different intercepts of the lines describing protein and lipid deposition.

These lines demonstrate that each increase in energy intakes results in an increase in the ratio between deposited lipid and deposited protein. The linear relationships between energy intake and PD and LD imply that the ratio between *extra* lipid deposited and *extra* protein deposited from energy intake (DEI) is constant. This ratio between *extra* lipid deposition and *extra* protein deposition can be calculated from table 1 by dividing the slopes of both lines. This value is presented in most right column of table 1 as the marginal ratio. It represents the ratio between *extra* lipid and *extra* protein deposition from one extra unit of energy intake. This marginal ratio is larger than the ratio's of *total* lipid to protein deposition. Therefore, each additional unit of energy intake produces a fatter pig. The lowest value for this ratio is 1.79, for the entire males in Campbell et al. (1985). For the castrates in Campbell and Taverner (1988) the ratio is 6.19. The differences clearly illustrate major animal factors, like gender, influencing energy partitioning between protein and fat deposition.

In growth response prediction, it is important to consider the increase in the relative amount of lipid deposited for a given change in energy intake. The frequently used assumption that, below maximal protein deposition, there is a constant ratio of lipid to protein deposition for any given pig type is challenged by the present results. For accurate prediction of responses to increasing energy intake, the assumed constant ratio between

protein and lipid deposition below maximal protein deposition at increasing levels of energy intake (Whittemore and Fawcett, 1976; Whittemore, 1983) has to be reconsidered. A better quantification and understanding of energy partitioning is crucial for accurate prediction of performance and body composition in growing pigs. In the authors lab, experiments have been performed in order to quantify the effects of body weight and energy intake on the relation between protein and lipid deposition. Results from these studies will be published in a following paper (De Greef, Verstegen, Kemp and Van der Togt, 1992).

In conclusion, it can be stated that the linear-plateau response in protein deposition of pigs to increasing energy intake is accompanied by an increase in the relative amounts of lipid deposited. Therefore, each increase in energy intake produces a fatter pig. This is not accounted for in many published porcine growth models, but is essential for accurately predicting the responses of pigs to different amounts of energy. It was concluded that when protein deposition is linearly related to energy intake, the ratio between *extra* lipid and *extra* protein deposition is constant, but not the *total* (actual) lipid to protein deposition ratio.

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Chapter III

THE EFFECT OF BODY WEIGHT AND ENERGY INTAKE ON THE COMPOSITION OF DEPOSITED TISSUE IN PIGS

K.H. DE GREEF, M.W.A. VERSTEGEN, B. KEMP AND P.L. VAN DER TOGT

SUMMARY

Many swine growth models assume that there is no effect of energy intake and of body weight on the ratio of lipid to protein deposition rate in pigs below their maximal protein deposition rate. An experiment was performed to check whether an effect of body weight and of amount of energy intake on this partitioning of energy is absent. Two constant amounts of energy were given above maintenance requirement (12.6 and 16.3 MJ DE per day for production, treatment LOW and HIGH, respectively). A total of 52 entire male pigs were slaughtered at 25, 45, 65, 85 or 105 kg live weight. Results showed that, for both levels of intake, the ratio of lipid to protein deposition rate increased with increasing body weight. At the LOW energy intake, the ratio of lipid to protein deposition rate increased from 0.74 at 25 kg to 0.99 at 105 kg body weight. In animals receiving the HIGH treatment, the ratio of lipid to protein deposition rate increased from 0.82 to 1.35 in that weight range. This change in nutrient partition was also reflected in daily gain. Daily gain declined with increasing live weight, a decrease of 150 grams/day over the weight range 25-105 kg. The 3.7 MJ DE difference in energy intake between treatment HIGH and LOW resulted in an average overall difference of 105 g daily gain. An *ad libitum* fed control group showed that protein deposition capacity was above 200 g/day, thus the pigs at the LOW and HIGH treatment were below their protein deposition capacity. It was concluded that both live weight and energy intake influence the ratio of lipid to protein deposition rate. The mechanism of partitioning between lipid and protein deposition needs further specification in order to improve the predictions of growth models which use the Linear-Plateau concept.

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THE EFFECT OF BODY WEIGHT AND ENERGY INTAKE ON THE COMPOSITION OF DEPOSITED TISSUE IN PIGS

INTRODUCTION

Most swine growth models suggest a constant ratio of lipid to protein deposition rate when pigs are fed below their protein deposition capacity (Whittemore and Fawcett, 1976; Moughan, Smith and Pearson, 1987; Watt, DeShazer, Ewan, Harold, Mahan and Schwab, 1987; Pomar, Harris and Minvielle, 1991). In this approach (Whittemore, 1983), the ratio between lipid and protein deposition rate is independent of live weight and of energy intake. It would be expected, therefore, that at low levels of feeding additional energy intake does not increase fatness. It also means that when given equal amounts of energy above maintenance requirement, composition of tissue gain is the same at low and high body weights. An experiment was designed to verify this concept. The effects of live weight and of energy intake on the partitioning of retained energy into protein and lipid deposition were studied independently. This was achieved by giving animals in various weight ranges one of two different but constant amounts of energy above maintenance requirements. The primary aim of the study was to investigate whether composition of gain depends on body weight and on energy intake, tested at two fixed amounts of energy above maintenance.

MATERIAL AND METHODS

Experiment

The experiment was performed with entire male pigs of a commercial synthetic cross. The pigs were fed a constant amount of energy above their maintenance requirement. Maintenance requirement (kJ ME/day) was quantified as 719 × Live weight$^{0.63}$ (ARC, 1981). ME is assumed to be 0.96 of DE (ARC, 1981). The daily energy allowance was either 12.6 MJ DE above maintenance requirement (treatment LOW) or 16.3 MJ DE above maintenance (treatment HIGH). Protein allowance was above ARC (1981) recommendations. Other essential nutrients were also assumed non limiting. Water was available *ad libitum*. Pigs were weighed twice a week. Feed allowances were adjusted after each weighing of the animals. The pigs were housed individually in pens in a thermoneutral environment and were fed twice daily.

Composition of the experimental ration is presented in table 1. Digestibility of energy and crude protein of the diet was measured in a balance trial using 4 boars drawn from the same batch as the pigs in the experiment. The balance trial was performed at an average live weight of 50 kg.

In table 2, numbers of animals in each treatment group are presented. At 25 kg live weight, all pigs were randomly assigned to the experimental groups of four pigs each. The first group was slaughtered at 25 kg live weight. Four treatment groups were fed according to treatment LOW and slaughtered at 45, 65, 85 and 105 kg live weight, respectively. Four other groups were fed according to treatment HIGH and were also slaughtered at subsequent 20 kg live weight intervals, identical to treatment LOW. In order to derive

whether the HIGH fed pigs were producing below their maximal protein deposition capacity (Whittemore and Fawcett, 1976; Campbell, 1988), four groups of four control pigs were fed identical to pigs of treatment HIGH, but were fed *ad libitum* in the last 20 kg of their live weight range. Results of these *ad libitum* fed pigs are used to estimate the protein deposition capacity of the animals in this Target weight range. Thus, in the experimental design, in each of four weight intervals there were three treatment groups: LOW, HIGH and ADLIBITUM.

Table 1. Composition of the experimental ration.

Ingredients	(%)	Analyzed composition	(g/kg)
Maize	32.5	Crude protein	186
Wheat	20	Moisture	122
Peas	10	Ether extract	68
Soya bean meal extruded	14.9	Crude fibre	26
Cane molasses	2.5	Ash	57
Skim milk powder	5.3		
Fish meal	1	Total lysin	11.3
Animal meat meal	1.6	Total methionine	4.0
Animal fat	3.8	Total threonine	8.4
Soya oil	1.2		
Lysin 10%	3.42	Digestible crude protein	159
Methionine 10%	1.34	Digestible energy (MJ/kg)	14.6
Threonine	0.16		
Tryptophan	0.04		
Salt	0.1		
Choline chloride	0.1		
Mono calcium phosphate	0.9		
Calcium propionate	1.0		
Vitamins and minerals	0.1		

At slaughter, blood and organs were collected separately. After emptying the entrails, individual organs and the blood were weighed, stored in plastic bags and frozen. The carcass was scalded, scraped and split longitudinally. After weighing both halves, the right half was frozen in a plastic bag to avoid water loss. The right carcass half (including head) was dissected into trimmed major joints according to the Dutch standard dissection method (Bergström en Kroeske, 1968; Walstra 1980). All dissected material was frozen in plastic bags. After homogenization of the frozen material in a cutter, chemical analysis (dry matter, nitrogen, lipid and ash) was performed in the three tissue groups: (*i*) blood and organs (*ii*) carcass dissected lean and (*iii*) other carcass parts. Dry matter content of the samples was assessed using a vacuum oven (4 kPa) at 50°C, using anhydrous calcium chloride as a drying agent. Nitrogen content was measured using the Kjeldahl technique, lipid was determined by petroleum-ether extraction. Ash content of the samples was analyzed using an oven at 500°C. Total body composition was calculated from the chemical composition of the three tissue groups and their respective weights. Empty body weight was calculated by summation of all body components collected at slaughter.

Calculations and statistics

Observations made on the 20 kg weight range before slaughter weight (the Target weight range) were used for statistical analysis of daily gain, body protein percentage and body lipid percentage. In this way, each experimental observation was made using different animals and therefore all observations were statistically independent. In this statistical analysis, only LOW and HIGH treatment groups were taken into account. The ADLIBITUM treatment was not incorporated in this model. This treatment group was used only to determine the upper limit of performance (*i.e.* protein deposition capacity) in the target weight ranges.

Table 2. Experimental design and number of animals in each treatment group.

Target weight range	-	25-45	45-65	65-85	85-105
Slaughter weight	25	45	65	85	105
Intake above maintenance					
-	4				
12.6 MJ DE (LOW)		4	4	4	4
16.3 MJ DE (HIGH)		4	4	3[1]	3[2]
ADLIBITUM		4	4	4	4

[1] one pig of treatment HIGH, Target weight range 65-85 died during the experiment
[2] one pig of treatment HIGH, Target weight range 85-105 was excluded in calculations due to illness and feed residuals.

The effects of Weight Range (WR_i, i=25-45, 45-65, 65-85, 85-105 kg) and Production Energy (PE_j, j=LOW, HIGH) on average daily feed intake, average daily gain and body composition (percentage protein and lipid in the empty body) were tested using the SAS-GLM procedure (SAS, 1990) with the model

$$\underline{Y}_{ijk} = \mu + \text{Weight Range}_i + \text{Production Energy}_j + WR{\times}PE_{ij} + \underline{e}_{ijk}$$

Calculation of deposition rates of protein and lipid in weight ranges requires an initial slaughter group at the beginning of each weight range. However, the increase in body weight in each weight range in the present experimental design is small in relation to the between animal variation in the initial group and in the treatment group. This means that the intervals for the successive classes of live weight are too short to use the slaughtered animals of a weight range as the initial slaughter group of the successive weight range (Süsenbeth, 1984). For this reason, another approach was chosen, by expressing the amount of protein and lipid as a function of body mass. Protein mass and lipid mass (Y, kg) were expressed as a function of empty body weight (EBW, kg) using the model

$$Y = a{\times}EBW^b \quad (1)$$

This model (Huxley, 1932) was chosen because it adequately describes the development of a tissue group (here: protein and lipid) as a function of body weight (Evans and Kempster, 1979; Moughan, Smith and Stevens, 1990). The parameters a and b were calculated for both feeding levels separately and for lipid and protein mass separately by regression (SAS, 1990). The goodness of fit of the curves was quantified by the coefficient of determination (r^2) and by the degree of autocorrelation (Durbin-Watson's D-statistic: SAS, 1990). Formula (1) describes the relation between total body mass (EBW) and either

protein or lipid mass. The first derivate of formula (1) describes the relation between the change in units of the component (Protein of Lipid) with one unit change in total empty body mass (2).

$$dY/dEBW = b \times a \times EBW^{b-1} \quad (2)$$

The ratio of lipid to protein deposition is derived by dividing formula (2) for Lipid by the formula (2) for Protein. In this way, the ratio between lipid and protein deposition rate as a function of body weight is quantified.

RESULTS

In table 3, data on performance in the target weight range and body composition at slaughter of treatment groups LOW and HIGH are presented. Significance of the experimental factors is presented in table 4.

Table 3. Daily feed intake and live weight gain in the target weight ranges and empty body parameters at slaughter.

Target weight range (kg)		25-45		45-65		65-85		85-105	
Slaughter weight (kg)		45		65		85		105	
	Treatment	mean	sd	mean	sd	mean	sd	mean	sd
Feed intake	LOW	1344	9	1502	4	1630	4	1744	6
(g/d)	HIGH	1573	27	1759	8	1889	4	2010	4
Daily gain	LOW	806	46	751	64	753	37	695	104
(g/d)	HIGH	924	9	856	68	879	71	765	59
At slaughter:									
Empty body weight	LOW	42.6	1.1	61.6	0.5	81.4	2.1	98.6	1.1
(kg)	HIGH	42.1	1.0	60.7	1.1	79.3	0.4	98.7	3.3
Protein	LOW	16.7	0.6	17.5	0.2	18.0	0.2	17.7	0.2
(% in empty body)	HIGH	16.6	0.4	17.0	0.1	17.3	0.5	17.5	0.6
Lipid	LOW	11.3	0.7	13.5	0.7	13.8	1.6	15.5	1.2
(% in empty body)	HIGH	12.5	0.7	14.6	1.1	15.7	1.7	19.1	2.3

Average daily gain was affected both by amount of production energy ($P<0.001$) and by weight range ($P<0.01$). There was no significant interaction between production energy and weight range. On average, the difference of 3.7 MJ digestible energy per day resulted in a difference in average daily gain of 105 g. Live weight gain decreased with increasing live weight at both levels of energy intake. Between 25 and 105 kg live weight, the estimated effect of live weight on average daily gain amounted to -1.65 and -2.24 gram/kg live weight change for treatments LOW and HIGH respectively.

Table 4. Significance of the experimental factors on the tested parameters.

	WR	PE	WR×PE
Daily gain	**	***	ns
Protein % in empty body	***	*	ns
Lipid % in empty body	***	***	ns

WR: Weight range; PE: Production Energy
Significance: *:P<0.05; **:P<0.01; ***:P<0.001

Pigs slaughtered at 25 kg live weight contained 16.1% (sd=0.2) protein and 10.3% (sd=0.6) lipid in the empty body. In the experimental groups, body protein percentage was affected by weight class (P<0.001) and by amount of production energy (P<0.05). Body lipid percentage increased with increasing live weight (P<0.001) and with increasing energy for production (P<0.001).

Table 5. Parameter estimates and quality parameters of the relation between empty body weight (kg) and protein and lipid mass (kg) for both treatment groups using the model aEBW^b.

	Treatment	a	b	se (b)	r^2	DW-D	autocorr
Protein mass	LOW	0.1271	1.0754	0.0096	0.9997	1.394	0.189
	HIGH	0.1351	1.0562	0.0099	0.9994	2.372	-0.263
Lipid mass	LOW	0.0420	1.2768	0.0034	0.9936	1.776	0.100
	HIGH	0.0289	1.3970	0.0362	0.9912	2.596	-0.319

DW-D: Durban Watson D-statistic; autocorr: degree of autocorrelation

In figure 1, protein and lipid mass as quantified by the model a×EBW^b are plotted against the empty body weight. As table 5 shows, the estimated curves fit the data well (r^2 >0.99) and there is no significant autocorrelation within the lines. Protein mass and lipid mass both have b-values larger than 1. This indicates that both tissues increase faster than empty body weight. For both treatments, lipid mass increased faster than protein mass. This is indicated by the parameters describing lipid mass, which have higher values of parameter b as compared to the parameters describing protein mass as a function of empty body weight in formula (1).

From the parameters in table 5, it can be calculated that at an empty body weight of 61 kg (live weight=65kg), protein mass was 0.2 kg higher for the LOW pigs as compared to the HIGH pigs. This difference increased to 0.5 kg at an empty body weight of 99 kg (105 kg live weight). Lipid mass was 1.0 and 2.9 kg lower for LOW fed pigs at 65 and 105 kg live weight respectively compared to HIGH pigs. Therefore, a constant difference of 3.7 MJ of digestible energy intake per day from 25 kg live weight onwards resulted in a difference of about 3 kg of lipid at 105 kg live weight.

The parameters describing the amount of lipid and protein as a function of empty body weight (figure 1 and table 5) can be used to calculate composition of deposited tissue. In figure 2, the ratio between lipid and protein deposition rate calculated from the lines describing protein and lipid mass is drawn. For both treatments, the ratio between lipid and protein increased with live weight. For treatment LOW, the estimated ratio between

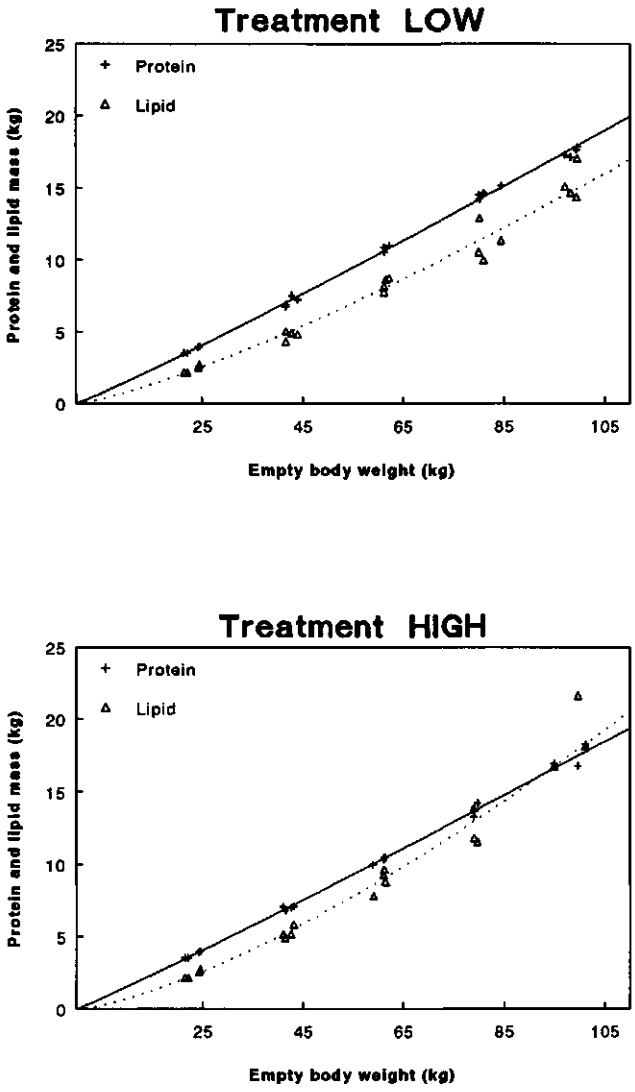

Figure 1. Observed protein and lipid mass (kg) and their described curves as a function of empty body mass (kg).

lipid and protein is 0.74 at 25 kg live weight (23 kg empty body weight), and increased to 0.99 at 105 kg live weight (99 kg empty body weight). For treatment HIGH, the ratio increased from 0.82 at 25 kg live weight to 1.35 at the highest slaughter weight. Within the weight range 25-105 kg live weight, both lines in figure 2 are virtually linear. Within this weight range, each kilogram increment of live weight increases the ratio between lipid and protein deposition rate by 0.0031 units and 0.0066 units for treatment LOW and HIGH respectively.

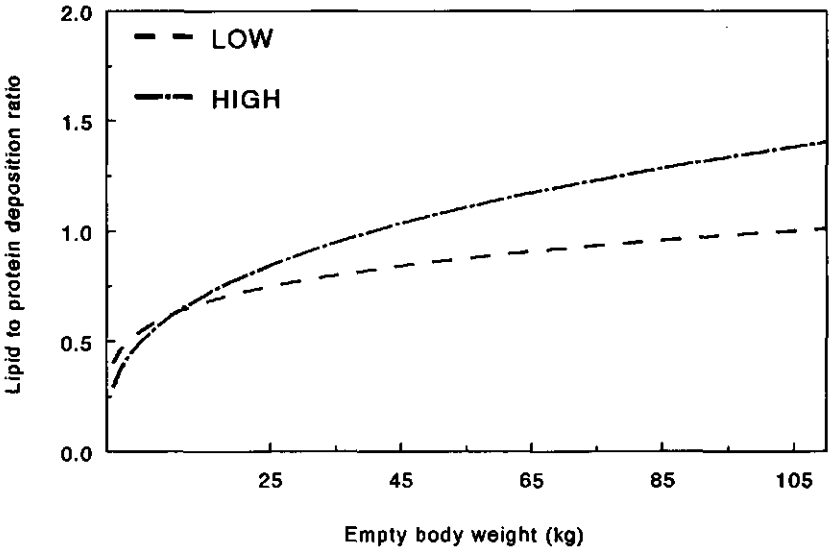

Figure 2. Ratio between lipid deposition and protein deposition as a function of empty body weight, calculated using the parameters in table 5.

DISCUSSION

Method of quantifying the effect of live weight.

The effect of treatments on composition of deposited tissue can be estimated in several ways. The most commonly used method is to analyze the experiment as a comparative slaughter experiment. In this approach, animals receiving the same treatment but slaughtered at a lower live weight are used as a reference to quantify the body composition of the remaining animals at the beginning of their experimental range. The increase of body weight in the experimental range has to be at least half of the initial body weight (Süsenbeth, 1984). Therefore, within the normal weight range for fattening pigs (20 to 90-105 kg), the comparative slaughter technique may not be sensitive enough to predict tissue deposition rates in more than two separate live weight ranges. Besides this insensitivity there is the problem of dependency between successive experimental groups. Each group will influence the estimations of two successive live weight ranges. In one weight range, it is the experimental group, in the next weight range, it is the reference group. For this reason, in the present paper, it was chosen to estimate the effect of live weight by describing total mass of protein and lipid as a function of body mass. In this way, the

pattern of lipid to protein deposition can be calculated from serially slaughtered pigs. The used method of describing components as a function of the whole body using the function as proposed by Huxley (1932) has proven to be a proper method (Evans and Kempster, 1979; Moughan et al., 1990).

Comparison of treatment HIGH *with treatment* ADLIBITUM

In literature, it has been suggested (Whittemore and Fawcett, 1976) and observed (Campbell, Taverner and Curic, 1983 and 1985; Campbell and Taverner, 1988) that pigs have an intrinsic maximum for daily protein deposition. When the energy intake is higher than needed to support maximal protein deposition, the surplus of energy is used to deposit lipid (Whittemore and Fawcett, 1976; Whittemore, 1983). In order to check the concept on nutrient partitioning below this maximal protein deposition rate, the present study was performed at energy intakes which limited protein deposition at rates below the possible maximal protein deposition rate. To check whether pigs were below their protein deposition capacity, a control group was fed *ad libitum* in each weight range. Before reaching the initial weight of the target weight range, they were treated equally as the HIGH pigs. After switching to ADLIBITUM, these pigs increased their daily feed intake. This resulted in higher daily gains and, in most weight ranges, also in fatter bodies. The aim of the present study was derive whether the HIGH pigs were depositing protein below their maximum. For this reason, in table 6, protein deposition rates of the *ad libitum* fed pigs are also presented. As stated before, statistical comparison of these values is not reliable due to short weight ranges. Results indicate that beyond 45kg live weight, ADLIBITUM fed pigs deposited protein at rates of 200 g/day and higher. The average protein deposition rate for HIGH fed pigs was 144 g/d, It can thus be stated that HIGH pigs were depositing protein well below their maximal protein deposition capacity. Therefore, results of treatment HIGH and LOW can be used to test the hypothesis that the ratio of lipid to protein deposition is constant at these intake levels below maximal protein deposition rate (Whittemore, 1983).

Table 6. Protein deposition rates (g/d) in the four weight ranges of ad libitum fed pigs. Pigs of the preceding slaughter weight, receiving the* HIGH *treatment were used as initial slaughter group.

Target weight range (kg)	25-45	45-65	65-85	85-105
Slaughter weight (kg)	45	65	85	105
	mean sd	mean sd	mean sd	mean sd
Protein deposition rate (g/d)	159 12	198 26	221 36	250 70

Effect of energy intake on tissue deposition

In the present experiment, the amount of feed was varied to vary energy intake. The ration was composed to have energy be the first limiting nutrient for all treatment groups. Thus, levels of amino acids and other essential nutrients can be regarded as non limiting. Amount of energy intake influenced the partitioning of production energy into protein and lipid. The difference of 3.7 MJ DE intake per day between HIGH and LOW resulted in different daily gains (105 g/d), and different body lipid contents, about 3 kg lipid at 105 kg body weight. ARC (1981) estimated the effect of each additional MJ of ME to give an additional 25 grams of daily gain, which is in close agreement to findings in the present

experiment. In the present study, entire male pigs were used. It can be expected that the difference in growth rate between the two treatment levels would have been smaller in other sexes due to a different partitioning of production energy between lipid and protein deposition (Campbell, 1988; De Greef, Verstegen and Campbell, 1992a). The average ratio of lipid to protein deposition was 0.90 for LOW fed pigs and 1.15 for HIGH fed pigs. This is close to the value 1.0 as proposed by Whittemore and Fawcett (1976) and Moughan et al. (1987). The fatter bodies and higher lipid to protein deposition ratios in HIGH fed pigs as compared to LOW fed pigs demonstrates the influence of energy intake on the partitioning of production energy into protein and lipid deposition. This is contrary to the assumption made in most growth models that, below maximal protein deposition rate, there is no effect of degree of restriction on the ratio of lipid to protein deposition (Whittemore and Fawcett, 1976; Moughan et al., 1987; Watt et al., 1987; Pomar et al., 1991).

Effect of live weight on tissue deposition

The lines describing the ratio of lipid to protein deposition rate showed that with increasing body weight this ratio increased (figure 2). Thus, at constant energy available for production, partitioning of production energy changes gradually from protein to lipid with increasing weight. This phenomenon has not been accounted for in the previously mentioned growth models (Whittemore and Fawcett, 1976; Moughan et al., 1987; Watt et al., 1987; Pomar et al., 1991). A validation of one of these growth models (Moughan et al., 1987) also manifested an effect of body weight on partitioning between protein and lipid deposition (De Greef, Verstegen and Kemp, 1992b).

Pigs at high live weights had lower rates of live weight gain as compared to low live weights. The estimated effect of live weight is a decrease of 2 grams of daily gain per kg increase of body weight. Thus, live weight gain decreased with increasing body weight, despite the constant amount of energy available for production. The assumption in this is that maintenance requirement was quantified correctly. When maintenance requirement was underestimated at higher live weights, this may have attributed to the decrease in live weight gain. However, this seems unlikely (ARC, 1981). Production energy calculated from net energy in lipid and in protein and their respective costs of synthesis indicates that available energy was not decreased with live weight. This means that energy allowance above maintenance did not decrease with live weight. The increase in live weight also resulted in a considerable increase in body lipid content for both treatments. Lipid gain gives a lower body gain as compared to protein gain, as protein gain is accompanied by deposition of water and minerals. Thus, at equal or even higher energy deposition, daily gain can decrease due to a change in the composition of gain. Furthermore, at higher live weights, each unit of protein deposition results in a lower body weight gain due to a decreasing ratio of water to protein in accreted tissue at higher live weights (Kotarbinska, 1969; ARC, 1981). This is also illustrated by table 3. The estimates of the b-values were higher than unity. This means that both protein and lipid increased relatively faster than body weight. Thus, other body components (ash and water) increased slower than live weight. However, this change was not very large during the weight range presently studied, but may have attributed to the decrease in live weight gain. In conclusion, it can be stated that a change in partitioning between protein and lipid was a major factor causing the

reduced live weight gain with increasing body weight. Other factors like water deposition and maintenance requirement can be expected to play minor and no role, respectively.

Combined effect of energy intake and body weight

There was no significant interaction between weight range and amount of production energy on any of the parameters tested. This suggests that weight range and amount of production energy exert their effects independently and additionally. Although results did not show a significant interaction between amount of production energy and weight range, they do suggest that the increase of the ratio of lipid to protein deposition with increasing live weight does depend on amount of energy. In figure 2, the difference between the lines describing HIGH and LOW increases with increasing live weight. This increase in ratio of lipid to protein deposition rate in treatment LOW was 0.0031 units per kg increase in live weight and for treatment HIGH the increase was more than twice as much (0.0066). Because these figures are derived from derivates of the describing lines (see MATERIAL AND METHODS), they were not compared statistically. Thus, although not statistically evidenced, results indicate that the difference in ratio between lipid and protein deposition rate between the two energy intake levels increases with body weight. Experiments with higher feed allowances or larger contrasts between treatment groups are needed to further quantify this change.

Implications of present results

Knowledge of the partitioning of production energy into lipid and protein is needed in order to predict performance of pigs as a function of animal characteristics and feed characteristics. In the modelling approach as proposed by Whittemore and Fawcett (1976), the ratio of lipid to protein deposition is assumed to be constant when pigs are depositing protein below their maximum. This principle is used in various modelling exercises (Whittemore, 1983; Moughan et al., 1987, Watt et al., 1987; Pomar et al., 1991). This approach assumes that ratio of lipid to protein deposition rate is independent of energy intake until maximal protein deposition capacity. Whittemore (1983) has proposed various ratios for several types (strains and sexes) of pigs. The present work shows that, below maximal protein deposition, a higher intake of energy results in a higher ratio of lipid to protein. Furthermore, the ratio between lipid and protein deposition increases also with live weight at constant energy available for production. These results indicate that the assumption of constant ratio of lipid to protein deposition rate below maximal protein deposition at least has to be reconsidered. Present results show a positive effect of both energy intake and live weight on the ratio of lipid to protein deposition rate. This is in agreement with results from Black and Griffiths (1975) in growing lambs. A reanalysis of the work of the group of Campbell also revealed an increase in lipid to protein deposition ratio in pigs fed well below their capacity (De Greef et al., 1992a). The ARC (1981) also assumed an increase in the ratio of lipid to protein deposition rate with increasing energy intake. In their approach, a higher capacity to deposit protein coincides with a lower increase in ratio of lipid to protein deposition rate with increasing energy intake. This mechanism will allow pigs with a moderate or low protein deposition capacity to become fatter at any energy intake as compared to pigs of better potential. It also allows pigs which are genetically very lean to increase their tissue accretion rate with each extra amount of energy with a very low increase in the ratio of lipid to protein deposition rate.

This type of characterization is an alternative to characterization of the pig using a minimal ratio of lipid to protein (Whittemore, 1983).

CONCLUSIONS

The present work shows a clear effect of both energy intake and of body weight on the composition of deposited tissue. The ratio between lipid and protein deposition increases with an increase in live weight and with an increase in energy intake. Thus, present work does not support the concept of a constant partitioning between lipid and protein deposition with increasing energy intake below maximum protein deposition. The results challenge a concept widely used in swine growth models. This emphasises the need to study the mechanism of partitioning of energy into differential body tissues in more detail.

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Chapter IV

PERFORMANCE AND BODY COMPOSITION OF FATTENING PIGS OF TWO STRAINS DURING PROTEIN DEFICIENCY AND SUBSEQUENT REALIMENTATION

K.H. DE GREEF, B. KEMP AND M.W.A. VERSTEGEN

SUMMARY

The influence of protein restriction and subsequent realimentation on protein and lipid deposition was studied. Between 28 kg and 65 kg live weight (LW), entire male pigs of two strains (a commercial and a sire strain) were given diets either deficient or adequate in protein content. From 65 to 105 kg LW all pigs were fed a protein adequate ration. Animals were slaughtered and dissected at start, 65 and 105 kg LW. Body composition and deposition rates of protein, lipid, lean and fatty tissue for both the restriction period (28-65 kg LW) and the realimentation period (65-105 kg LW) were calculated. Protein restriction reduced feed intake (28%), live weight gain (60%), and rate of protein (75%) and lipid deposition (15%) between 28 and 65 kg live weight. At 65 kg, restricted animals had twice as much lipid and were 60 days older than controls. During realimentation, previously restricted pigs (compared to controls) had slightly (7%) reduced feed intake and 15% increased weight gain and efficiency. Protein deposition rate beyond 65 kg LW was increased by 13% and ratio of lipid to protein deposition rate was decreased from 1.69 to 1.23. At 105 kg, the previously restricted pigs still were older and fatter than controls, so compensation was not complete. Strains of pigs responded similar to both restriction and realimentation. Dissection at 105 kg LW was not sensitive enough to show the effects revealed by chemical analysis. The experiment revealed that nutritional history may influence the relation between lipid deposition rate and protein deposition rate.

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PERFORMANCE AND BODY COMPOSITION OF FATTENING PIGS OF TWO STRAINS DURING PROTEIN DEFICIENCY AND SUBSEQUENT REALIMENTATION

INTRODUCTION

Many investigations have reported compensatory effects in terms of growth performance, efficiency and lean tissue accretion in pigs after a period of protein undernutrition (Wyllie et al., 1969; Gilster and Wahlstrom, 1973; Zimmerman and Khajarern, 1973; Campbell and Biden, 1978; Hogberg and Zimmerman, 1978; Wahlstrom and Libal, 1983). However, studies on effects of realimentation after protein restriction on deposition rates of protein and lipid in the body are scarce. Relevant factors in compensation are feed intake, maintenance expenditure and deposition of protein and lipid.

The partitioning of energy above maintenance into lipid and protein accretion in growing pigs is thought to have a constant ratio if pigs are moderately below their maximal protein deposition (Whittemore and Fawcett, 1976; Moughan et al., 1987). The question arises whether partitioning of energy between protein and lipid deposition is influenced by previous nutritional manipulation, and whether such a change in partitioning between protein and fat deposition is a possible mechanism of compensation.

The aim of the present study was to investigate the performance of pigs during a period of severe protein deficiency and during the subsequent realimentation. Two strains of pigs were compared. Emphasis was placed on the effects on protein deposition rate and on the relationship between protein and lipid deposition.

MATERIAL AND METHODS

Animals and housing

Forty four entire male pigs, 22 of a synthetic sire strain (strain S1) and 22 of a commercial strain (strain S2) were used. Animal parameters at the start of the experiment are given in table 1. All animals were housed individually in 2×1 m pens on half slatted floors with water available *ad libitum*. Ambient temperature was kept above 18°C. The experiment was performed from March to August 1989.

Table 1. Weight, age and body composition (±sd) of the animals at the start of experiment.

	STRAIN	
	S1	S2
Live weight (kg)	28.1 (2.4)	27.6 (1.9)
Age (d)	67 (4)	68 (6)
Body protein%	16.5 (0.4)	16.8 (0.3)
Body lipid%	9.9 (1.2)	10.6 (1.4)
Carcass lean%	58.9 (0.9)	59.3 (0.7)

Treatments

The experiment had a 2×2 factorial design, using the two pig strains and two feeding strategies. The two feeding strategies were as follows. The control group (treatment C) was fed a protein sufficient ration from 28 to 105 kg live weight (CP/DE ratio declining gradually from 14.0 g CP/MJ digestible energy (DE) at 30 kg live weight to 12.4 g CP/MJ DE at 65 kg, thereafter declining to 11.3 g CP/MJ DE at 105 kg live weight). The protein restricted group (treatment P) was fed a protein deficient diet until 65 kg live weight (CP/DE ratio of 6.4 g CP/MJ DE at 30 kg declining to 5.9 g CP/MJ DE at 65 kg live weight). From 65 kg LW onwards, the P animals received the same ration as the C pigs. Protein allowances in the protein adequate rations were in excess of current estimates of requirement for pigs of similar weight (ARC, 1981).

Control pigs were offered 22 MJ Digestible Energy per day above maintenance requirement (ARC, 1981) at 28 kg live weight, increasing with 1 MJ/day for each kg live weight above 28 kg live weight until 32 kg live weight, after which increasing with 50 kJ for each kg live weight above 32 kg live weight. This feeding level was about the feed intake capacity for the used strains of pigs (De Greef, Knol and Smulders, unpublished results). Protein restricted pigs were fed above feed intake capacity until 65 kg LW. After 65 kg LW, they were fed the same amounts as the Control pigs. Feed residues were collected, weighed and discarded weekly. The rations were adjusted weekly.

The rations were prepared by using two basal diets, a high protein diet and a low protein diet. Both treatment groups received a mixture of both basal diets. The high protein diet contained 25.3% crude protein, 1.47% lysin and had a calculated DE content of 15.8 MJ/kg. The low protein diet contained 7.2% crude protein, 0.34% lysin and a calculated DE content of 14.1 MJ/kg. The basal feeds were mixed prior to feeding to formulate a feed containing the desired protein and energy levels. Feed intake parameters will be presented as standardized to 15 MJ DE/kg feed. Composition of the two basal diets is presented in table 2.

Measurements

At the start of the experiment, four S1 and four S2 animals were slaughtered to determine initial body composition. At 65 kg live weight, 16 animals (4 of each treatment×strain combination) were slaughtered. At 105 kg live weight the remaining 20 animals (5 of each group) were slaughtered. At slaughter, blood was collected, the alimentary tract was emptied and the carcass (including head) was split into two halves. After cooling overnight in a plastic bag, the right carcass half was dissected into trimmed major joints and fat depots according to the Dutch standard dissection method (Bergström and Kroeske, 1968; Kanis, 1988). Three tissue groups were made, *1.* trimmed major joints: 'LEAN', *2.* other carcass parts: 'FAT' and *3.* blood and organs. The tissue material of each animal was frozen at -20°C, and subsequently homogenized using a 45 l cutter[2]. After homogenization, chemical analysis (dry matter, nitrogen, ether extract and ash) was performed in the three tissue groups separately. Chemical composition of the whole empty body was calculated from the chemical composition and weights of the tissue groups. To calculate the

[2] Rohwer, 45 liter, 2 speeds

deposition rates from 28 to 65 kg and from 28 to 105 kg live weight, chemical body composition (CBC) of the initial slaughter group was used to estimate the initial CBC for the other pigs. CBC at 65 kg live weight was used similarly to calculate deposition rates between 65 and 105 kg live weight.

Table 2. Composition of the basal diets.

Ingredient (g/kg)	DIET I	DIET II
Maize	400	426
Toasted Phaseolus beans		56
Peas	100	
Soya bean meal extr.	300	
Cassava	10	369
Cane molasses	30	35
Sugar	5	5
Soya bean oil	57	60
Fish meal	50	
Meat meal	18	
L.threonine	0.25	0.04
L.tryptophan	0.07	0.03
L.lysin 10%		11.53
DL.methionine 10%	15.22	1.14
Salt	0.92	0.88
Vitamins and minerals	13.39	34.91
Analysed composition (g/kg)		
Moisture	108	124
Crude protein	253	72
Ether extract	79	79
Ash	57	60
Crude fiber	27	29
Total lysin	14.7	3.6
Total methionine	4.9	1.1
Total threonine	10.1	2.8
Calculated from table values		
digestibility of protein (%)	0.88	0.65
Digestible Energy (MJ/kg)	15.8	14.1

Variables

The following variables were measured and calculated for each animal from performance characteristics, dissection data and chemical analysis.

FI	daily feed intake (g/day), standardized to 15 MJ/kg feed
ADG	daily live weight gain (g/d)
FCR	feed conversion ratio, FI/ADG
AGE	age at slaughter (days)
LEAN%	carcass lean percentage
LTD	daily lean tissue deposition rate (g/d)
FTD	fatty tissue deposition (g/d)
FTD/LTD	ratio between FTD and LTD, FTD/LTD
PROTEIN%	empty body protein percentage
LIPID%	empty body lipid percentage
PD	protein deposition rate (g/d)
LD	lipid deposition rate (g/d)
LD/PD	ratio between LD and PD, LD/PD

Statistics

The variables were analyzed separately per weight range, according to the following model (using SAS-GLM; SAS, 1985)

$$\underline{Y}_{ijk} = \mu + \text{Treatment}_i + \text{Strain}_j + (\text{Treatment} \times \text{Strain})_{ij} + \underline{e}_{ijk} \quad (1)$$

in which:

Y = variable
μ = overall mean
e = residual error.

RESULTS

Restriction phase

Treatment effects during restriction phase.

Performance and body composition parameters for the restriction period are presented in table 3. In the restriction period, the protein deficient group had on average a 28% decrease in daily feed intake compared to the control group ($P<0.001$). Live weight gain was reduced by 60% from 1057 to 424 g/d ($P<0.001$) and feed conversion ratio was increased by 82% from 1.85 to 3.38 ($P<0.001$).

Table 3. Performance and body composition in the restriction phase, 28-65 kg live weight.

TREATMENT	C	C	P	P		SIGNIFICANCE		
STRAIN	S1	S2	S1	S2	SEM	Treatment	Strain	TxS
Feed intake (g/d)	1937	1952	1396	1415	73	***		
Daily gain (g/d)	1088	1025	417	430	45	***		
FCR	1.79	1.91	3.40	3.35	0.15	***		
LTD (g/d)	518	448	171	166	24	***		
FTD (g/d)	358	349	169	189	15	***		
FTD/LTD	0.70	0.78	0.98	1.16	0.04	***	**	
Protein dep. (g/d)	187	153	42	43	8	***	0.074	0.057
Lipid dep. (g/d)	193	198	150	181	17	0.089		
LD/PD	1.05	1.30	3.61	4.23	0.17	***	*	
At slaughter (65 kg LW):								
Age (d)	104	103	164	164	6.8	***		
Lean%	58.9	57.5	53.6	51.6	0.7	***	*	
Protein%	17.3	16.3	13.7	13.4	0.2	***	*	
Lipid%	14.8	16.3	27.8	30.6	0.8	***	*	

***:P<0.001; **:P<0.01; *:P<0.05

Chemical analysis of the body showed that at 65 kg live weight the protein content of empty body was decreased (13.5% vs. 16.8%, $P<0.001$) and the empty body lipid content was increased (29.2% vs. 15.5%, $P<0.001$) compared to the controls. The lower daily live weight gain in the protein restricted group was reflected in lower rates of gain in lean tissue, fatty tissue, and also in protein and lipid. Lean tissue deposition rate was decreased more than fat deposition rate (reductions were 65% and 49%). This resulted in an increase in fat to lean ratio from 0.74 in C animals to 1.07 in P animals ($P<0.001$). Protein deposition in restricted pigs was decreased by 128 g/d, a reduction of 75% on average ($P<0.001$), lipid deposition was decreased by 10-20% ($P=0.09$) in restricted pigs compared

to controls. The ratio of lipid to protein deposition was much higher ($P<0.001$) for the P animals than for the control animals (3.92 and 1.18 respectively).

Strain effects during restriction phase.
Table 3 shows that there were no significant effects of strain on feed intake, daily gain or feed conversion ratio during the restriction period. Interactions of treatment × strain on feed intake, daily gain or feed conversion ratio were not found in this period. In the S2 animals, lipid content was higher on both treatments ($P<0.05$) and protein content was lower as compared to S1 pigs ($P<0.05$). The ratio of lipid to protein deposition was higher for the S2 than for the S1 pigs ($P<0.05$). Similarly, the ratio between fatty tissue gain and lean tissue gain was significantly higher for the S2 strain than for the S1 strain ($P<0.01$). There tended to be a strain effect ($P=0.07$) and an interaction between treatment and strain ($P=0.06$) on protein deposition rate. On the C treatment, S1 animals deposited daily on average 28 grams of protein more than S2 animals did. Both strains deposited similar amounts of protein on the P treatment. There were no further interactions of treatment × strain during the restriction period.

Realimentation phase
Treatment effects during realimentation phase.
Performance and body parameters after realimentation are presented in table 4. During realimentation, pigs previously given the protein deficient diet grew 165 g/d (15%) faster than controls ($P<0.001$) with a 7% lower daily feed intake ($P<0.001$). Feed conversion ratio was 19% lower in the P pigs compared to C pigs ($P<0.001$).

Table 4. Performance and body composition in the realimentation phase, 65-105 kg live weight.

TREATMENT	C	C	P	P		SIGNIFICANCE		
STRAIN	S1	S2	S1	S2	SEM	Treatment	Strain	TxS
Feed intake (g/d)	2500	2495	2266	2384	45	***		
Daily gain (g/d)	1105	1158	1305	1288	41	***		
FCR	2.27	2.16	1.74	1.86	0.07	***		0.097
LTD (g/d)	490	501	589	576	23	***		
FTD (g/d)	457	479	502	507	33			
FTD/LTD	0.94	0.98	0.86	0.88	0.09			
Protein dep. (g/d)	171	192	194	216	9	**	*	
Lipid dep (g/d)	304	299	247	251	34	0.095		
LD/PD	1.77	1.60	1.29	1.17	0.20	*		
At slaughter (105 kg LW):								
Age (d)	138	138	194	183	8	***		
Lean%	56.1	54.9	53.9	52.3	1.0	**		
Protein%	16.8	16.5	14.7	14.7	0.3	***		
Lipid%	20.0	20.4	25.2	26.0	1.1	***		

***:P<0.001; **:P<0.01; *:P<0.05

Both the rates of lean and fatty tissue deposition were higher for the pigs previously given the low protein diets compared to the control pigs (differences were 17.5%, $P<0.001$ and 17.7%, n.s., respectively). The ratio between the deposition rates of these two tissue groups did not differ significantly between the C and P groups ($P>0.1$). Protein deposition in the realimentation phase was 182 g/d in the control group and 205 g/d in the realimented animals ($P<0.01$). Lipid deposition tended to be lower in the P animals as compared to

the controls (P: 249 g/d, C: 301 g/d, $P<0.10$). The ratio of lipid to protein deposition was reduced from 1.69 to 1.23 ($P<0.05$).

Strain effects during realimentation phase.
There were no significant effects of strain on FI, ADG, FCR, LTD, FTD and FTD/LTD during the realimentation period (table 4). Protein deposition rate after realimentation was on average 204 g/d for the S2, and 182 g/d for the S1 animals (strain effect, $P<0.05$). There was no significant effect of strain on rate of lipid deposition and on ratio of lipid to protein deposition rate. At 105 kg, chemical body compositions of both strains were similar (table 4).

There was a tendency for a treatment×strain interaction ($P=0.10$) for feed conversion ratio after 65 kg. On the Control treatment, the S2 animals had a lower feed conversion ratio. In the compensating group, however, the S1-animals had a lower feed conversion ratio. Other parameters did not show interaction between treatment and strain during realimentation.

Whole experimental period
Table 5 shows performance parameters for both weight ranges combined. Combining performance parameters both weight ranges shows that the nutritional treatments differed significantly for all parameters tested. Overall performance of the C pigs was superior to the overall performance of the P pigs.

Table 5. Performance and body composition for the whole experimental period, 28-105 kg live weight

TREATMENT	C	C	P	P		SIGNIFICANCE		
STRAIN	S1	S2	S1	S2	SEM	Treatment	Strain	T×S
Feed intake (g/d)	2207	2216	1630	1698	71	***		
Daily gain (g/d)	1072	1108	640	673	36	***		
FCR	2.06	2.00	2.58	2.53	0.06	***		
LTD (g/d)	492	482	278	280	18	***		
FTD (g/d)	401	421	253	280	17	***		
FTD/LTD	0.82	0.88	0.92	1.00	0.04	*	0.080	
Protein dep. (g/d)	174	175	86	91	6	***		
Lipid dep (g/d)	244	254	185	202	15	**		
LD/PD	1.41	1.46	2.20	2.25	0.12	***		

***:P<0.001; **:P<0.01; *:P<0.05

Except for a tendency for a strain effect on the ratio between fatty tissue and lean tissue deposition, there was no effect of strain of pig on overall performance.

DISCUSSION

Results in the restriction period showed that protein restriction reduced feed intake. The reduced feed intake was maintained after realimentation until about 85 kg live weight. After this, the daily feed intake resembled that of the control group. Wyllie et al. (1969) also observed a decreased feed intake during realimentation after a period of protein deficiency. In the present work, improved performance can not be explained by the

mechanism of compensatory feed intake as mentioned by Reid and White (1977). The present work therefore shows that compensatory effects of previous restriction on live weight gain and protein deposition may occur without increased feed intake. Compensation is largely associated with enhanced protein deposition and a consequent decreased lipid deposition.

Increased growth rate between 65 and 105 kg live weight was not sufficient to fully compensate for the decreased growth rate before 65 kg live weight. For all parameters tested, degree of compensation was small as compared to the degree of previous restriction. Therefore, P pigs were considerably older (about 50 days) at 105 kg live weight than C pigs were. Similarly, the increased protein deposition rate and the reduced lipid deposition rate did not compensate fully for the increased fatness caused by the previous protein restriction of the body at 65 kg. At 105 kg live weight, previously restricted pigs still were fatter than control pigs. This also implies a non-complete compensation. Most other studies (Gilster and Wahlstrom, 1973; Zimmerman and Khajarern, 1973; Campbell and Biden, 1978; Hogberg and Zimmerman, 1978; Wahlstrom and Libal, 1983) also found incomplete compensation. Wyllie et al. (1969), on the other hand, found that previously protein restricted pigs (restricted in protein intake until 24 kg LW) were leaner at the end of the realimentation period (at 92 kg LW) than controls. In the present work, the restriction was severe as compared to the cited literature, and the pig had a relative short period for compensation. Literature is not clear in the influence of degree of restriction and the influence of live weight on the degree of compensation.

The increase in protein deposition rate and decrease in lipid deposition rate after realimentation has two important implications for describing the response of the animal to nutrition. The first is that the theory suggesting that protein deposition capacity (Whittemore and Fawcett, 1976; Moughan et al., 1987) is fully expressed under normal *ad libitum* intake does not hold for the nutritional situation described in this paper. This means that the suggested maximum protein gain capacity (Whittemore and Fawcett, 1976; Moughan et al., 1987) is not as a constant animal factor as thought before. The severe nutritional constraints imposed on the pigs have to be taken into account in this, and it is likely that in less severe situations, effects on protein deposition are smaller. The second implication is that the fixed relation between lipid deposition and fat deposition (Whittemore and Fawcett, 1976; Whittemore, 1983; Moughan et al., 1987) does not hold for this nutritional situation. This means that the composition of deposited tissue can be altered by nutritional history. Although fat deposition was reduced in the compensating animals, they still deposited substantial amounts of lipid (249 g/d on average).

The change in composition of growth may be derived from the theory that animals grow to fulfil their genetic capacity (Parks, 1982). Thus, if animals are restricted to reach this capacity, they may try to compensate this if environment and nutrition allow it. After the restriction period, the animals had relatively low body protein contents and relatively high body fat contents. By altering protein and lipid deposition rate, the animals manipulated their tissue deposition to create a body which resembles their intrinsic ideal. This implies an influence of the actual body composition on the partitioning of energy between protein and lipid deposition. So far, this mechanism has not been incorporated in production prediction systems like swine growth models (Whittemore and Fawcett, 1976; Whittemore,

1983; Moughan et al., 1987; Black et al., 1986). The present work illustrates the need to study this mechanism in order to be able to predict effects.

The influence of body composition on partitioning of production energy also has consequences for the 'protein deposition capacity' of pigs. Capacity is defined here as the protein deposition rate at ideal nutritional circumstances (Whittemore et al., 1988). An animal which is relatively fat (due to nutritional manipulation) can, compared to a less fat animal, put a higher preference towards protein deposition, and a lower preference to lipid deposition. This means that at a certain feed intake, this animal can deposit more protein per day. Therefore, at this definition of capacity, this animal temporarily has a higher protein deposition capacity. The fact that the previous restriction decreased feed intake will reduce the total effect on energy retention in the pig. Compensation is merely a repartitioning of feed energy for production.

The two strains of pigs in our study responded similarly to realimentation. Therefore, effects of type of pig in response to restriction and realimentation and in degree of compensation as found by Hogberg and Zimmerman (1978) were not found in the present work. It may be that our strains of pig did not differ enough in leanness to respond differently to restriction and subsequent realimentation. The two strains used by Hogberg and Zimmerman (1978) differed more than those used in the present work. Hogberg and Zimmerman (1978) explained the lack of compensation in their lean strain of pig with the hypothesis that the protein restriction (10% CP in the ration) had been too severe for that type of pig. In the present study, however, we used an even leaner strain (based on body lipid content in the control pigs). Moreover, protein restriction was more severe (about 9% CP in the ration) in our pigs than in the lean strain of Hogberg and Zimmerman (1978). This means that a severe protein restriction does not prevent the pigs from compensating.

S2 pigs had a higher ratio of fat to lean deposition rate and of lipid to protein deposition rate in the restriction phase than S1 animals had. This resulted in fatter bodies of S2 at 65 kg live weight (table 3). After 65 kg their LD/PD ratio was lower. This resulted in similar body compositions for the two strains at 105 kg live weight. The overall performance data were similar, although the two strains of pigs apparently had different pathways to reach this performance. Thus, the partitioning of energy and nutrients into protein and lipid deposition changed with live weight and this change was different for the two strains of pigs. This finding may have consequences when designing feeding strategies for different types of pigs. Additional to this, selection for leanness of pigs which takes only a part of the fattening period may make a false discrimination between strains of pigs. The used two strains, which are essentially comparable in their overall performance, would have been qualified as being different when testing only until 65 kg live weight, or after 65 kg LW.

Dissection results showed no significant effects of treatment on the ratio of fat to lean tissue deposition rate after realimentation (0.87 and 0.96 in the compensating and control group, respectively). Chemical deposition rates, however, showed a highly decreased lipid to protein ratio (1.23 vs. 1.69). This change in chemical composition agrees with the highly increased efficiency of the P animals compared to the C animals in the realimentation period. Table 5 shows that during realimentation, the ratio of lipid to protein in the

deposited lean was 0.40 and 0.67 for P and C animals, respectively. Therefore, in the compensating group, a smaller part of the deposited 'lean' consisted of lipid (intra and/or intermuscular fat) than in the control group. This is an indication that dissection is not very sensitive to discriminate in body composition between treatments. Wyllie et al. (1969) also observed effects of compensation on chemical deposition which were not detected by dissection. To have a better estimate of muscle deposition rate, as compared to the technique used in this study, a full anatomical dissection can be advised. However, if the increased lipid in the 'lean' consisted of a substantial amount of intramuscular lipid, then even a full anatomical dissection would still discriminate less than chemical analysis. Therefore, for measuring certain effects, it may be necessary to assess the animals' chemical body composition. To be able to compare deposition rates, comparative slaughter technique is required.

Table 6. Ratio (±sd) between the deposition rate of lipid and protein in the whole empty body and in the three tissue groups after realimentation.

	TREATMENT		
	C	P	Sign.
Whole body	1.69 (0.42)	1.23 (0.37)	*
Lean tissue	0.67 (0.15)	0.40 (0.14)	***
Fatty tissue	3.81 (1.01)	3.34 (1.06)	
Non-carcass	0.89 (0.45)	0.42 (0.25)	*

***:P<0.001; *:P<0.05

From the results of this study we conclude that the nutritional history of the pig is an important topic. It may well be that the effects found in this study may be extrapolated towards less severe nutritional treatments. This means that in prediction of the partitioning of energy towards different types of tissue deposition, nutritional history or actual body composition has to be taken into account. Especially dependency of growth composition on previous nutrition is relevant for predicting responses to nutritional manipulation. The relation between actual body composition and partitioning of production energy is unclear and deserves further study.

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Chapter V

PARTITIONING OF PROTEIN AND LIPID DEPOSITION IN THE BODY OF GROWING PIGS

K.H. DE GREEF AND M.W.A. VERSTEGEN

SUMMARY

Two amounts of energy intake (12.6 and 16.3 MJ DE/day, respectively) above maintenance requirement were given to entire male pigs from 25 to 105 kg body weight. Slaughter, dissection and chemical analysis in three body fractions on 25, 65 and 105 kg live weight allowed calculation of tissue deposition and tissue partitioning in two weight ranges. On average, 57% of total protein deposition was deposited into the LEAN fraction. Moreover, 68% of total lipid deposition was deposited into the non-lean carcass parts on average. An increase in energy intake resulted in an increase in both lipid and protein deposition. Of this *extra* protein deposition and *extra* lipid deposition, 42% and 75% respectively were directed to the non-lean carcass parts in the live weight range 25-65 kg. Above 65 kg live weight, these respective percentages were 71% and 82% on average. Thus, despite the relatively low intake levels, the major part of extra tissue deposition was deposited into non lean carcass tissue. The effect of a 40 kg increase in body weight on protein and lipid deposition rates was small as compared to the effect of an extra 250 grams of feed. Thus, for the used feeding regimen, partitioning of protein within the body was mainly effected by nutrition, and not by body weight. It was concluded that a nutrition induced increase in lean tissue deposition rate will be coincided with a substantial increase in body fatness. This is due to an increase in lipid deposition accompanying an increase in protein deposition. Furthermore, an increase in protein deposition enhanced the partitioning of protein to tissues other than lean.

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PARTITIONING OF PROTEIN AND LIPID DEPOSITION IN THE BODY OF GROWING PIGS

INTRODUCTION

Several studies have focused on the chemical and anatomical composition of the pigs body (a.o. Doornenbal, 1971 and 1972; Walstra, 1980; Fortin, 1982; Süsenbeth and Keitel, 1988). Knowledge of body conformation, especially combined with studying the site of tissue deposition will help to understand changes in body composition with increasing body weight. Changes in the distribution of growth components were predominantly associated with changes in live weight. Little emphasis, however, has been given to the effects of nutrition on the distribution of chemical components into tissue groups within the body. Furthermore, not much effort has yet been made to differentiate between the effect of live weight and the effect of feed intake.

For this reason, an experiment was performed to study the effect of body weight and of feed intake on amount and site of tissue deposition. Energy allowances were designed to be independent of live weight. In this way, the effect of live weight and the effect of energy intake on the site of tissue deposition could be compared unconstrained. In the present study, special emphasis will be put on the partitioning of *extra* tissue deposition (calculated from experimental contrasts in energy intake).

MATERIAL AND METHODS

The experiment was performed in a 2×2 factorial design. Two energy intake levels were studied in two live weight ranges. Entire male pigs of a commercial synthetic cross were fed a constant amount of production energy, either 12.6 or 16.3 MJ DE above maintenance energy requirement. In this way, amount of energy available for production was taken to be independent of body weight. The maintenance energy requirement (MJ ME/day) was assumed to be 719×Body weigh$^{*0.63}$ (ARC, 1981). ME was converted into DE by multiplying DE with the factor 0.96 (ARC, 1981). Other nutrients than digestible energy in the ration were above requirements (a.o. ARC, 1981).

At 25 kg live weight, an initial slaughter group (4 pigs) and at 65 and 105 kg live weight, also 4 pigs of each treatment group were slaughtered, dissected and chemically analyzed. Dissection into trimmed major joints was performed according to the Dutch standard dissection technique (Bergström and Kroeske, 1968; Walstra, 1980). Chemical analysis (dry matter, nitrogen and lipid) was performed as described by De Greef et al. (1992b) in three tissue groups: (*i*) trimmed major joints (LEAN), (*ii*) other carcass parts (DEPOT) and (*iii*) blood and organs (ORGANS). LEAN was defined as the weight of trimmed ham, trimmed shoulder, trimmed loin and meat scraps, a group of miscellaneous muscles (Walstra, 1980). The other carcass parts (DEPOT) consisted of back fat, belly, fat trimmed from ham and shoulder, first four ribs, lower jaw fat, flare fat, head, feet, tail and a group of fatty tissue depots originating from various locations (Walstra, 1980). The tissue group ORGANS consisted of blood, emptied abdominal contents, contents of the thorax, tongue, kidneys

and brains. Composition of the whole body was calculated from the composition and weights of the three separate tissue groups.

Chemical composition of pigs slaughtered at 25 kg live weight was used to estimate initial chemical composition of pigs slaughtered at higher live weights. Similarly, data on chemical composition of pigs slaughtered at 65 kg live weight were used as initial data for pigs receiving the same treatment, but slaughtered at 105 kg live weight. Deposition rates of protein (PD, g/d) and lipid (LD, g/d) between 25 and 65 kg live weight and between 65 and 105 kg live weight were calculated in the three tissue groups separately and in the whole body. The individually housed pigs were fed twice daily and weighed twice a week. Average daily gain (g/d) was calculated from initial live weight and the live weight at the day before slaughter. Water was available *ad libitum*. Back fat thickness was measured on 8 points (4 on each side) at the back at the day before slaughter. The pigs studied in the present paper were involved in a larger study on the effect of body weight and energy intake on the relation between lipid and protein deposition. Details of feed composition, feeding and chemical analysis have been described previously (De Greef et al., 1992b).

Average daily gain (g/d), parameters measured at slaughter weight (back fat thickness, carcass lean percentage, body protein and lipid content (%)) and deposition rates of protein and lipid (g/d) were analyzed using the model

$$\underline{Y}_{ijk} = \mu + \text{Production Energy}_i + \text{Weight Range}_j + (PE \times WR)_{ij} + \underline{e}_{ijk}$$

in which:

μ	= overall mean
Production Energy$_i$ (PE)	= DE intake above maintenance energy requirement (i = 12.6, 16.3 MJ/d)
Weight Range$_j$ (WR)	= weight range in which parameters are measured (j = 25-65kg, 65-105kg)
$(PE \times WR)_{ij}$	= interaction between Production Energy and Weight Range
e_{ijk}	= residual error.

When the interaction between Production Energy and Weight Range was not significant ($P>0.10$), data were reanalysed excluding the interaction PE×WR. Due to illness and refusal of feed, one pig from treatment HIGH, aimed to be slaughtered at 105 kg was excluded from the calculations.

RESULTS

Performance and body composition

In table 1, performance and body composition of the experimental groups is presented. An increase in the amount of production energy increased average daily gain ($P<0.01$) and body lipid percentage ($P<0.01$). Body protein percentage and carcass lean percentage decreased with increasing intake of production energy. There was a tendency for body protein percentage to increase and for daily gain to decrease with increasing live weight ($P<0.10$). Body lipid content increased with live weight ($P<0.001$). This increase in lipid content with increasing live weight tended to be higher at the HIGH amount of production energy compared to the LOW energy intake (PE×WR interaction, $P<0.10$).

Protein and lipid deposition

Table 2 shows deposition rates of protein and lipid in the whole body and in the three separate tissue groups. There were no significant interactions between energy intake and body weight. There was a significant effect of energy intake on both protein and lipid deposition rate. An increase in energy intake resulted in an increase in whole body protein deposition rate ($P<0.01$). This was reflected in protein deposition rate in the DEPOT ($P<0.001$) and also somewhat in the ORGANS ($P<0.10$), but not significantly in the LEAN fraction ($P>0.10$). The HIGH energy intake also resulted in higher lipid deposition rates in the whole body ($P<0.001$), LEAN ($P<0.05$), DEPOT ($P<0.01$) and ORGANS ($P<0.01$), as compared to the LOW energy intake.

Table 2 further shows the effect of live weight on protein and lipid deposition. While receiving a constant amount of digestible energy for production, protein deposition rate in the whole body tended to be lower between 65 and 105 kg live weight as compared to 25-65 kg live weight. In the tissue groups, this decrease with live weight was only significant in the ORGAN fraction ($P<0.001$), but not in LEAN and DEPOT. The contrast between treatment HIGH and LOW in whole body protein deposition was comparable for both weight ranges (HIGH = 1.15×LOW and HIGH = 1.18×LOW, respectively). Body lipid deposition rate was increased above 65 kg live weight as compared to below 65 kg ($P<0.05$). This effect of weight range on lipid deposition was significantly reflected in the tissue group LEAN ($P<0.01$) and showed a tendency in the DEPOT tissue ($P<0.10$).

Furthermore, in table 2, the effects of energy intake (HIGH - LOW) and weight range (65-105kg - 25-65kg live weight) are given to allow separate comparison of the magnitude of the effects of energy intake and weight range on protein and lipid deposition. The 3.7 MJ increase in energy intake increased protein deposition by an average 21 g/d. The increase in live weight of 40 kg affected protein deposition rate by an average 13 g/d. For lipid deposition rates, respective figures were 57 and 31 g/d. This shows that the effect of energy intake on deposition rates of protein and lipid was larger than the effect of weight range.

Partitioning of total protein deposition and total lipid deposition.

In table 3, deposition rates of protein and lipid in the separate tissue groups are expressed as a percentage of whole body protein deposition and of whole body lipid deposition, respectively. The percentage of total protein deposition which is deposited into the LEAN decreased by 4% with increasing energy intake ($P<0.05$), but increased an average 3% with increasing body weight ($P<0.001$). The percentage of protein deposited into the DEPOT fraction increased with energy intake (29% and 33% for LOW and HIGH, respectively, $P<0.01$). The percentage of protein deposited into DEPOT was constant (29% on average) over the live weight ranges for the LOW fed pigs. On the other hand, for the HIGH fed pigs this percentage increased from 31 to 35% with live weight (PE×WR interaction, $P<0.10$). The percentage of lipid which was deposited into the DEPOT tissue tended to be increased with energy intake ($P<0.10$). Weight range had no significant effect on the partitioning of lipid between the body fractions LEAN, DEPOT and ORGANS.

Table 1. Performance and body composition (± sd) for the treatment groups.

Weight range		25-65 kg		65-105 kg		Significance		
Slaughter weight		65 kg		105 kg				
		LOW	HIGH	LOW	HIGH	PE	WR	PE×WR
Daily gain	g/d	758 (29)	904 (71)	707 (84)	829 (49)	**	t	.
Carcass lean	%	60.9 (1.7)	58.5 (0.3)	60.9 (1.3)	56.3 (2.3)	***		.
Body protein	%	17.5 (0.2)	17.0 (0.1)	17.7 (0.2)	17.5 (0.6)	*	t	.
Body lipid	%	13.5 (0.7)	14.6 (1.1)	15.5 (1.2)	19.1 (2.3)	**	***	t
Back fat	mm	8.7 (0.9)	8.9 (0.4)	10.2 (1.3)	12.8 (2.9)	*	**	.

Significance: ***: P<0.001; **: P<0.01; *: P<0.05; t:P<0.10
.: not significant, interaction excluded from the model

Table 2. Protein and lipid deposition rates (±sd) in the body and in the three tissue groups. The contrast between either HIGH and LOW or between the first and the second weight range is presented as estimated effect (g/d).

		BODY	LEAN	DEPOT	ORGANS
Treatment group		**PROTEIN DEPOSITION (g/d)**			
LOW	25- 65 kg	133 (6)	76 (2)	39 (4)	18 (2)
HIGH	25- 65 kg	153 (10)	84 (6)	48 (3)	22 (2)
LOW	65-105 kg	120 (16)	73 (12)	35 (6)	12 (2)
HIGH	65-105 kg	141 (18)	79 (11)	50 (6)	12 (2)
Significance					
Production Energy		**	0.15	***	t
Weight Range		t	0.36	0.62	***
PE*WR		.	.	.	.
Estimated effect (g/d)					
Production Energy		21	[6]	12	3
Weight Range		-13	[-4]	[-1]	-8
Treatment group		**LIPID DEPOSITION (g/d)**			
LOW	25-65 kg	112 (8)	30 (2)	75 (6)	7 (1)
HIGH	25-65 kg	149 (12)	36 (5)	104 (9)	10 (1)
LOW	65-105 kg	124 (18)	37 (4)	80 (18)	7 (1)
HIGH	65-105 kg	202 (48)	46 (10)	144 (43)	12 (4)
Significance					
Production Energy		***	*	**	**
Weight Range		*	**	t	0.25
PE*WR		.	.	.	.
Estimated effect (g/d)					
Production Energy		57	8	45	4
Weight Range		31	9	21	[1]

Significance: ***: P<0.001; **: P<0.01; *: P<0.05; t:P<0.10
.: not significant, interaction excluded from the model
Estimated effects: [ ] represent estimates which are not significantly different from 0

Table 3. Protein and lipid deposition rates (±sd) in the whole body and in the three tissue groups, relative to deposition rate in the whole body. The contrast between either HIGH and LOW or between the first and the second weight range are presented as estimated effect (%).

		BODY	LEAN	DEPOT	ORGANS
Treatment group		**RELATIVE PROTEIN DEPOSITION (%)**			
LOW	25- 65 kg	100	57 (3)	29 (2)	13 (1)
HIGH	25- 65 kg	100	55 (0)	31 (1)	14 (1)
LOW	65-105 kg	100	61 (2)	29 (3)	10 (2)
HIGH	65-105 kg	100	56 (1)	35 (2)	9 (1)
Significance					
Production Energy			**	*	0.89
Weight Range			*	0.20	***
PE*WR			.	t	.
Estimated effect (%)					
Production Energy			-4	4[1]	[0.1]
Weight Range			3	[2][1]	-4
Treatment group		**RELATIVE LIPID DEPOSITION (%)**			
LOW	25-65 kg	100	27 (2)	68 (2)	6 (1)
HIGH	25-65 kg	100	24 (2)	69 (2)	7 (1)
LOW	65-105 kg	100	30 (6)	64 (6)	6 (0)
HIGH	65-105 kg	100	24 (6)	71 (6)	5 (0)
Significance					
Production Energy			t	t	0.54
Weight Range			0.43	0.57	0.31
PE*WR			.	.	.
Estimated effect (%)					
Production Energy			-4	4	[0.3]
Weight Range			[2]	[-1]	[-0.5]

Significance: ***: P<0.001; **: P< 0.01; *: P<0.05; t: P<0.10
.: not signifant, interaction excluded from the model.
Estimated effect: [ ] represent estimates which are not significantly different from 0
[1] biased estimate, due to the significant interaction between Production Energy and Weight Range

Distribution of extra protein deposition and of extra lipid deposition.

The effect of dietary treatment on the distribution of lipid and protein deposition between tissue groups can be studied from the difference between the HIGH and the LOW energy intake. This is presented in table 4. The difference between HIGH and LOW in protein and lipid deposition will be referred to as *extra* protein deposition and *extra* lipid deposition, respectively. In order to illustrate the difference in partitioning of *total* and *extra* protein deposition, figure 1 is given. *Total* deposition refers to the total amount of protein deposited (137 g/d on average), as presented in table 2. *Extra* deposition refers to the extra protein deposition (on average 21 g/d) caused by the 3.7 MJ difference in energy intake, as presented in table 4.

In weight range I (25-65 kg live weight), 37% of the *extra* protein deposition (HIGH compared to LOW) was deposited in the LEAN fraction. In the second weight range (65-105 kg live weight), this decreased to 26% (table 4). The percentage of *extra* protein deposition which was deposited into the DEPOT increased from 42% to 71% with increasing body weight. In weight range I and II, respectively 75% and 82% of the *extra* lipid deposition (difference between HIGH and LOW) was deposited in the DEPOT tissue group. The percentage of *extra* protein and *extra* lipid deposited into the other two tissue groups LEAN and ORGANS decreased with an increase in live weight.

***Table 4. Distribution of extra protein gain and extra lipid gain, calculated from the difference in deposition rates between the HIGH and LOW energy intake*[1].**

	contrast	weight range	BODY	LEAN	DEPOT	ORGANS
			Difference between HIGH and LOW (g/d)			
Protein deposition	HIGH-LOW	25- 65 kg	20	7	8	4
	HIGH-LOW	65-105 kg	22	6	15	1
Lipid deposition	HIGH-LOW	25- 65 kg	38	6	28	3
	HIGH-LOW	65-105 kg	79	10	64	4
			Relative to the whole body (%)			
Protein deposition	HIGH-LOW	25- 65 kg	100	37	42	21
	HIGH-LOW	65-105 kg	100	26	71	4
Lipid deposition	HIGH-LOW	25- 65 kg	100	16	75	9
	HIGH-LOW	65-105 kg	100	12	82	5

[1]Deposition rates in fractions do not always add up to whole body values due to rounding of values.

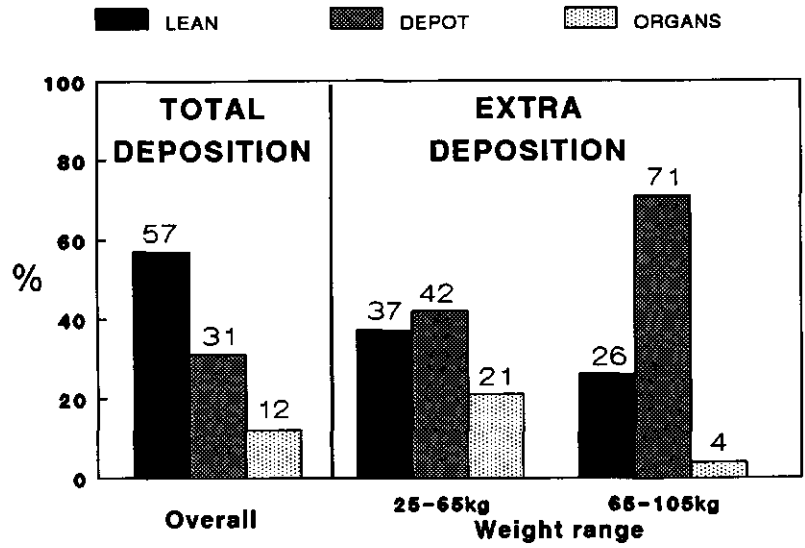

Figure 1. The partitioning of total and extra protein deposition between the three tissue groups.

DISCUSSION

The present experiment was performed with entire male pigs at a relatively low nutritional plane. Highest average protein deposition rates amounted to about 150 g/d, whereas the protein deposition capacity of this type of pigs exceeds 200 grams per day (De Greef et al., 1992a, De Greef et al., 1992b). Thus, pigs were fed well below their protein deposition capacity. The low energy intake was chosen in order to be able to compare performance at two weight ranges. The 22 MJ DE intake (16.3 MJ + maintenance requirement) which was offered at 25 kg live weight to the pigs receiving the HIGH treatment is close to the upper limit of appetite at this live weight for this type of pigs (De Greef et al., unpublished results). ARC (1981) and NRC (1987) propose even lower energy intake capacities at this

live weight. When studying partitioning of nutrients at higher body weights only, effects of higher energy intakes can be studied. Despite the relatively low intake and the small contrast between the treatments, the treatment groups showed very distinct differences in performance and body composition. Especially body lipid content differed clearly between treatments. It is interesting to note that, even at these relatively low intakes, only little more than half of the protein (57%) was deposited into the body parts which are of most commercial relevance (lean tissue). Similarly, an average 68% of total lipid deposition was deposited into the DEPOT fraction.

Partitioning of extra protein and extra lipid deposition
Below 65 kg, 37% of the *extra* protein (calculated from the difference between HIGH and LOW) was distributed into LEAN. This percentage decreased with body weight. In both weight ranges, the partitioning of *extra* protein into LEAN was lower than partitioning of *total* protein deposition into this tissue group. This indicates that composition of *extra* deposited tissue affected body composition in both weight ranges, especially in the second weight range. In weight range II, the major part of the *extra* tissue deposition (both protein and lipid) was deposited into the DEPOT tissue. From these observations, it can be concluded that the conformation of the body changed faster in the second weight range than below 65 kg live weight.

Comparison of Energy effect and Weight effect
The difference between the two feed intake levels was relatively small (about 250 grams of feed). However, in virtually all parameters tested, the effect of Production Energy was larger than the effect of Weight Range. This means that the effect of live weight on partitioning of tissue is relatively small compared to the effect of energy intake. This illustrates the importance of nutrition on body composition. Many studies on body formation have neglected the effects of nutrition, or undervaluated it by only comparing some degree of restriction with *ad libitum* feeding (e.g. Doornenbal, 1971 and 1972; Walstra, 1980; Süsenbeth and Keitel, 1988). Furthermore, in most studies, energy intake increased with body weight. Present work shows that observed patterns in the development of body formation in those studies may then as well be attributed to nutrition as to body weight. The fact that, in literature, energy intake and body weight nearly always are confounded with each other makes it impossible to differentiate between these two effects in those data sets.

The classical view on growth (Hammond, 1932; McMeekan, 1941) implies that there is a certain rhythm in the development of the body. It was suggested that accretion of muscle precedes that of fatty tissue. Furthermore, it was assumed that an increase in nutrient intake does not change this rhythm, but increases this rate of development. Present study shows that there is indeed some priority for protein deposition, but that, even at the low energy intake, there is a substantial fat deposition. Thus, fat deposition and protein deposition are both regulated and there is some partitioning mechanism for this within the body. This mechanism increases the priority for lipid deposition relative to protein deposition with increasing live weight. Because *extra* energy intake is converted into lipid to a major extent shows that the priority depends on energy intake. This phenomenon still resembles the concept presented by the Cambridge school, but also indicates that

formation of depot tissue and accretion of muscle are not two distinct development phases, but occur rather simultaneously.

Static relations between chemical and physical body tissues
Several sources quantified relations between chemical and physical body composition (Whittemore, 1983; Rook et al., 1987; Süsenbeth and Keitel, 1988). From these relations, one can calculate chemical body composition from physical body composition or vice versa, which is often required in growth models (Rook et al., 1987). From studies on the composition of the *deposited* tissue (dynamic relations, as presented in the present work), the nutritional effects on static relations (between chemical and physical masses) can be assessed. However, not only partitioning of protein deposition has to be taken into account, but also the deposition rates of other body components like lipid deposition. Relations between protein and lipid deposition (an increase in protein deposition due to an increased energy intake also increases lipid deposition) make interpretation of mentioned static relations complicated. More knowledge of partitioning of body components and their relations with nutrition is needed for further evaluation of body formation.

Lean tissue deposition rates are easier to determine and available to a larger extent than protein deposition rates. However, for some purposes, knowledge of protein deposition rates is required. Whittemore (1983) proposed a linear relation between protein deposition and lean tissue deposition, in order to estimate protein deposition rate from lean tissue deposition rate. Present work confirms that there is a clear relation between these two deposition rates. However, the nutritional situation causing both deposition rates has to be taken into account as it influences the relation between protein deposition and lean tissue deposition due to the nutritional effects on the site of tissue deposition. The low percentage of *extra* protein which was deposited into the LEAN indicates that, especially at higher live weights, the response in valuable parts (LEAN) to increasing energy intake was decreasing. This is relevant for determination of the feeding level for an optimal lean and fat accretion. In order to increase desired protein deposition (protein in lean tissue), *total* protein deposition has to be increased to a substantially higher degree. Such an increase will be coincided with an even higher increase in lipid deposition. In conclusion, it can be stated that an increase in protein growth in the LEAN will be accompanied with an increase in fatness.

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GENERAL DISCUSSION

GENERAL DISCUSSION

INTRODUCTION

Effects of nutrition on body composition in pigs deal both with the accretion of tissue mass in the whole body as with the distribution of tissue groups within the body. In the present general discussion, these two aspects of partitioning of components in the pigs body will be discussed. First aspect is the partitioning of production energy into protein and lipid deposition. The second aspect of partitioning is the distribution of deposited protein and lipid into three major tissue groups: (*i*) lean tissue, (*ii*) other carcass tissue and (*iii*) organs. The distribution of muscles or other tissues within these major tissue groups has not been an issue in the present studies, but has been dealt with extensively in literature (see Walstra, 1980).

At first, a classical view on growth will be presented briefly. Next, the partitioning of production energy between whole body protein deposition and lipid deposition will be discussed on the basis of one of these literature concepts, the Linear-Plateau concept[3]. Using the studies in the present thesis, validity of the Linear-Plateau concept will be studied and alternatives to improve the concept will be proposed. Second major theme of the present discussion deals with the partitioning of deposited tissue (protein and fat) between different tissue groups. It was studied whether there are specific nutrition and body weight effects on the partitioning of deposited tissue in the body of pigs (chapter V). Both studied aspects of partitioning are not independent, because both are involved in determination of the optimum production strategy for lean production. Finally, the relation between both aspects of partitioning will be discussed.

A classical view on growth

The classical studies of Hammond (1932) revealed growth waves in the bodies of growing mammals: varying growth rates in different tissues and regions in the body. McMeekan (1940abc and 1941) intensively studied such aspects of tissue development in growing pigs and concluded that *"the major modifications in form and anatomical composition do not occur as isolated effects but rather as orderly changes spread over a number of correlated parts and originating in "some deep-seated rhythm of growth""*. A major conclusion from the classical growth studies (McMeekan 1941) is that nutritional manipulation affects fatty tissue more than muscle tissue. The latter is more affected than organs and bones. For pigs, this concept was previously proposed by Hammond (1932), and illustrated with figure 1. This figure shows three tissue groups, and their differential development in time. A nutritional manipulation (e.g. a decrease in feed intake) will affect tissue deposition, but relative development of these three tissue groups does not change. Furthermore, it was proposed that faster or later developing tissues (e.g. fatty tissue) are affected more by nutritional manipulations than early developing tissues.

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[3] Mentioning the Linear-Plateau concept references to both the linear-plateau relation between energy intake and protein deposition and to the additional assumption of a constant relation between lipid deposition and protein deposition (i.e. the integrated use of these two assumptions in growth models).

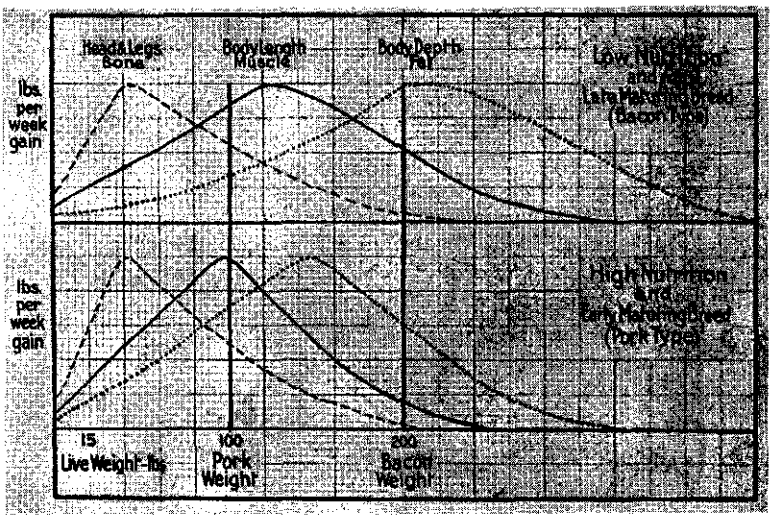

FIG. 8.—*Diagrams showing how the changes in body form and composition are brought about by differences in the time and rate of growth of different parts and tissues of the body.* The upper diagram shows how growth occurs in a late maturing breed (bacon type), or under conditions of a low plane of nutrition; while the lower diagram shows these changes in an early maturing breed, and under conditions of a high plane of nutrition. The curves represent growth of the parts of the body: (1) Head and legs, (2) Body length, and (3) Body depth—or alternatively of body tissues: (1) Bone, (2) Muscle, and (3) Fat. The thick vertical lines mark off the amount of growth made up to the time the animals have reached pork (100 lb.) or bacon (200 lb.) weight; this varies according as the breed is late or early maturing, and as the plane of nutrition is low or high (compare with Fig. 2).

Figure 1. A classical view on differences in growth rate between tissues, and the influence of nutritional manipulation (From Hammond, 1932).

The concept as presented by Hammond and other workers of the Cambridge school (McMeekan, Palson) have been studied and discussed intensively in literature. This resulted in a somewhat other view on the data of McMeekan (1940abc and 1941). Elsley et al. (1964, reanalysing McMeekans material) stated that fat deposition is not closely related to the growth of fat free body mass. When the effect of variation of fat was excluded in the material of McMeekan, Elsley et al. (1964) found no difference in development between bone and muscles. From these results, McMeekans interpretation was questioned. The reanalysis showed that muscle and bone do not have separate growth curves, thus they do not develop differently in time, as suggested by Hammond and illustrated by him in figure 1. When expressed as a function of muscle and bone mass, the composition of the body was very consistent. Davies (1983) also reanalysed the work of McMeekan and confirmed that the interpretation of McMeekan was caused by effects on fat deposition. Except for fatty tissue, Davies (1983) concluded that McMeekans work did "not support a concept of retardation of development by poor nutrition of those parts with the highest relative growth rates". Except for stomach and components of head and neck, there were no significant differences in relative proportions in the body caused by the nutritional treatments. The organs in the body showed a varying pattern in development, the "metabolic active" organs were retarded in their development, but others were not (Davies, 1983). In conclusion, the reanalysis of Davies showed that the fat free carcass shows a very consistent rate of development, but development of organs is quite diverse. Seebeck (1968) stated in this respect that "*to study the major components of the body, empty*

body weight would be used as the independent variate. To study the major components of the dressed carcass, dressed carcass weight would be used as the independent variate. To study the distribution of different parts of the musculature, total muscle weight would be used as the independent variate". Thus, total muscle weight is thought to determine to a large extent the distribution of individual muscles within the muscle group (within pig type). This was confirmed by studies on muscle conformation in cattle. From these studies, it was concluded that relative weights of muscles are rather constant when comparisons are made at equal total muscle weight (Butterfield and Johnson, 1968). A similar observation was made on fat depot tissues in pigs. In pigs receiving distinctly different nutritional treatments in the fattening period, there were clear systematic effects on the distribution of dissectible depot tissues (Van Bommel and De Greef, unpublished results). This will not be discussed further, because distribution of physical tissues within the three major tissue groups is not an aspect in the present thesis, but it illustrates an important issue in development of the body of mammals.

Summarizing, it can be stated that, although there are several deficiencies in the classical view on growth, some general aspects are still noticeable. A major agreement with the classical view is that functional tissue groups develop in a very constant rhythm, which is clearest when comparing comparable tissues as a function of the total group of those tissues (Seebeck, 1968; Butterfield and Johnson, 1968). Nutritional manipulation does not specifically affect those parts with the highest relative growth rates. A more general view may be that fatty tissue and other tissues can be regarded as rather independent (Elsley et al., 1964). Nutritional manipulations affect both tissue groups, and development within tissue groups follows regular patterns. An extensive review on this item, together with results of a large experiment was presented by Walstra (1980).

In general, deposition rates of lean and fatty tissue or of protein and lipid determine growth and body composition to a predominant degree. The previous paragraph indicated that they are regarded to be relatively independent from each other, but both are affected by nutrition. Thus, partitioning of production energy between protein and lipid deposition are principal aspects in research on performance in pigs. According to the general view presented in the previous paragraph, partitioning of deposited tissue within the body follows general rules. These rules are not known in detail. However, for the relation between protein and lipid deposition with lean production, a study of partitioning of this protein and lipid deposition between lean tissue and other tissues will be sufficient.

THE LINEAR-PLATEAU CONCEPT

As stated in the general introduction, production of meat with pigs requires an optimization between lean deposition and fat deposition. In order to quantify optimal feed allowances, several attempts have been made in literature to describe changes in tissue deposition as a function of varying nutritional input (feeding strategies). As early as in 1968, Blaxter stated that performance of pigs is predictable from nutritional input and theoretical assumptions. The only item which lacked was information on the ratio between lipid and protein deposition or (in other words) on the partitioning of production energy. The Linear-Plateau concept will be analyzed to study partitioning of energy into protein and lipid deposition. The Linear-Plateau concept has been chosen in this respect as it is

used most frequently in growth models and because its assumptions are rather close to experimental results (chapter II).

The Linear-Plateau concept assumes that, for an animal in a specified weight range, an increase in energy intake results in a linear increase in protein deposition. From a certain energy intake onwards, the protein deposition plateaus. This function was proposed by Whittemore and Fawcett (1976), and demonstrated experimentally by Campbell et al. (1983 and 1985) and by Campbell and Taverner (1988). Whittemore and Fawcett used this linear-plateau relation to propose a concept on partitioning of energy between protein and lipid deposition. It was assumed that, below the maximal protein deposition rate (*PDmax*), the ratio of lipid to protein deposition rate is constant. The minimal amount of lipid deposited for each unit of protein was characterized by the parameter *r*. Thus, it was assumed that composition of deposited tissue (protein and lipid) is independent of energy intake, up to the plateau level of protein deposition. Furthermore, it was assumed that the minimal ratio of lipid to protein deposition is constant over the fattening weight range. An example of the structure of a growth model which incorporates the Linear-Plateau concept has already been presented in the General Introduction of the present thesis.

Characterization of pigs in the Linear-Plateau concept

In the Linear-Plateau concept, pigs are characterized by two parameters, their maximum protein deposition *PDmax* and the minimal ratio between lipid and protein deposition *r* (Whittemore and Fawcett, 1976; Moughan et al., 1987; Watt et al., 1987; TMV, 1991; to some extent: Pomar et al., 1991). The version of the Massey model which was validated in chapter I characterized pigs only with their *PDmax*. Parameter *r*, the minimal ratio of lipid to protein deposition rate, was set to the value 1 in that version of the model. At low feed intakes (insufficient to allow maximal protein deposition rate), there is no effect of *PDmax* on composition of growth. At high feed intakes (sufficient to allow for maximal protein deposition rate), there is no effect of *r* on composition of growth. Most of the studies presented and discussed in the present thesis were performed with a restricted feeding regimen. Thus, under the assumptions of the Linear-Plateau concept, in the present studies, *r* was more important in our data than *PDmax*. Furthermore, conclusions of chapter I, II and III illustrate that the importance of the partitioning between protein and lipid (*r*) increases relative to *PDmax*. This is caused by the positive effect of both live weight and energy intake on the ratio of lipid to protein deposition. More lipid is deposited below the maximal protein deposition rate than assumed by the Linear-Plateau concept. Energy intake therefore has to be considerably higher to reach PDmax as compared to the expectations of the Linear-Plateau concept. Despite this increased relevance of energy partitioning at restricted energy intakes, characterization of pigs with regard to their maximal protein deposition capacity remains relevant, This is demonstrated by the results presented by studies of Campbell et al. (1983 and 1985) and Campbell and Taverner (1988). They clearly show that maximal protein deposition is attainable within the feed intake capacity for several types of pigs. Black et al. (1986) also expected pigs to have an intrinsic maximum for protein deposition capacity. In the following paragraphs, the validation of the Linear-Plateau concept will be discussed with emphasis on the partitioning between protein and lipid (parameter *r*) from the studies reported in chapter I - IV.

Validity of the Linear-Plateau concept: experimental data

From the validation of the Massey model (chapter I: De Greef et al., 1992a), it was concluded that "*... the parameter which accounts for the minimal ratio of lipid to protein is dependent on live weight*". With restricted feeding, a higher lipid to protein deposition ratio was observed beyond 65 kg live weight as compared to body weights below 65 kg live weight. This increase in lipid to protein deposition ratio was attributed to the increase in live weight. A later study using data from literature (chapter II: De Greef et al., 1992b) revealed that "*The linear-plateau response in protein deposition of pigs to increasing energy intake is accompanied by an increase in the relative amounts of lipid deposited. Therefore, each increase in energy intake produces a fatter pig*". Thus, studies indicated that there were both effects of live weight and effects of energy intake on tissue partitioning in pigs. The assumed constancy of the ratio of lipid to protein deposition *r*, (independent of energy intake and of live weight: Whittemore and Fawcett, 1976) was therefore seriously questioned by these results.

The effect of energy intake on *r* (as presented in chapter II) was studied later, and thus not yet known during the validation of the concept using the Massey model. In the validation study, chapter I, the increase lipid to protein deposition ratio was attributed to the effect of live weight. In that study, however, energy intake above maintenance requirement increased with body weight. Therefore, the increased energy intake above maintenance was confounded with the effect of live weight. Thus, the observed increase in ratio of lipid to protein deposition which was attributed to live weight was also influenced by energy intake. In order to quantify the effect of energy intake and body weight simultaneously, an experiment was designed and performed in which both the effects of body weight and of energy intake were studied independently (chapter III: De Greef et al., 1992c). The results revealed that "*both live weight and energy intake influence the ratio of lipid to protein deposition rate*". Thus, there are effects of both body weight and of amount of feed on the ratio between lipid and protein deposition. An increase in energy intake of 3.7 MJ increased the ratio of lipid to protein deposition rate by 0.2 units on average. This estimate can be used to discuss how much of the increase in ratio of lipid to protein deposition in the validation study was indeed attributable to the effect of live weight, and not to the increased energy intake. The results in chapter III indicate that the extra intake of 2.1 MJ DE/day beyond 65 kg live weight in the validation study may have accounted for about 0.1 units of the ratio of lipid to protein deposition. This is calculated as (difference in *r* between HIGH and LOW in chapter III) / (difference in energy intake between HIGH and LOW in chapter III) × (difference in energy intake in the validation) = 0.2/3.7×2.1 = 0.1 units of the ratio of lipid to protein deposition. The observed increase with live weight in *r* amounted to 0.3 units. Therefore, it can be concluded that in the validation, the mayor part of the observed increase in *r* can be attributed to the increase in live weight, and not to the increase in energy intake.

Moreover, in chapter III, interaction between live weight and energy intake on the relation between lipid and protein deposition was below significance. However, there is some indication that the increase of the ratio of lipid to protein deposition with increasing live weight does depend on amount of feed. The increase in LD/PD for treatment LOW was 0.0031 units per kg increase in live weight and for treatment HIGH the increase was more than twice as much (0.0066). Although not significantly verified, one has to discuss the

possible implications of such an interaction. The data in chapter III suggest that there may be a positive interaction between the two factors, which is most clearly demonstrated by figure 2 of chapter III: the difference in ratio of lipid to protein deposition between two feed intakes increased with live weight. This means that nutrition induced fattening of the pigs occurred especially at higher body weights. It is very common in practice and in experiments to increase feed allowances for pigs with increasing body weight. This increase in feed allowance offers the needed energy to deposit the live weight induced extra lipid deposition. However, as a result of the positive interaction between energy intake and live weight, this extra energy will again result in an increased fatness. According to the present results, only severe restriction of pigs will prevent such increases in fatness, but this will also largely reduce protein deposition rate.

In addition to the effects of body weight and feeding level, there are other factors which affect the relation between lipid and protein deposition. Results in chapter IV (De Greef et al., 1992d) showed that *"nutritional history can influence the relation between lipid deposition rate and protein deposition rate"*. From 65 kg live weight onwards, two very distinct groups of pigs were compared on an equal nutritional plane. The study revealed that pigs which were very fat due to a previous protein insufficiency had a reduced ratio of lipid to protein deposition as compared to control (more lean) pigs. Thus, partitioning of production energy into protein and lipid deposition is not only a function of type of pig (the Linear-Plateau concept, Whittemore 1983), of energy intake (chapter II and III) and of body weight (chapter I and III), but nutritional history also exerts an influence. This is a relevant item for designing feeding strategies. The effect of nutritional history thus illustrates that, apart from direct effects like energy intake and body weight, other effects also may influence composition of deposited tissue. Furthermore, chapter III revealed that nutritional history may also affect protein deposition rate in an *ad libitum* environment. In that study, pigs which had been fed restrictedly highly increased their feed intake after a switch to *ad libitum* feeding. This resulted in protein deposition rates which were considerably higher (well over 200 g/d) than normally observed protein deposition values. These effects were measured over a 20 kg weight range only, longer term effects were not measured. No further studies on carry over effects (effects of nutritional history) have been made, and this phenomenon will not be discussed in detail in the present thesis. It does illustrate, however, that not only direct factors, but also factors like energy intake at lower live weights have to be taken into account when assessing the effects of experimental factors on performance. The effects of nutritional history will be used shortly to illustrate aspects of development at the end of present general discussion, where relations between the Linear-Plateau concept and tissue partitioning are discussed.

As discussed in chapter II, literature data which supports the linear-plateau relation between energy intake and protein deposition do not support the additional assumption that r is constant. The observation that not only protein, but lipid also is linearly related to energy intake (chapter II) leads to another concept. It was derived in chapter II that these linear relations with energy intake imply that each additional unit of DE intake results in a constant composition of *extra* deposited tissue.

> This can be illustrated by an example, drawn from the data presented in chapter II, and originating from Campbell and Taverner (1988). Pigs which deposit 100 grams of protein and 100 grams of lipid at 25 MJ DE intake, have a ratio between lipid and protein of 100/100=1.0 at this energy intake. Each extra unit

of DE intake results in an increase of protein deposition of 5 grams, and an increase in lipid of 15 grams. In these pigs, therefore, the ratio between *extra* lipid deposition and *extra* protein deposition is 15/5 = 3. At 26 MJ DEI, the ratio changes from (100/100) = 1 to (115/105) = 1.1. At the highest energy intake, 40 MJ (15 MJ more than 25 MJ), protein deposition is increased by 15×5 grams = 75 grams, and lipid deposition is increased by 15×15 = 225 grams. The actual ratio is increased from 1.0 to 325/175 = 1.9. Thus, these pigs have a constant ratio between *extra* lipid deposition and *extra* protein deposition (15/5=3), but this does not imply a constant ratio between *total* lipid deposition and *total* protein deposition. This is an illustration of the statement in chapter II: "*It was concluded that ... the ratio between extra lipid and extra protein deposition is constant, but not the total (actual) lipid to protein deposition ratio*".

Thus, the relations between energy intake on one hand and protein and lipid deposition on the other hand show that not the ratio between *total* protein and *total* lipid deposition is constant, but the ratio between *extra* protein and *extra* lipid deposition. Hence, characterization can readily be adapted. It has to be changed from the parameter describing the relation between *total* protein and *total* lipid deposition into a parameter which describes *extra* protein and *extra* lipid deposition.

Conclusions of the evaluation of the Linear-Plateau concept

From the evaluation of the Linear-Plateau concept, it can be accepted that pigs can have a maximum protein deposition capacity. However, this only holds true in a constant nutritional environment, as data in chapter III indicate that this maximum can depend on nutritional history. The second major assumption in the Linear-Plateau concept is the constant ratio between lipid and protein deposition below maximal protein deposition rate. Results from experiments and literature showed a clear increase in this ratio caused by an increase in live weight and by an increase in feed intake. Thus, the ratio of lipid to protein deposition is not constant. It increases both with live weight and with energy intake. From the observation of a linear relation between energy intake and lipid deposition, it was derived in chapter II that the second assumption in the Linear-Plateau concept (a constant ratio between lipid and protein deposition), has to be adjusted into a constant ratio between *extra* lipid deposition and *extra* protein deposition.

Alternative characterization parameters for growing pigs

In the Linear-Plateau concept, pigs have two characterization parameters which are supplementary, the first parameter *r* operates below maximal performance, which maximum is quantified by the other characterization parameter, *PDmax*. From the previous paragraph, it is clear that the characterization parameter *r* does not describe the mechanism in partitioning between lipid and protein deposition accurately. For an improved quantification of the relation between protein and lipid deposition, the following aspects have to be taken into account.

(*1*) There is an effect of the level of energy intake on the partitioning of energy between protein and lipid (chapter II and III). In a previous paragraph, it was concluded that the partitioning of *extra* energy intake into *extra* protein and *extra* lipid deposition is a constant factor. This can be used as a characterization parameter.

(*2*) There is also an effect of live weight on the partitioning of energy into protein and lipid deposition. This effect of live weight on partitioning of production energy was demonstrated in chapter I and III. This effect should also be taken into account for the characterization of pigs in their partitioning of energy.

(*3*) In the Linear-Plateau concept, deposition of protein and lipid could readily be calculated from energy intake and the parameters *PDmax* and *r*. The discussion on the effect of energy intake on *r* will result in an alternative to this parameter. This alternative parameter describes the *change* in the ratio of lipid to protein deposition. As this alternative parameter is a relative parameter, a reference level for calculations is needed. Two approaches for this will be presented.
(*3a*) Such a reference level of performance could be derived from theory on energy metabolism. It will be studied whether energy metabolism at very low energy intakes may provide a reference level of performance.
(*3b*) A second possible reference level can be derived from an energy intake at which an absolute level is actually measured.

Parameters proposed in (*1*) and (*3a*) are theoretical in nature, and therefore may be general for growing pigs. (*2*) and (*3b*) have to be estimated empirically. Each of the points (*1*) to (*3b*) will be discussed in the following paragraphs, in combination with related assumptions in literature. Summarizing, a further study is needed on additional aspects towards a better characterization of growing pigs:
(*1*) effect of energy intake on partitioning between protein and lipid deposition
(*2*) effect of live weight on partitioning between protein and lipid deposition
(*3*) a reference level used to calculate the absolute level of performance
- (*3a*) energy metabolism at very low energy intakes
- (*3b*) energy metabolism at an empirically chosen fixed energy intake.

Alternative characterization parameters: (1) the effect of energy intake
The Linear-Plateau concept deals with the relation between energy intake and total protein and total lipid deposition. As stated in chapter II, from the linear relation between both protein and lipid deposition with energy intake, one can expect a constant ratio between *extra* lipid deposition and *extra* protein deposition.

This verifies the existence of a parameter which quantifies partitioning of production energy into protein and lipid deposition. However, in contrast to parameter r, which describes *total* protein and *total* lipid deposition, this parameter describes the *change* in the ratio of lipid to protein ratio with and increase in energy intake. This parameter will be referenced to as the *marginal ratio* (*mr*). In other words, *mr* describes the ratio between *extra* lipid deposition and *extra* protein deposition synthesized from an extra amount of energy.

This approach is very much comparable to an approach presented by the Agricultural Research Council (1981). In that study, it was suggested that pigs have a constant amount of extra nitrogen retention for each extra unit of Metabolizable Energy intake above maintenance. Assuming constant energetic efficiencies for protein and lipid deposition, this results in a constant ratio between *extra* lipid deposition and *extra* protein deposition. This is similar to the proposed mechanism of the constant marginal ratio (*mr*). When the approach was proposed by the ARC, appropriate experimental data were not sufficient to evaluate the concept adequately. Present work clearly supports this part of the ARC approach.

Black et al. (1986) also proposed a change in the composition of deposited tissue with a change in energy intake. They described extra nitrogen deposition as dependent on extra energy intake by the function G_{nbe}. This function is described as $G_{nbe} = (0.7 \times e^{-.092 \times W} + 0.65) \times X_s$, in which W = body weight and X_s is a scale factor to allow for differences between genotypes. Calculation of deposition rates is performed as follows. Pigs were assumed to have zero nitrogen accretion at an energy intake of 0.55 × maintenance energy requirement. Actual nitrogen deposition can be calculated from the amount of energy intake above 0.55×maintenance requirement, multiplied by the amount of extra nitrogen deposition per MJ extra energy intake (G_{nbe}). This approach was derived "*according to empirical equations established following consideration of published information*" (Black et al., 1986). Fat deposition is calculated from the energy which remains after all other energy demanding body functions have been met. This function G_{nbe}, describing the effect on nitrogen retention, can be converted to a function which describes extra protein deposition (ΔPD), by multiplication G_{nbe} with 6.25 (assuming 16% of nitrogen in protein). In table 1, values of ΔPD are calculated for 6 types of pigs, as proposed by Black et al. (1986). Assuming the amount of ME required for deposition of protein and lipid, ΔPD can be converted to a function which describes the ratio between *extra* lipid and *extra* protein deposition per extra MJ of ME. This is equal to the marginal ratio *mr*, presented at page 82. For calculation of these values, ME cost for protein deposition and ME cost for lipid deposition were assumed to be 53 kJ/gram protein or lipid. In table 1, values of *mr* are presented for the 6 types of pigs as proposed by Black et al. (1986). Table 1 indicates that the amount of extra protein deposited for each unit of extra energy (ΔPD) is lowest for the animals with the lowest genetic capacity. This is also reflected by the higher values of *mr* for the lower capacity pigs.

Table 1. Change in protein deposition per unit change in energy intake (ΔPD, g/MJ) at 25, 65 and 105 kg live weight and corresponding values for the marginal ratio between lipid and protein deposition (mr). Derived from Black et al. (1986).

Sexe	genotype	Xs	ΔPD_{25kg}	mr_{25kg}	ΔPD_{65kg}	mr_{65kg}	ΔPD_{105kg}	mr_{105kg}
Male	'fast growing'	1.2	8.1	1.3	6.4	2.0	5.6	2.4
Female	'fast growing'	1.0	6.8	1.8	5.3	2.5	4.6	3.1
Castrate	'fast growing'	0.78	5.3	2.6	4.1	3.5	3.6	4.2
Male	'slow growing'	1.0	6.8	1.8	5.3	2.5	4.6	3.1
Female	'slow growing'	0.85	5.8	2.3	4.5	3.2	3.9	3.8
Castrate	'slow growing'	0.65	4.4	3.3	3.5	4.5	3.0	5.2

Xs: scale factor to quantify differences between genotypes (Black et al., 1986)

The approaches of ARC (1981) and Black et al. (1986) both assumed a positive nitrogen balance at maintenance energy intake (and thus a negative lipid deposition at maintenance energy intake). As composition of *extra* tissue comprises both of protein and lipid, fatness increases with increasing energy intake. This is equal to the statement in chapter II "*This marginal ratio is larger than the ratio's of total lipid to protein deposition. Therefore, each additional unit of energy intake produces a fatter pig*".

Below maximal protein deposition rate, the ratio between *extra* lipid deposition and *extra* protein deposition is constant within pig type and within weight range. This parameter

marginal ratio, *mr*, can readily be used to predict effects of additional energy intake on deposition rates of protein and lipid. It allows an increase in fatness of deposited tissue with an increase in energy intake.

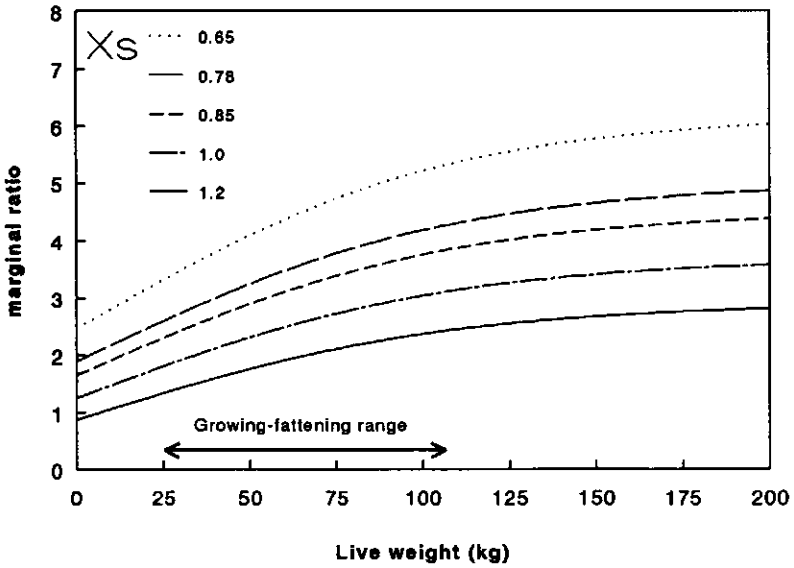

Figure 2. The relation between live weight and the marginal ratio (ratio between extra lipid and extra protein deposition) derived from Black et al. (1986). Xs represents a genetic scale factor.

Alternative characterization parameters: (2) the effect of live weight

In the approach as presented by ARC (1981, described in the previous paragraph), a constant ratio of *extra* lipid and *extra* protein deposition for the whole fattening range was proposed. Thus, a specific effect of live weight on partitioning of *extra* protein and *extra* lipid deposition was not taken into account. However, in chapter I and III, such an effect of body weight on composition of deposited tissue was clearly shown. In the experiments of the group of Campbell (reviewed by Campbell, 1988 and partly discussed in chapter II) grossly two weight ranges were studied, 20-45 kg live weight and 45 or 48-90 kg live weight. These data do not provide information to derive a specific weight effect on partitioning between *extra* protein deposition and *extra* lipid deposition because the used types of pigs were not comparable between weight ranges. An effect of live weight on the partitioning of extra energy into *extra* protein and *extra* lipid has been taken into account in the approach of Black et al. (1986, presented in the previous paragraph). The change in ratio between *extra* lipid and *extra* protein deposition with an increase in live weight is presented for 6 types of pigs in figure 2, using the approach of Black et al. (1986). For three live weights, these data were already presented as *mr* in table 1. In the fattening weight range (25-105 kg live weight, as indicated in the figure), the function increases. This approach therefore assumes that an additional unit of energy intake will result in fatter extra tissue at higher live weights. In other words, partitioning of extra production energy shifts from extra protein to extra lipid with increasing live weight. For the best genotype presented in

figure 2 ('Fast growing' males, x_s = 1.2), *mr* increases from 1.3 at 25 kg live weight to 2.4, an increase of 0.023 units per kg live weight on average. For the worst genotype ('slow growing' castrates, X_s = 0.65), *mr* increases from 3.3 to 5.3, an average increase of 0.053 units per kg live weight. Thus, this approach quantifies the increase with live weight of the ratio between *extra* lipid and *extra* protein deposition. Furthermore, it assumes that the increase with live weight in *mr* is more in pigs with lower genetic capacity. This differential increase with live weight shows an example of an interaction between genotype and live weight effect; the difference between the types of pigs increases with live weight.

Alternative characterization parameters: (3a) energy partitioning at low energy intakes
In the characterization parameters presented sofar, the only available parameter below *PDmax* is *mr*. However, this parameter does not quantify an absolute level, but the change of a level. Therefore, an absolute level is required. Such a reference level of performance can be derived from theory on energy metabolism. In this paragraph, it will be studied whether energy metabolism at very low energy intakes may provide a reference level of performance.

In most swine growth models and several literature sources, it is assumed that pigs partition their energy as described by a factorial approach (Kielanowski, 1972; ARC, 1981). Energy is partitioned into maintenance, protein deposition and lipid deposition. This is presented in formula (1). In this formula, MEI is daily metabolizable energy[4] intake, ME_m represents maintenance energy requirement, k_p and k_f represent energetic efficiency for protein and lipid deposition, respectively, e_p and e_f represent the net energy content of protein and lipid, respectively and PD and LD represent the amount (grams) of protein and lipid deposition, respectively.

$$MEI = ME_m + \frac{e_p}{k_p} * PD + \frac{e_f}{k_f} * LD \qquad (1)$$

At maintenance energy intake (MEI = ME_m), protein deposition is greater than 0 (Fowler, 1978; ARC, 1981; Close et al., 1983; Black et al., 1986). This means that, at energy equilibrium, pigs deposit protein at the expense of body lipid. The magnitude of this can be assessed by rearranging formula (1). When MEI = ME_m, then $PD \times e_p/k_p = LD \times e_f$. The ratio between lipid and protein deposition can then be quantified as $LD/PD = e_f/(e_p/k_p)$. Using the ARC (1981) estimates for k_p, e_p and e_p, PD/LD = -39.6/(23.8/0.54)= -0.90. When the efficiencies as presented by Moughan et al. (1987) are used, PD/LD = -39.6/(23.8/0.45)= -0.75. Thus, it can be expected that different types of pigs have a common point in their relation between lipid to protein ratio and energy intake. This point is defined at maintenance energy intake and described as (maintenance energy intake, ratio of lipid to protein at that point). Depending on the assumption of energy cost of protein deposition, this ratio at energy equilibrium will be between -0.75 and -0.90.

[4]When necessary, DE is converted to ME using the factor 0.96 (ARC, 1981).

In the data of Campbell et al. (1985) and of Campbell and Taverner (1988), it can be verified whether the curves describing LD/PD, when extrapolated, indeed cross the proposed common point. In figure 3, the lines describing the ratio of lipid to protein deposition rate as a function of energy intake are presented. These lines are calculated from data in table 1 of chapter II and drawn at a wider range of energy intakes than studied in the original data. The pigs presented in figure 3 grew from 45 or 48 kg live weight to 90 kg live weight. These pigs had an average maintenance requirement of 10.2 MJ ME (10.6 MJ DE) per day. One can expect from the calculations earlier in this paragraph that at maintenance energy intake (10.6 MJ DE/day) these lines have a common point, where LD/PD is about -0.90. Thus, the lines describing LD/PD would have to cross about the point (10.6, -0.90). Figure 3 illustrates that most lines indeed cross the horizontal axis at a positive DE value, and seem to have a mutual point. This point, however, is located at a somewhat higher DE intake than derived in the previous paragraph. Figure 3 indicates that the common point for the curves describing the ratio LD/PD for most treatment groups is located at a point where lipid deposition is about zero. Thus, the energy intake which is just adequate to prevent lipid breakdown is similar for most groups of pigs (figure 3). This point (14.5 MJ DE) is about 1.4×maintenance requirement for these pigs and this point is at a considerably higher energy intake than derived from formula (1). Summarizing, there may be a common point in the relation between energy intake and ratio of lipid to protein deposition for different types of pigs in a certain weight range. This point is located at energy intakes which are higher than maintenance energy requirement.

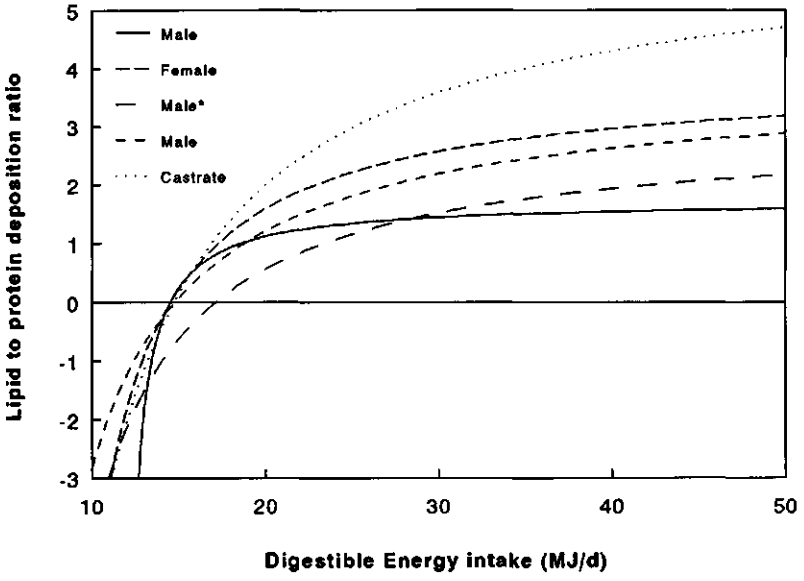

Figure 3. Relations between energy intake and ratio of lipid to protein. Calculated from linear estimates presented in chapter II, table 1. Drawn curves exceed the range of energy intakes studied.

ARC (1981) and Black et al. (1986) also proposed a fixed point at low energy intakes. From such a point onwards, one can calculate actual protein deposition. ARC (1981) assumed 5 grams of nitrogen retention at energy equilibrium. Black et al. (1986) assumed nitrogen deposition to be zero at 0.55 × maintenance energy intake. The previous paragraph showed that the data of Campbell et al. suggest a common point at zero lipid deposition. All three approaches for a common point at low energy intakes are comparable. However, the present theoretical approach could not be confirmed by (the sparsely available) actual data as presented in chapter II and the two approaches in literature lack a theoretical basis. Therefore, in the next paragraph, another approach for a reference point will be proposed.

Alternative characterization parameters: (3b) a reference energy intake

As stated before, for calculation of protein and lipid deposition from *PDmax* and *mr*, an absolute level is required. The previous paragraph failed to demonstrate clearly that such a point derived from theory indeed represents a mechanism. As more theoretical points are not available, such a point has to be assessed empirically. A possible method is to measure PD and LD at a certain energy intake. This is comparable to the approach as presented by ARC (1981). In this approach, performance at a certain energy intake was used as a reference (*i.e.* 25 MJ DE per day). Protein and lipid deposition rates at other energy intakes can be calculated from the protein and lipid deposition rates at this reference energy intake using the parameter marginal ratio (*mr*), the change in lipid to protein deposition ratio with one unit change in energy intake.

When a relatively low intake is chosen for a reference (like the 25 MJ DE), it can also be assessed in young pigs. A further advantage of a low reference level that the intake is not likely to be beyond *PDmax*. Thus, pigs receiving 25 MJ DE per day on average can be used to assess the performance of that type of pig at that energy intake. From literature data, it can be expected that 25 MJ DE per day will be within the limits of appetite for pigs above 30 kg live weight (ARC, 1981; NRC, 1987).

Assessment of parameters for characterization of pigs

The proposed parameters to quantify effects of body weight and nutrition on the partitioning of energy into protein and lipid deposition need to be quantified. These parameters are *PDmax*, marginal ratio *mr*, and protein and lipid deposition at a reference energy intake. A direct method for this is assessing these parameters from comparative slaughter techniques. The parameter *PDmax* can be assessed by measuring protein deposition rate in a nutritionally unlimiting environment (see Moughan and Verstegen, 1988). As discussed previously, the level of protein deposition capacity is affected to some degree by previous nutritional treatment. This means that the nutritional history of pigs which are used to be to be characterized has to be standardized. The value of the parameter *mr* can be quantified in two ways. Firstly by establishing the linear relation between energy intake and protein deposition (line PD) and the relation between energy intake and lipid deposition (line LD). The slopes of these two lines describe the change in protein and lipid deposition rate with a unit change in energy intake. The marginal ratio *mr* can then be calculated by dividing the slope of line LD by the slope of line PD. However, this requires full titration of deposition rates of protein and lipid on energy intake by means of comparative slaughter using several treatment groups. The second way

to quantify *mr* is by comparing tissue deposition at two feed intakes. From the difference between the two treatments in rates of lipid deposition and from the difference in rates of protein deposition, the composition of the extra tissue can be calculated. In table 2, *mr* values from the experiments studied in chapter II are presented. These are derived from the slopes of the curves describing protein deposition and lipid deposition. Also, *mr* in own data (chapter VI) are given, calculated from the difference between two restricted energy intakes. *mr* values in Table 2 show quite a broad range. Data from chapter VI indicate that live weight emerges a large effect on *mr*. This increase with live weight is larger than proposed by Black (1986), as drawn in figure 2.

Table 2. Ratio between extra lipid deposition and extra protein deposition(marginal ratio, mr) from experimental sources presented in this thesis.

Sexe	weight range	mr	Source
Male	20-45kg	2.3	Campbell et al., 1983
Female	20-45kg	2.4	Campbell et al., 1983
Male	48-90kg	1.7	Campbell et al., 1985
Female	48-90kg	3.9	Campbell et al., 1985
Male	45-90kg	3.0	Campbell and Taverner, 1988
Male	45-90kg	3.8	Campbell and Taverner, 1988
Castrate	45-90kg	6.2	Campbell and Taverner, 1988
Male	25-65kg	1.9	De Greef and Verstegen, 1992 (chapter V)
Male	65-105kg	3.6	De Greef and Verstegen, 1992 (chapter V)

Present work shows that *mr* is a parameter which is acceptable from a theoretical point of view, and that it is promising for characterization. The effect of live weight on this parameter can not be explained in physiological terms. As there is no systematic information on the effect of live weight, this has to be assessed empirically. Therefore, characterization will have to be performed at several live weights. This can be performed by performing a feeding trial with assessment of deposition rates using the comparative slaughter technique, as presented in the chapters I, III, IV and VI and proposed for assessing *Pdmax* and *mr*. However, this is an expensive and labour intensive routine. Two alternative methods will be presented here concisely. First is the method of nitrogen balances, second is the use of frequent *in vivo* measurements on pigs.

Performance can be quantified by conducting nitrogen and energy balance trials (Van Es and Boekholt, 1987; Verstegen et al., 1987). Assumptions in this are that nitrogen balances quantify protein deposition and that lipid accretion can be calculated from the difference between the energy balance and the nitrogen balance. A major problem in this respect is that N balance trials usually overestimate protein deposition (Just Nielsen, 1971; Just et al., 1982; Metz et al., 1984). An overestimation of protein deposition will result in a underestimation of lipid deposition. The degree of overestimation is also quite variable (Just et al., 1982). This results in a relatively high uncertainty about the level of lipid deposition and thus of the ratio of lipid to protein deposition.

Consequences of this can be seen in the following example. A pig receives 25 MJ metabolizable energy and 350 grams digestible crude protein (N×6.25). Nitrogen balance is measured to be 35 grams/day and energy balance is measured to be 11 MJ/day. This results in a calculated protein deposition of 35×6.25 = 219 grams of protein deposition and (energy balance - PD×e_p)/e_f = (11000 - 219×23.8)/39.6 = 146 grams of lipid deposition. Lipid to protein deposition ratio is estimated to be 146/219 = 0.67. If nitrogen balance was overestimated by 10%, lipid to protein deposition ratio would have been 159/197 = 0.81. At an overestimation of N balance of 30%, the ratio would have been 186/153 = 1.21. Thus, the range from the measured value to the value calculated using a frequently observed degree of overestimation (30%) results in a range of estimates of lipid to protein deposition of 0.67 to 1.21.

Differences in literature estimates of energetic efficiency of protein deposition have not been taken into account, but also exert some effect. Summarising, nitrogen balances can not be used to assess an absolute reference level for the proposed characterization of pigs.

An other approach in assessing performance characteristics *in vivo* is using data on energy intake and live weight gain. Average daily gain is composed of accretion of protein, lipid, water, ash and gut fill. According to literature, the amounts of ash and water deposition are closely related to protein deposition (Kotarbinska, 1969). Therefore, if one of the factors (especially protein or water) is known, total lean body mass (fat free empty body) can be calculated. When nutritional influences on gut fill can be quantified, growth rate can be calculated from protein and lipid deposition. Most swine growth models use this principle (Whittemore, 1983; Moughan et al., 1987; Pomar et al., 1991; TMV, 1991). This algorithm can also be inverted, calculating LD and PD from daily gain. This can be performed on frequent observation of live weight gain and energy intake. This gives as many estimates on protein deposition and lipid deposition as the number of observations in live weight gain and energy intake.

The principle is as follows. Protein deposition (PD) and lipid deposition (LD) are calculated by combining two approaches. First is the factorial approach, as presented in formula (1) in the previous paragraph (energy intake is directed to maintenance, protein deposition and lipid deposition). Second is the assumption that daily gain is a summation of (*a*) protein deposition, (*b*) lipid deposition, (*c*) water deposition, (*d*) ash deposition and (*e*) increase in gut fill. Deposition of water and ash are a function of PD, and gut fill is suggested to be a constant percentage of body weight. Input data are energy intake and live weight gain, derived from observations on feed intake and live weight. The two unknown parameters in both formulas are PD and LD for each observation in energy intake and live weight gain. Substitution of the formula describing the factorial approach into the formula describing daily gain will solve the relations, and present estimates for PD and LD.

This approach requires a dataset with frequent accurate measurements of live weights of animals, knowledge of feed composition and accurate recording of individual feed intake. This way of assessing model data using *in vivo* data succeeds well in characterizing performance, provided that the used data are correct. In literature, some comparable efforts have been proposed (Close, 1970). The fact that some of the assumptions in calculations are supported by experimental observations (chapter I) increases the accuracy to a substantial extent. Using the data as presented in chapter III, the procedure predicts lipid mass in pigs very well (r>0.9, Knol and De Greef, unpublished results).

Alternative characterization parameters: summary and final remarks

From the previous paragraphs, it can be summarized that some new parameters are needed to describe the response in lipid and protein to energy intake. The first parameter suggested was the change in ratio of lipid to protein deposition with a change in energy intake, the marginal ratio *mr*. This can be calculated from the difference between feed intakes. Comparable parameters were also proposed by ARC (1981) and Black et al. (1986). In the latter, a formula is proposed that quantifies the second parameter which is needed: the effect of live weight on this *mr*. Present work shows that this weight effect varies considerably between types of pigs. In the pis studied in present thesis, the effect of live weight was considerably higher than proposed by Black et al. (1986). Theory about this parameter is lacking. Furthermore, for calculation of absolute levels of protein and lipid deposition, a reference parameter is needed. On theoretical grounds, it was expected that the lipid to protein deposition ratio at energy equilibrium is such a point. Available data (presented in figure 3) indicated that this was not a common point for the studied groups of pigs. On the other hand, figure 3 does suggest that there is a mutual point for the groups at the DE intake at which lipid deposition is zero. An other alternative reference parameter was proposed: a reference energy intake (for example 25 MJ DE intake per day). This presents a level of performance (i.e. protein and lipid deposition rate) which can be used as a reference to calculate effects of other intake levels, using the other characterization parameters. Three proposed methods for assessing these parameters were presented: comparative slaughter, nitrogen balance and evaluation of frequent observations on energy intake and live weight gain.

TISSUE PARTITIONING IN PIGS

Introduction

The study presented in chapter V (De Greef and Verstegen, 1992) dealt with the relation between protein gain and lean tissue gain. The study indicated that, when assessing response of pigs, distribution into tissue groups also has to be taken into account. The study on partitioning of protein and lipid deposition between tissue groups clearly showed that *extra* tissue (caused by an increased energy intake) is partitioned between lean tissue and other tissue groups differently as compared to *total* tissue. This means that nutrition not only determines the type of tissue deposited (protein or lipid. chapter I - IV), but also influences the site of tissue deposition. Such effect of nutrition are closely related to body development.

A possible method for studying body development is the allometric approach. This can be performed either from physical data or from chemical data. The chemical approach, protein and lipid mass as a function of empty body weight, was presented in chapter III. In the present section, at first, weights of tissue groups will be quantified as a function of empty body weight (physical approach). Secondly, the physical and chemical approach will be combined by studying the partitioning of protein and lipid between the physical tissue groups. Description of weights of tissue groups as a function of larger groups or the whole body have received much attention in literature. The most commonly applied technique is the allometric approach. In this approach, the weight of a tissue(group) (Y) is expressed as a function of the whole body or a larger group (X) in the form $Y = aX^b$ (Huxley, 1932). The value of b is called the growth coefficient and quantifies the relative increase of tissue

(Y) in relation to the increase of the reference tissue (X). When the value of b is equal to 1, Y increases at an equal rate as X, this is called isometric growth. A value of b>1 indicates that Y increases faster than X. When the value for b is lower than 1, tissue Y increases relatively slower than tissue or tissue group X (Seebeck, 1978; Walstra, 1980).

Quantification of development of three tissue groups

In chapter V, partitioning of protein and lipid deposition between tissue groups was studied by comparing two feed allowances in two weight ranges. Effects of live weight and energy intake on partitioning of protein and lipid deposition were illustrated by calculating deposition rates in three tissue groups. An other approach was adopted in the study on whole body protein and lipid deposition as dependent on body weight and nutrition in chapter III. In that study, whole body protein and lipid mass were quantified as a function of total empty body mass with the allometric formula $Y = aX^b$. From this, the ratio between lipid and protein deposition rate was quantified and discussed. The data on whole body protein and lipid mass presented in chapter III were derived from chemical analysis in three tissue groups: (*1*) trimmed major joints (LEAN), (*2*) other carcass parts (DEPOT) and (*3*) blood and organs (ORGANS). Therefore, the allometric approach adopted in chapter III for the whole body can be used to describe nutrient partitioning within the whole body, thus between the three tissue groups, too. In the study on partitioning of protein and lipid deposition between body fractions (chapter V), data of pigs slaughtered at 25, 65 and 105 kg live weight were included. The dataset as presented in chapter III, also included pigs slaughtered at 45 and 85 kg. Therefore, the allometric approach allows more data to be included as compared to chapter V. This approach allows quantification of tissue development, and thus increase understanding growth and development of the pigs body. As an indication of development of the body, weight and chemical composition of three tissue groups are given in table 3.

Table 3. Weights and chemical composition (mean and standard deviation) of the tissue groups used to study partitioning of deposited tissue.

	LEAN			DEPOT			ORGANS		
Treatment	mass (kg)	%protein	%lipid	mass (kg)	%protein	%lipid	mass (kg)	%protein	%lipid
NONE 25	11.2 (0.7)	17.5 (0.3)	5.5 (0.4)	7.3 (0.7)	14.9 (0.5)	22.3 (1.1)	4.7 (0.3)	14.9 (0.7)	3.3 (0.5)
LOW 45	21.2 (0.9)	18.1 (0.8)	6.0 (0.1)	13.3 (0.3)	15.2 (0.6)	24.2 (1.7)	8.1 (0.4)	15.1 (0.4)	3.7 (0.4)
LOW 65	31.4 (1.1)	19.2 (0.3)	7.0 (0.4)	19.9 (0.6)	15.9 (0.4)	28.3 (1.3)	10.4 (0.6)	15.7 (0.3)	4.9 (0.3)
LOW 85	41.9 (1.3)	19.7 (0.2)	7.2 (0.9)	27.0 (0.5)	16.5 (0.6)	27.5 (2.5)	12.5 (0.5)	15.4 (0.4)	5.8 (2.2)
LOW 105	51.6 (1.1)	19.6 (0.2)	8.2 (0.4)	32.4 (1.3)	15.7 (0.3)	31.2 (2.6)	14.7 (0.9)	15.5 (0.5)	6.3 (0.7)
HIGH 45	20.3 (0.9)	18.1 (0.5)	6.7 (0.7)	13.5 (0.6)	15.2 (0.6)	26.1 (1.1)	8.2 (0.4)	15.3 (0.4)	4.5 (0.4)
HIGH 65	29.4 (0.7)	18.9 (0.1)	7.4 (0.8)	20.8 (0.5)	15.2 (0.2)	29.8 (1.9)	10.8 (0.6)	15.1 (0.2)	5.4 (0.4)
HIGH 85	38.9 (1.8)	19.1 (0.3)	7.6 (0.7)	28.0 (1.4)	15.5 (0.8)	31.2 (2.5)	12.5 (0.1)	15.6 (0.1)	5.6 (0.5)
HIGH 105	47.8 (2.3)	19.8 (0.1)	9.3 (0.8)	36.6 (2.7)	15.3 (1.2)	36.0 (3.7)	14.2 (0.0)	15.8 (0.6)	8.1 (1.3)

In table 4, the weights of the tissue groups LEAN, DEPOT and ORGANS are expressed as a function of empty body weight by means of the allometric approach. All presented b-values in table 4 were significantly different from 1, indicating that at both treatments all three tissue groups developed non-isometrically. Organs developed slower than the whole body (b<1), whereas LEAN and DEPOT increased faster than the empty body (b>1). In the classical approach, as presented by Hammond (1932, figure 1 at page 76), development

of organs is assumed to precede development of LEAN and DEPOT tissue. This is confirmed by the low b-values for ORGANS. There was no significant difference between the two feed intake levels on the development of the organ tissue group. On the other hand, the difference in energy intake (HIGH versus LOW) significantly affected the development of the carcass tissues. An increase in energy intake enhanced the relative development of DEPOT tissue ($P<0.01$), and reduced the relative development of LEAN tissue ($P<0.01$). The relatively small difference between b-values for LEAN and DEPOT at the LOW treatment indicate that development of these tissue groups is at a comparable rate at this feed intake level. Development of DEPOT tissue was enhanced more by an increase in nutrition than that of LEAN. This is also in line with the general view of the Cambridge school, as presented in figure 1 on page 76.

Table 4. b-values of physical tissue groups (Y, kg) expressed as a function of empty body weight (X, kg) using the allometric approach $Y = aX^b$.

	LOW		HIGH		
Y	b	se	b	se	significance[1]
ORGANS	0.77	0.02	0.76	0.03	ns
LEAN	1.07	0.01	1.03	0.01	$P<0.01$
DEPOT	1.06	0.02	1.13	0.02	$P<0.01$

[1] significance of the difference in b-value between LOW and HIGH
se: standard error of parameter estimate

Partitioning of protein and lipid between tissue groups

In table 5, protein masses in the three tissue groups are expressed as a function of total protein mass. Thus, partitioning of protein within the total protein mass is quantified. Similarly, lipid masses in the three tissue groups are expressed as a function of total lipid mass. Within the protein fraction, both LEAN protein and DEPOT protein increase faster than total protein (b is significantly higher than 1). In the LEAN protein tissue, the b-value is higher at the LOW energy intake as compared to the HIGH energy intake ($P<0.01$). In the DEPOT protein tissue, this effect is inverted. In this tissue group, protein develops relatively faster at the HIGH energy intake. In other words, at the LOW intake, LEAN protein develops faster than DEPOT protein. But, at a DE intake of 3.7 MJ more per day, the relative contribution of DEPOT tissue is increased to such an extent that protein in this tissue develops faster than protein in the LEAN tissue. This indicates that there is a preference to deposit protein into the LEAN, but that with an increase in energy intake there is an increasing proportion of protein deposited into the DEPOT. This agrees with the observation in chapter V that more protein is deposited into the LEAN than into DEPOT, but that *extra* protein is preferentially deposited into DEPOT. The relative amount of protein in ORGANS declines with an increase in total protein mass ($b<1$) for both treatments. There was no significant effect of treatment on ORGANS-protein development (similar b-values for LOW and HIGH). This indicates that nutrition did not affect the proportion of total protein which was partitioned to the organs. Different organs have very different functions. Therefore, the effect of nutrition on development is also diverse (Davies, 1983). Therefore, the absence of a nutritional effect on development of protein in the ORGANS tissue group can be caused by a summation of different effects on different

organ groups. In general, data on ORGANS protein development show a comparable trend as data on development of total organ mass (previous paragraph).

The effect of nutrition on partitioning of total lipid between LEAN and DEPOT showed a similar pattern as partitioning of total protein between these tissue groups. An increase in energy intake enhanced development in DEPOT, but relatively reduced development of lipid in LEAN. The development of lipid in ORGANS is more isometric (b-value closer to 1) than that of protein in the organs, suggesting that some tissues included in the ORGANS group serve to some extent as storage tissue of lipid. In general, the b-values describing distribution of lipid between body fractions differed less from 1 as compared to the b-values of the protein masses. Thus, the effect of live weight on partitioning of lipid between the three studied tissue groups is less obvious than the comparable partitioning of protein.

Table 5. Distribution of total protein and lipid (X, kg) between protein and lipid in the physical tissue groups (Y, kg) using the allometric approach $Y = aX^b$.

	LOW		HIGH		
	b	se	b	se	significance[1]
PROTEIN					
ORGANS	0.750*	0.020	0.766*	0.025	ns
LEAN	1.059*	0.008	1.029*	0.009	P<0.01
DEPOT	1.022t	0.016	1.072*	0.016	P<0.05
LIPID					
ORGANS	0.975	0.042	0.948t	0.033	ns
LEAN	1.032t	0.022	0.953t	0.029	P<0.05
DEPOT	0.989	0.010	1.021*	0.011	P<0.05

se: standard error of parameter estimate
b-values tagged with t tend to differ from 1 (P<0.10)
b-values tagged with * differ significantly from 1 (P<0.05)
[1] significance of the difference in b-value between LOW and HIGH

The difference between the two nutritional treatments (HIGH and LOW) allows assessment of the cumulative difference in weights of protein and lipid in the three tissue groups. The allometric approach quantified protein mass as a function of empty body weight. Substraction of the estimated formula describing protein mass for the LOW fed pigs from this formula of the HIGH fed pigs quantifies the difference in protein mass between both treatments as a function of empty body weight. In figure 4, this difference in protein mass between the HIGH and LOW treatment is presented. This has been performed for the whole empty body and for the three tissue groups separately. At any body weight, the amount of protein in the empty body is lower for the pigs receiving the HIGH treatment, as compared to the pigs receiving the LOW treatment. This effect on whole body composition is a summation of the effect on LEAN, DEPOT and on ORGANS. The figure shows that the effects on the tissue groups were different. The amount of protein in LEAN was considerably lower for the HIGH treatment. However, this effect was counteracted partly by the higher protein mass in the DEPOT tissue. The nutritional effect on ORGANS was small. In the light of the present study on partitioning, this differential effect on whole body and DEPOT indicates that at a higher energy intake, there is a preference to deposit

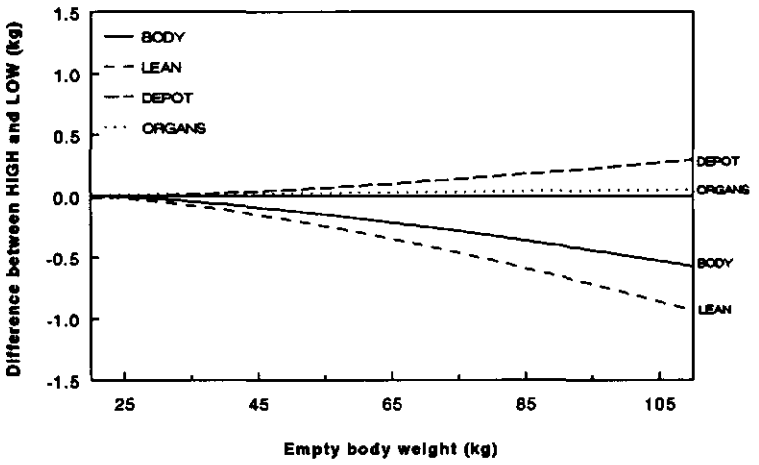

Figure 4. The difference in protein mass between HIGH and LOW fed pigs, as a function of empty body weight.

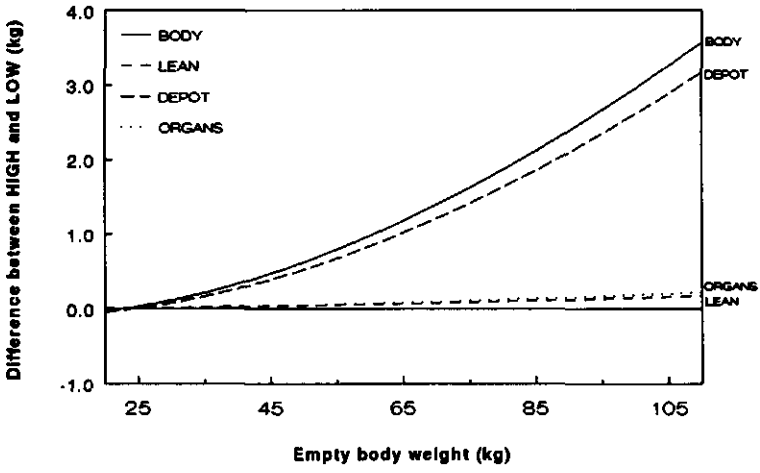

Figure 5. The difference in lipid mass between HIGH and LOW fed pigs, as a function of empty body weight.

protein into other carcass parts than LEAN. This is a clear illustration of the effect of nutrition on the site of protein deposition as mentioned in chapter V. Although protein in the LEAN is reduced due to the increased fatness, protein in other carcass tissues is even increased.

In figure 5, the difference between the HIGH and LOW treatment is presented for the lipid mass. This is calculated similarly to the method as described for protein mass. Lipid mass is higher in the whole body in pigs receiving the HIGH treatment. This is reflected in all three tissue groups. The predominant part of the extra lipid in HIGH pigs as compared to the LOW pigs was present in the DEPOT fraction.

Figure 4 and 5 clearly show that *extra* tissue (both protein and lipid) is predominantly deposited into the DEPOT tissue. Furthermore, it illustrates that when pigs have a lower protein content due to an increased feed intake, these pigs may still have more protein in non-lean carcass parts. This stresses the relative priority of the DEPOT fraction for protein deposition at higher feed intakes.

In conclusion, it can be stated that, in the studied weight range, the majority of tissue is deposited into carcass LEAN and DEPOT. On average, LEAN tissue developed at a comparable rate as DEPOT tissue. However, these development rates were highly affected by energy intake and live weight. An increase in both of these factors affected DEPOT more than LEAN. Thus, even in the used pigs, boars of high genetic merit, there is a strong trend for the body to fatten. The partitioning of protein and lipid mass showed comparable trends as development of the physical tissue groups LEAN and DEPOT, respectively. The difference in energy intake (3.7 MJ DE) resulted in clear differences in protein and lipid masses in the body fractions. Lipid mass was lower for the LOW fed pigs in all three body tissues. In HIGH fed pigs, protein mass in the whole body was reduced but not in DEPOT tissue. This last phenomenon illustrates a clear intrinsic propensity to deposit protein in other tissues than those commercially relevant (LEAN).

RELATIONS BETWEEN THE LINEAR-PLATEAU CONCEPT AND TISSUE PARTITIONING IN PIGS

In the previous paragraphs, partitioning of body tissue was presented from several angles. In the present paragraph, relations between the Linear-Plateau concept on one hand and the partitioning of protein and lipid between the tissue groups on the other hand will be analyzed shortly. At first, the ratio of lipid to protein deposition rate will be presented in the three tissue groups separately. Secondly, information on tissue partitioning will be used to assess the acceptability of a consistent relation between physical lean tissue deposition rate and energy intake. Finally, a number of general remarks on relations between aspects discussed in present thesis will be made.

The ratio of lipid to protein deposition in tissue groups

Similar to the method of quantifying the ratio of lipid to protein deposition rate in the whole body as applied in chapter III, this can be performed to derive this ratio in the three tissue groups. Figure 6 presents this. Data in chapter III showed that the ratio between lipid deposition and protein deposition in the whole body increased with an increase in

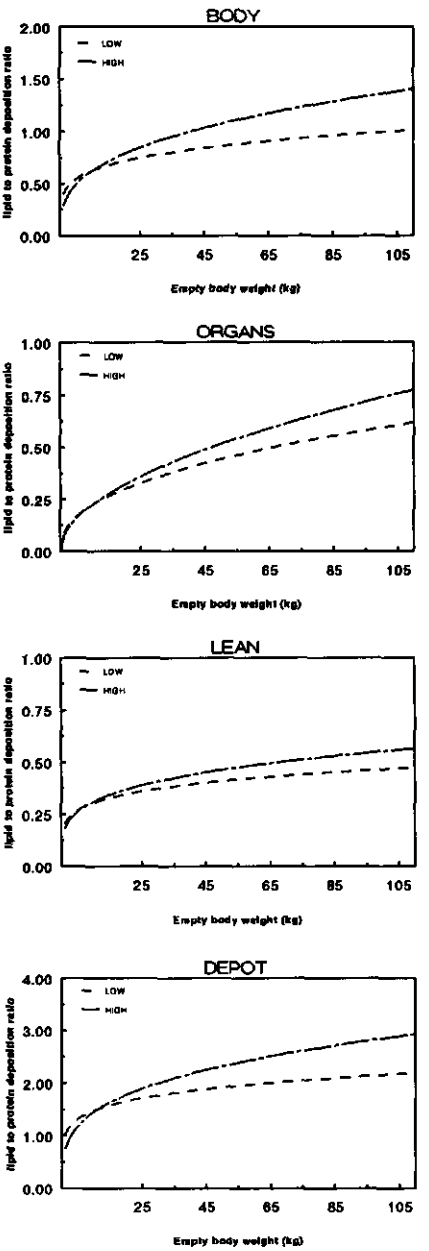

Figure 6. The ratio of lipid to protein deposition in the tissue groups, as dependent on body weight and feed intake. Calculated from the allometric description of protein and lipid mass in the tissue groups.

energy intake and with an increase in live weight. Figure 6 shows that this aspect is present especially in the DEPOT fraction and in the ORGAN fraction. The ratio of lipid to protein deposition in the LEAN tissue hardly differed between treatments. Furthermore, again except for the LEAN tissue, the difference between HIGH and LOW increased with live weight. As discussed in chapter III, this interaction between feed intake and live weight cannot be confirmed statistically, but it is clearly illustrated by the graphs. The fact that the observed fattening in the whole body is hardly reflected in the LEAN tissue illustrates that this tissue is a rather good measure of lean body mass. Furthermore, it demonstrates that composition of DEPOT tissue and ORGAN tissue are more dependent on factors like nutrition and body weight.

Data and conclusions in present thesis clearly showed that the deposition of lipid can not be regarded as only depositing a surplus of energy in the body. Results showed that a decrease in energy intake both affects lipid and protein deposition. Therefore, there must be some mechanism of priorities between these tissues. It has been suggested that there are functionally two groups of lipid tissue: a functional one and one for storage of energy (Fowler, 1968; Emmans, 1981). The fact that a decrease in energy intake also reduces protein deposition rate even at considerable lipid deposition rates shows that the assumed 'functional' lipid mass is more than physiologically essential lipid in tissues like membranes etcetera. This is illustrated by the substantial level of the ratio of lipid to protein deposition in all three body fractions. Present work stresses the need for quantification of the relation between protein and lipid deposition. Especially the priority between these two processes is important.

The relation between energy intake and lean tissue deposition.

A similar relation with energy intake has been proposed both for lean tissue deposition and for protein deposition. For both tissues, a linear increase with increasing energy intake was proposed, up to a maximum (Whittemore and Fawcett, 1976; Whittemore, 1986). In the first part of this thesis and in the first part of the present discussion, it was concluded that the linear-plateau relation between energy intake and protein deposition is confirmed by experimental data and therefore is a true relation. Proposing the linear-plateau relation between energy intake and lean tissue deposition, the author stated "*This proposition is remarkably fundamental to animal production strategies, and should be assessed critically if it is to become inherent within the structure the structure of an animal growth model. Maximum lean tissue growth rate is assumed to be largely independent of animal weight and age*" (Whittemore, 1986). There are no studies in literature which allow a reliable assessment whether lean deposition indeed has a linear relation with energy intake, as proposed by Whittemore (1986). Combining results from the previous chapters may help in discussing the acceptability of the linear-plateau relation between energy intake and lean tissue deposition. Two main conclusions from the studies were that (*1*) there is indeed a linear relation between energy intake and protein deposition and that (2) a decreasing proportion of that protein is partitioned into the lean. Combined with the fact that composition of the lean itself was not very sensitive to nutritional manipulations (table 3; figure 6; Hovenier and De Greef, unpublished results), it can be studied whether the linear-plateau relation between lean deposition and energy intake can be expected. The study on partitioning of protein and lipid deposition (chapter V) showed that an increase in protein deposition resulted in a change in the physical site (tissue group) where *extra*

tissue was deposited. Although an increase in energy intake resulted in an increase in protein deposited in the lean tissue, the percentage of protein deposited into the lean decreased with increasing protein deposition rate. Thus, the response of protein deposition into the lean mass as a function of total protein deposition shows diminishing returns. When the protein content of the lean is assumed to be largely independent of nutrition, each additional unit of whole body protein will result in a lower amount of *extra* lean tissue. However, this does not imply that LTD cannot be linearly related to DE intake.

> Assume: PD and DEI are linearly related in the form PD = a + b×DEI (1). The relation between protein deposited into the lean (PDlean) and DEI can then also be linearly related in the form PDlean = c + d×DEI (2), in which c < a and d < b. In this way, PDlean is a fraction of PD, which decreases with increasing DE intake (due to partitioning of protein to other tissue groups, chapter V). Chapter V also shows that the relation between lean tissue deposition and PDlean is independent of nutrition. LTD = e + f×PDlean (3). Substitution of (2) into (3) results into the formula LTD = e + f×(c + d×DEI) = g + h×DEI (4). Thus, it can be derived indirectly that LTD may be linearly related to DEI. It is important to note that this is not a proof that LTD is linearly related to DEI, but illustrates (using experimental data) that this is not impossible.

Thus, present data indicate that lean tissue deposition may be linearly related to energy intake. It has to be taken into account, however, that in order to increase LTD, an increasing proportion of protein will be deposited into less valuable parts, and that lipid deposition will increases to a larger extent. This means that an increase in lean tissue deposition will result in a fatter body (lower lean yield) at a certain slaughter weight. Present data do not allow to discuss this aspect more quantitatively, but the qualitative approach is clear in recognizing that in the relation between energy intake and lean tissue deposition, partitioning of deposited protein and simultaneous lipid deposition have to be taken into account.

Comparison of the classical approach with present results: DEVELOPMENT

As mentioned in page 77, several authors proposed that mass of total tissue determines development of that tissue. For example, total muscle mass determines the relative amounts of the individual muscles present. This implicates that there is a certain rhythm which determines development. In fact, this conclusion is still close to propositions made by the Cambridge school, as supposed by McMeekan (1941), except for the interpretation of effects of fatty tissue deposition. Especially the chemical body composition may give a clear illustration of this phenomenon. In chapter I, it was demonstrated that the composition of lean body mass (fat free tissue: protein, water and ash) is a function of total lean body mass and not of body weight. In figure 7, water mass in the whole body is plotted against chronological age, empty body weight and protein mass. The data originate from the studies presented in chapter I and III. The pigs ranged widely in body composition (empty body lipid percentage ranged from 10% to 38%). It is clear that water mass is more related to body weight than to chronological age. Water mass is closest related to protein mass. In the plot of water mass, the line as used generally in growth models (Kotarbinska, 1969) is drawn. Also, this line increased with 10% is drawn, this fits very well to the observed data. From the close relation of water mass to protein mass, irrespective of lipid mass, it can be concluded that the relation between protein mass and water mass was not affected by the amount of lipid in the body. From this, it can be concluded that lipid mass is not influencing the relation between protein and water. Thus,

development of fat free tissue is independent of lipid mass, but dependent on the weight of fat free tissue itself. It further indicates that lipid deposition does not replace water, which is sometimes suggested in elderly literature. This example illustrates that development of the fat free mass is consistent and largely independent on lipid mass.

Figure 7 clearly illustrates that chronological age did not affect composition of the fat free mass, but only the amount of fat free mass (lean body mass) itself (or one of its components like protein). Fat free mass can thus be regarded as a measure of physiological age or degree of development. The fact that the very fat pigs in chapter IV were depositing lipid and protein at a rate which is normally seen in much younger pigs may illustrate this. These pigs were chronologically much older (60 days), but their fat free body masses were considerably lower. The composition of growth was more in line with their protein mass than with their body weight or chronological age. This is an illustration of the hypothesis that performance in pigs is more dependent on body weight than on age. The studies of McCance and Widdowson (1974) strongly support this. Pigs held at a constant body weight for a long period showed, after realimentation, growth rates typical for pigs of that body weight. Summarizing, it can be stated that weight is more important than chronological age. A further refinement of this is that fat free body mass is even closer related to performance and body composition.

Physiological age (development) in the growing-fattening weight range in pigs cannot be seen apart from total development, the animal grows towards a mature mass. Several concepts of growth deal with such a mature (lean) mass. Mature lean mass may be a good characterization parameter, and degree of maturity may allow assessment of effects of nutritional history by affecting partitioning of deposited tissue. However, experimental data on mature masses are scarce, thus evaluation of their relation to performance characteristics is hardly possible. It remains unclear to what extent the span from mature mass determines partitioning of tissues and to what extent it explains compensatory effects after restrictions. In the present thesis, only short term effects on tissue partitioning were evaluated. The effects of nutritional history indicate the relevance of an extension towards higher body weights.

Figure 1 presented a classical view on effects on body composition in mammals. This view combined rate of tissue deposition ('growth') and composition of deposited tissue ('development'). Present work shows that these cannot be regarded as being independent. The observed effect of live weight on the marginal ratio between lipid and protein deposition may well be a parameter quantifying the degree of development rather well. A physiologically older animal will have an increased *mr* at a given live weight as compared to a physiologically younger animal. This difference in physiological age will be reflected in composition of growth at a certain nutrient intake. Furthermore, such an increase in *mr* will coincided by a relative increase in non lean tissue mass. This is the major combination between the concept on energy partitioning between protein and lipid deposition (part I of present general discussion) and the concepts on partitioning of deposited tissue between tissue groups within the body (part II of present general discussion). As differences in development are reflected in body composition, growth models should take aspects of development into account as major determinants of tissue partitioning.

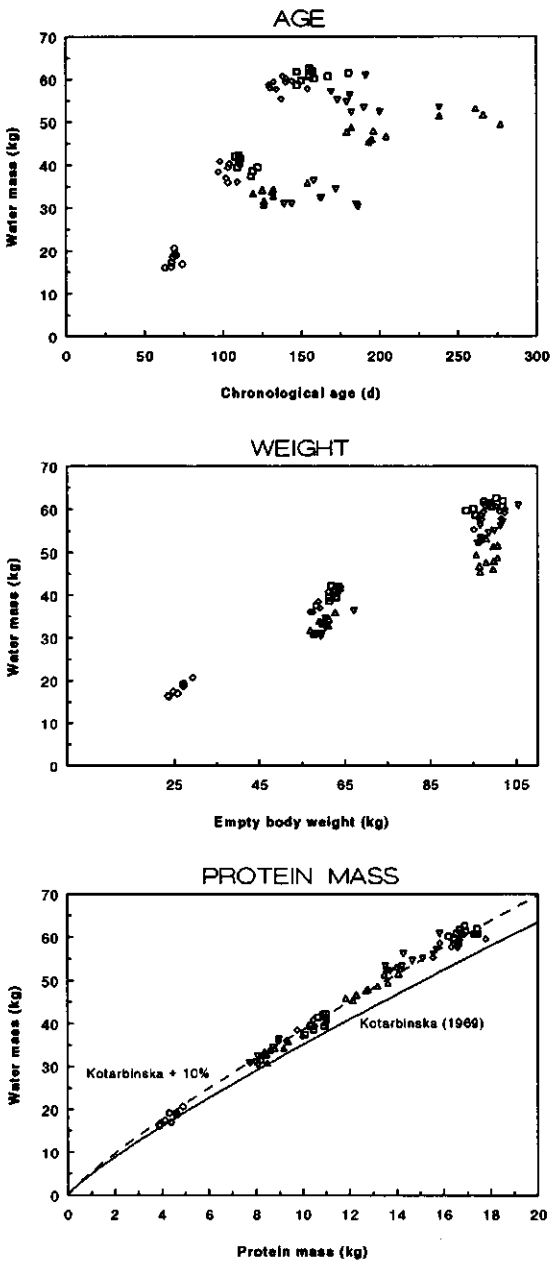

Figure 7. Relation of water mass with chronological age, body weight and protein mass in pigs varying widely in body composition. Different symbols represent different treatment groups.

Final remarks of the general discussion

In conclusion, an increase in energy intake will result in an increase in protein deposition, up to a plateau. For practical nutrition, a clear optimum in feed allowance is difficult to assess for two reasons. First reason is that the increase in protein deposition is associated with a larger increase in lipid deposition. Thus, there is no clear optimum, as suggested by the Linear-Plateau concept (General introduction, page 5). Second practical consideration must be that the response in protein deposition is not fully reflected in lean tissue deposition although lean tissue deposition is the ultimate aim of swine production. Both effects (increasing lipid deposition and decreasing response in lean) are not beneficial, and therefore, optimization between nutrient allowance and performance has to be performed. Present knowledge, both of the physiological and of the quantitative relations between the respective factors, is insufficient to identify an optimum. Present study emphasises the need both for assessing relations between nutrition intake and performance and the need for a theoretical basis of nutrient partitioning. This is especially relevant when concepts on nutrient partitioning are used in growth models to determine optimal feeding allowances. An important item in this is taking into account whether there are carry over effects. If these are present, it may well be that a suboptimal performance in a weight range results in an optimal overall performance, due to positive carry over effects. An example for this is feeding pigs restrictedly in order to influence feed intake capacity in a later weight range. Such effects of nutritional history have shortly been mentioned in the present thesis, but more information is needed for taking such aspects into account for optimizing feeding strategies.

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SUMMARY

GENERAL INTRODUCTION

General theme of present thesis is partitioning. Two aspects of growth in the pig which have to do with partitioning are presented in this thesis. First aspect of partitioning is the partitioning of production energy into protein and lipid deposition. This is studied by means of the evaluation of a concept on this, the Linear-Plateau concept. Second aspect of partitioning is that of deposited protein and lipid within the body of pigs. This is especially relevant for the relation between chemical and physical body composition.

Many factors influence body composition of pigs. A major factor in this is nutrition. As the many factors are not independent, description of production has to be multifactorial. In literature, several approaches have been presented to calculate performance of growing pigs from aspects of the animal, nutrition and environment. An important step forward in relating tissue deposition to nutritional input was the Linear-Plateau concept. It describes the relation between energy intake and protein and lipid deposition in growing pigs. The Linear-Plateau concept assumes that protein deposition increases linearly with increasing energy intake, up to a maximal protein deposition rate, where the relation plateaus. Below this plateau, there is a constant minimal amount of lipid deposition (LD) accompanying each unit of protein deposition (PD). Thus, below maximal protein deposition rate, there is a minimal ratio between LD and PD. Above the protein deposition capacity, all energy not used for protein deposition and the minimal lipid deposition is used for lipid deposition. The mentioned maximal protein deposition rate (*PDmax*) and minimal ratio between lipid and protein deposition (r) are assumed to be constant for pigs during the growing-fattening range.

VALIDATION OF A GROWTH MODEL

In Chapter I, a growth model for pigs which incorporates the above mentioned Linear-Plateau concept was validated. Two strains of pigs were fed either a protein sufficient ration at one of two levels or a protein deficient (50% of recommendation) ration fed *ad libitum*. Average daily gain, feed conversion ratio, chemical body composition at slaughter and deposition rates of protein and lipid were compared to values calculated by the model. In general, the model predicted the effects of the nutritional treatments adequately. Especially protein deposition rate and live weight gain were predicted well. However, above 65 kg live weight, the model underestimated lipid deposition rate in the protein sufficiently fed pigs by 20-30%. An increase in the ratio between lipid to protein deposition rate at the higher live weights was observed. The model did not account for this, it assumed no effect of live weight on this ratio. In contrast to the protein sufficient rations, in the protein deficient treatment, lipid deposition was overestimated considerably while protein deposition rate was predicted adequately. Results also indicated that the amount of water deposited for each unit of deposited protein is higher than predicted by the model. Main conclusion from the validation was that the parameter which accounts for the minimal ratio of lipid to protein deposition (r) depends on live weight.

ENERGY INTAKE AND ENERGY PARTITIONING

In chapter II, data from three experiments presented in literature which support the linear relation between energy intake and protein deposition were studied. Aim of this was to check whether these data also support the assumption of the Linear-Plateau concept that there is a constant ratio between lipid and protein deposition at energy intakes lower than needed for maximal protein deposition. The study revealed that not only protein deposition, but also lipid deposition was related linearly to energy intake. However, the ratio between lipid deposition and protein deposition increased with each increase in energy intake. Therefore, results of this study questioned one of the major assumptions of the Linear-Plateau concept, the constancy of *r*. In the discussion of chapter II, it was derived that, on theoretical grounds, one can indeed expect a constancy in the relation between protein deposition and lipid deposition. However, there is not a constant ratio between *total* lipid deposition and *total* protein deposition, but a constant ratio between *extra* lipid deposition and *extra* protein deposition. This extra deposition is the deposition of tissue caused by an extra unit of energy intake. Every increase in energy intake results in fatter composition of growth because the ratio between *extra* lipid and protein deposition is higher than the ratio between *total* lipid and protein deposition.

BODY WEIGHT, ENERGY INTAKE AND ENERGY PARTITIONING

Chapter I and chapter II both questioned the constancy of the ratio of lipid to protein deposition. Effects of live weight and of energy intake on *r* were demonstrated, respectively. In chapter III, these two factors were studied simultaneously in an experiment on entire male pigs. Special effort was made to have the effect of energy intake independent of live weight. Results showed that, for both levels of intake, the ratio of lipid to protein deposition rate increased with increasing body weight. At the LOW energy intake, the ratio of lipid to protein deposition rate increased from 0.74 at 25 kg to 0.99 at 105 kg body weight. In animals receiving the HIGH treatment, the ratio of lipid to protein deposition rate increased from 0.82 to 1.35 in that weight range. It was concluded that both live weight and energy intake influence the ratio of lipid to protein deposition rate. There was no significant interaction between the effect of energy intake and of body weight, but graphs indicated an increase with live weight of the difference between the two energy intake levels. Thus, results suggested that nutrition induced fattening was most pronounced at higher live weights.

NUTRITIONAL HISTORY AND ENERGY PARTITIONING

The effect of previous nutritional treatment on the partitioning of production energy was illustrated with a study presented in chapter IV. Between 28 kg and 65 kg live weight, entire male pigs were given diets either deficient or adequate in protein content. From 65 to 105 kg LW all pigs were fed a protein adequate ration. Below 65 kg live weight, the protein restriction reduced feed intake, live weight gain and rate of protein and lipid deposition to a high degree. At 65 kg, restricted animals had twice as much lipid and were on average 60 days older than controls. During realimentation, (beyond 65 kg live weight), previously restricted pigs (compared to controls) had slightly (7%) reduced feed intake and 15% increased weight gain and efficiency. The most prominent finding was that

composition of growth was highly affected by previous nutritional treatment. The previously protein restricted pigs were much fatter than the control pigs, but their composition of gain beyond 65 kg live weight was much leaner. Between 65 and 105 kg live weight, the ratio of lipid to protein was 1.23 for these pigs, whereas it was 1.69 for the control pigs. In fact, composition of growth of the previously restricted pigs resembled gain of normal fed pigs at a considerable lower body weight. Thus, the experiment revealed that nutritional history may influence the partitioning of production energy between protein and lipid deposition.

PARTITIONING OF DEPOSITED TISSUE IN THE BODY

In chapter V, the effects of nutrition and live weight on the site of protein and lipid deposition were studied. Response in protein and lipid deposition to energy intake was studied in three body fractions between 25 and 65 and between 65 and 105 kg live weight. The three body fractions were dissected LEAN (including enclosed bones, according to the Dutch standard dissection method), other carcass tissue (DEPOT) and non carcass body components (ORGANS, including blood). On average, 57% of total protein deposition was deposited into the LEAN fraction and 68% of total lipid deposition was deposited into the non-lean carcass parts. An increase in energy intake resulted in an increase in both protein deposition and lipid deposition. Of this *extra* protein deposition and *extra* lipid deposition, 42% and 75% respectively were directed to the non-lean carcass parts in the live weight range 25-65 kg. Above 65 kg live weight, these respective percentages were 71% and 82% on average. Thus, despite the relatively low intake levels, the major part of extra tissue deposition was deposited into non lean carcass tissue. The effect of a 40 kg increase in body weight on protein and lipid deposition rates was small as compared to the effect of an extra 3.7 MJ DE (about 250 grams of feed). Thus, for the used feeding regimen, partitioning of protein within the body was mainly effected by nutrition, and not by body weight. It was concluded that, in order to increase lean tissue deposition rate by increasing feed intake, substantial increases in body fatness need to be accepted as well due to partitioning of protein to other tissues than lean, and due to an increase in lipid deposition accompanying an increase in protein deposition.

GENERAL DISCUSSION

In the general discussion, conclusions from the previous paragraphs were combined, and discussed in the light of a broader view on growth and development. Conclusions on the Linear-Plateau concept (mainly from the chapters I to IV) were combined into an alternative characterization of growing pigs. In the original approach, a pig is characterized with its protein deposition capacity (*PDmax*) and the minimal ratio between lipid and protein deposition (*r*). Especially the conclusion from chapter II that not *total,* but *extra* protein and lipid are closely related resulted in an alternative concept. This constant ratio between *extra* lipid deposition and *extra* protein deposition was called the marginal ratio (*mr*) and replaces the constant ratio (*r*). Furthermore, the presence of an effect of live weight on this ratio was discussed. This *mr* may differ between types of pigs. In the original characterization of pigs, performance could be calculated from the two parameters *PDmax* and *r*. However, the alternative parameter for *r* (*mr*) does not quantify an absolute level, but quantifies the effect of a change in energy intake. Therefore, a third parameter is needed, one which determines an absolute level. A proposed point for this in the

alternative characterization of pigs was the theoretical assumption of a common point for pigs at maintenance energy intake. Available data failed to confirm this, but there were indications that different pigs may produce similarly at very low energy intakes. As the presence of such a point could not be verified, another approach was adopted. It was proposed that for the type of pig to be characterized, a reference energy intake can provide a reference level of production. From this energy intake onwards, actual performance can be calculated using the parameters *PDmax* and *mr*. Some of proposed aspects of alternative characterization were implicitly adopted in other, previously published, concepts of growth in pigs. This verified the acceptability of the proposed parameters and urges the need to further evaluate them.

Possible methods to assess the required characterization parameters were shortly presented. First method is measuring protein and lipid deposition using the comparative slaughter technique. As this method is laborious and expensive, alternatives are desirable. The nitrogen balance is used often to assess performance at a certain energy intake. As this method does not assess protein deposition in a direct way, and as there is a substantial overestimation with this method, it is not really fit to determine an absolute level of performance in terms of protein and lipid deposition in pigs. Alternatively, frequently measured *vivo* data of pigs also allow assessment of performance characteristics. A provisional calculation effort indicated that performance of pigs in terms of protein and lipid deposition can be calculated rather accurately using biweekly collected data on energy intake data and changes in body weight. This method is based on the same assumptions as most growth models adopt, but the calculations are inverted. In growth models, growth is calculated from energy intake using an assumption on composition of growth. In the alternative method, growth and energy intake allow assessment of the composition of growth. So, the method is independent of assumptions made in the Linear-Plateau concept.

In the last part of the general discussion, partitioning of deposited tissue within the body was discussed. In the studied weight range (25-105 kg live weight), the majority of tissue is deposited into carcass LEAN and DEPOT. On average, LEAN tissue developed at a comparable rate as DEPOT tissue. These development rates were highly affected by energy intake and live weight. An increase in both of these factors affected DEPOT more than LEAN. Thus, even in the used pigs, boars of high genetic merit, there is a strong trend for the body to fatten as a result of an increase in energy intake. The partitioning of protein and lipid mass showed comparable trends as development of the physical tissue groups LEAN and DEPOT, respectively. The studies on partitioning clearly showed that with increasing energy, an increasing part of the protein is deposited into other tissues than lean. The high impetus for this was demonstrated by the fact that pigs which had considerably decreased protein masses due to a high feed intake still had more protein in the non-lean carcass parts. Furthermore, it was analytically derived in the general discussion that a linear relation between energy intake and protein deposition does not exclude a linear relation between energy intake and lean tissue deposition, despite the fact that with increasing energy, an increasing part of the protein is deposited into other tissues than lean.

Finally, a classical view on growth, as presented by the Cambridge School of Agriculture in the nineteen thirties and forties was compared to present findings. This comparison

showed that a concept on physiological age is close to a view on development of the body. Unexpectedly better performance of chronologically older and considerably fatter pigs as compared to control pigs in chapter IV could be explained by a lower physiological age. In the proposed view on physiological age (or degree of development), the amount of fat free mass plays a predominant role. This was illustrated with the very robust relation between protein mass and water mass.

The first major conclusion from the studies was that the Linear-Plateau concept, which demonstrates a gross nutritional principle well, is an oversimplification in quantitative terms. The minimal ratio between lipid and protein deposition, which was assumed to be constant is dependent on energy intake, body weight and nutritional history. Models adopting the Linear-Plateau concept should take this into account. A second major general conclusion is that a nutrition induced increase in protein deposition rate will be coincided with an increase in lipid deposition. Furthermore, such an increase in protein deposition will also result in a decreasing percentage of protein which is deposited into valuable body parts.

SAMENVATTING

ALGEMENE INLEIDING

'Verdeling' is het algemene thema van dit proefschrift. Twee aspecten van groei van varkens die te maken hebben met 'verdeling' zijn bestudeerd. Allereerst de verdeling van produktie-energie tussen eiwit- en vetaanzet. Dit is bestudeerd aan de hand van een evaluatie van een theorie hierover, het Lineair-Plateau concept. Het tweede aspect van verdeling is de verdeling van aangezet eiwit en vet in het lichaam van varkens. Dit is van belang voor de relatie tussen de anatomische en chemische lichaamssamenstelling.

De lichaamssamenstelling van varkens wordt beïnvloed door vele factoren. Eén hiervan is de voeding. Omdat de factoren niet onafhankelijk zijn, zal beschrijving van de groei van varkens multi-factorieel moeten zijn. Er zijn verscheidene benaderingen gepubliceerd in de literatuur omtrent het berekenen van de produktie van varkens uit gegevens over dier, voeding en omgeving. Een belangrijke ontwikkeling in het relateren van weefselaanzet aan de voeding was het Lineair-Plateau concept. Hierin wordt de relatie tussen energieopname en eiwit- en vetaanzet beschreven voor varkens.

Het Lineair-Plateau concept veronderstelt dat eiwitaanzet lineair toeneemt met een toename in energieopname, tot aan een maximale eiwitaanzet, waarna de relatie een plateau laat zien. Onder deze maximale eiwitaanzet is er een minimale hoeveelheid vetaanzet voor elke hoeveelheid eiwitaanzet. Er is dus (bij niet-maximale eiwitaanzet) een konstante minimale verhouding tussen de vetaanzet en de eiwitaanzet. Bij energieopnames die voldoende zijn om de maximale eiwitaanzet te realiseren wordt alle energie die niet benodigd is voor eiwitaanzet en de minimale vetaanzet gebruikt voor vetaanzet. De maximale eiwitaanzet (*PDmax*) en de minimale verhouding tussen vet- en eiwitaanzet (*r*) worden konstant verondersteld voor het hele mesterijtrajekt.

VALIDATIE VAN EEN GROEIMODEL

In hoofdstuk I is een groeimodel gevalideerd waarin het genoemde Lineair-Plateau concept ingebouwd is. Aan varkens van twee verschillende lijnen werd een rantsoen verstrekt dat eiwit-deficiënt (50% van de norm) was of er werd een adequaat rantsoen op één van twee voerniveaus aangeboden. Gemeten waarden van groei, voederconversie, chemische lichaamssamenstelling bij het slachten en aanzetten van eiwit en vet werden vergeleken met door het model berekende waarden. In het algemeen voorspelde het model de effekten van de voedingsbehandelingen voldoende. Vooral eiwitaanzet en groei werden goed voorspeld. De vetaanzet boven 65 kg lichaamsgewicht werd echter 20-30% onderschat. De resultaten lieten een stijging zien in de verhouding tussen vet- en eiwitaanzet met toenemend lichaamsgewicht. Het model hield geen rekening met zo'n effekt van lichaamsgewicht op de samenstelling van de aanzet. In tegenstelling tot de eiwit-adequaat gevoerde dieren, werd de vetaanzet van de eiwit-deficiënt gevoerde dieren overschat. Bij deze dieren werd de eiwitaanzet goed berekend. Uit de resultaten bleek voorts dat de hoeveelheid aangezet water per hoeveelheid aangezet eiwit hoger was dan voorspeld door het model. De belangrijkste konklusie van de validatie was dat de

parameter die de minimale verhouding tussen vet- en eiwitaanzet kwantificeert afhankelijk is van lichaamsgewicht.

ENERGIEOPNAME EN ENERGIEVERDELING

In hoofdstuk II zijn gegevens bestudeerd uit de literatuur die de lineaire relatie tussen energieopname en eiwitaanzet aantoont. Doel was om na te gaan of deze data de tweede aanname (een konstante verhouding tussen vet- en eiwitaanzet bij eiwitaanzetten onder *PDmax*) ondersteunen. Uit de studie bleek dat niet alleen eiwitaanzet, maar ook vetaanzet lineair gerelateerd is met de energieopname. De verhouding tussen vet- en eiwitaanzet steeg echter met elke toename van de energieopname. Deze studie plaatste dus vraagtekens bij één van de twee aannames van het Lineair-Plateau concept, de konstantheid van *r*. In de diskussie werd afgeleid dat er inderdaad een konstante relatie bestaat tussen de vetaanzet en de eiwitaanzet. Er is echter geen konstante verhouding tussen *totale* vet- en eiwitaanzet, maar tussen *extra* vet- en eiwitaanzet. Deze extra aanzet is de aanzet veroorzaakt door een extra eenheid energieopname. Elke toename in energieopname veroorzaakt een vettere aanzet omdat de verhouding tussen *extra* vet- en eiwitaanzet hoger is dan de verhouding tussen *totale* vet- en eiwitaanzet.

LICHAAMSGEWICHT, ENERGIEOPNAME EN ENERGIEVERDELING

Hoofdstuk I en II nuanceerden beiden de konstantheid van de verhouding tussen eiwit- en vetaanzet. Effekten van lichaamsgewicht en van energieopname werden aangetoond. Deze factoren werden in hoofdstuk III gezamenlijk bestudeerd in een experiment met beren. De proefopzet was zodanig dat het effekt van energieopname onafhankelijk was van het lichaamsgewicht. De resultaten lieten zien dat de verhouding tussen vet- en eiwitaanzet steeg met toenemend lichaamsgewicht op beide energieopnameniveaus. Bij de LOW behandeling steeg deze verhouding van 0.74 op 25 kg lichaamsgewicht naar 0.99 op 105 kg lichaamsgewicht. Voor varkens op de HIGH behandeling steeg de verhouding tussen vet- en eiwitaanzet van 0.82 naar 1.35 in het genoemde gewichtstrajekt. Gekonkludeerd werd dat zowel lichaamsgewicht als energieopname de verhouding tussen vet- en eiwitaanzet beïnvloedt. Er was geen signifikante interaktie tussen het effekt van energieopname en lichaamsgewicht, maar de illustraties gaven een sterkere toename van de ratio met het lichaamsgewicht aan voor de HIGH dieren vergeleken met de LOW dieren. Dit betekent dat de door de voeding veroorzaakte vervetting vooral op hogere gewichten plaatsvindt.

VOEDINGSVERLEDEN EN ENERGIEVERDELING

Het effekt van een voormalige voedingsbehandeling op de verdeling van de produktie-energie werd geïllustreerd met een experiment beschreven in hoofdstuk IV. Tussen 28 en 65 kg lichaamsgewicht werd aan varkens ofwel een eiwit-adequaat rantsoen *ad libitum* verstrekt, of een sterk eiwit-deficiënt rantsoen. Tussen 65 en 105 kg lichaamsgewicht werd aan alle dieren hetzelfde eiwit-adequate rantsoen verstrekt. Tot 65 kg lichaamsgewicht veroorzaakte de eiwit-deficiëntie een sterke verlaging van de voeropname, groei en van de aanzetten van eiwit en vet. De eiwitbeperkt gevoerde dieren hadden op 65 kg twee maal zoveel lichaamsvet en waren gemiddeld 60 dagen ouder dan de kontroledieren. Na overschakeling op een adequaat rantsoen (na 65 kg lichaamsgewicht) lieten de voordien

beperkte dieren een licht (7%) verlaagde voeropname zien en 15% toename van de groei. Het belangrijkste verschil was de beïnvloeding van de samenstelling van de groei door de eerdere eiwitbeperking. De voormalig beperkte dieren waren veel vetter dan de kontroledieren, maar de samenstelling van hun weefselaanzet voorbij 65 kg was veel magerder. Tussen 65 en 105 kg lichaamsgewicht was de verhouding tussen vet- en eiwitaanzet gelijk aan 1.23 voor deze varkens, terwijl deze voor de kontrolevarkens 1.69 bedroeg. In feite leek de groeisamenstelling van de voormalig eiwitbeperkte dieren op die van beduidend lichtere jongere dieren. Uit het experiment kan gekonkludeerd worden dat voedingsverleden een invloed kan hebben op de verdeling van de produktie-energie tussen eiwit- en vetaanzet.

VERDELING VAN AANGEZET WEEFSEL IN HET LICHAAM

In hoofdstuk V zijn de effekten van gewicht en energieopname op de plaats van eiwit- en vetaanzet bestudeerd. De aanzet van eiwit en vet werd gekwantificeerd in 3 weefselgroepen in twee gewichtstrajekten, van 25 tot 65 kg lichaamsgewicht en tussen 65 en 105 kg lichaamsgewicht. De drie weefselgroepen waren: LEAN (uitgesneden magere delen inclusief ingesloten botten, volgens de IVO-snit), DEPOT (overig karkasweefsel) en niet-karkasdelen (ORGANS inclusief bloed). Gemiddeld werd 57% van de totale eiwitaanzet in LEAN aangezet, en 68% van de totale vetaanzet in DEPOT. Een toename van de energieopname verhoogde zowel de eiwit- als de vetaanzet. Van deze *extra* eiwitaanzet en *extra* vetaanzet werd respectievelijk 42% en 75% in de niet-karkasdelen (DEPOT) aangezet in het gewichtstrajekt 25-65 kg. Boven 65 kg lichaamsgewicht waren deze percentages respectievelijk 71% en 82%. Dus werd, ondanks de relatief lage opnameniveaus, het grootste deel van het extra weefsel aangezet in niet-LEAN karkasweefsel. Het effekt van een 40 kg toename in lichaamsgewicht was kleiner dan het effekt van een toename van de energieopname met 3.7 MJ DE (ongeveer 250 gram voer extra). Bij het toegepaste voerregime werd de verdeling van eiwit binnen het lichaam dus vooral door de voeding bepaald. Gekonkludeerd werd dat, om de vleesaanzet te verhogen met behulp van de voeding, een behoorlijke vervetting van het lichaam zal moeten worden geaccepteerd. Dit wordt veroorzaakt door verhoging van de aanzet van eiwit in andere weefsels dan vlees, en door een toename van de vetaanzet met een verhoging van de eiwitaanzet.

ALGEMENE DISKUSSIE

In de algemene diskussie zijn de konklusies gekombineerd en bediskussieerd in het licht van een bredere blik op groei en ontwikkeling. Konklusies omtrent het Lineair-Plateau concept (vooral uit hoofdstuk I - IV) zijn gekombineerd in een alternatieve karakterisering van groeiende varkens. In de oorspronkelijke benadering werd een varken gekarakteriseerd met zijn eiwitaanzetkapaciteit (*PDmax*) en de minimale verhouding tussen vet- en eiwitaaanzet (*r*). Met name de konklusie uit hoofdstuk II dat niet de *totale*, maar de *extra* eiwit- en vetaanzet nauw gerelateerd zijn had een alternatief concept tot gevolg. Deze konstante verhouding tussen *extra* vet en *extra* eiwitaanzet werd de marginale verhouding (*mr*) genoemd, en verving de konstante verhouding (*r*). Bovendien werd het effekt van lichaamsgewicht op deze ratio bediskussieerd. Deze *mr* kan verschillen tussen verschillende types varkens. In de oorspronkelijke karakterisatie van varkens kon de produktie berekend worden uit de twee parameters *PDmax* en *r*. De alternatieve parameter *mr* kwantificeert

geen absoluut niveau, maar het effekt van de verandering van de energieopname. Daarom is een derde parameter nodig, één die een absoluut niveau kwantificeert. Een voorstel hiertoe in de alternatieve karakterisering was de theoretische verwachting van een gezamenlijk punt voor varkens bij een energieopname ter hoogte van de energie-onderhoudsbehoefte. De beschikbare gegevens konden dit niet bevestigen, al waren er wel aanwijzingen dat verschillende types varkens vergelijkbaar produceren bij zeer lage energieopnames. Omdat het bestaan van zo'n punt niet bevestigd kon worden, is een andere benadering gevolgd. Voorgesteld is om met behulp van een referentie-energie-opname een referentie-produktie te bepalen. Vanuit dit referentiepunt kan dan de feitelijke produktie berekend worden met behulp van *PDmax* en *mr*. Enkele aspecten van de alternatieve karakterisering waren voordien al impliciet aangenomen in andere concepten omtrent groei van varkens. Dit bevestigde de aanvaardbaarheid van de voorgestelde parameters, en benadrukt de noodzaak ze verder te evalueren.

Mogelijke methoden om de alternatieve parameters te bepalen zijn kort behandeld. De eerste methode om deze te bepalen is de vergelijkende slachtmethode. Daar deze methode arbeidsintensief en duur is, zijn alternatieven wenselijk. De stikstofbalanstechniek wordt vaak gebruikt om produktie-eigenschappen op een bepaald energieniveau te bepalen. Deze methode bepaalt de eiwitaanzet op een indirekte manier, en geeft in het algemeen een substantiële overschatting van de eiwitaanzet. Daarom is de stikstofbalansmethode niet erg geschikt om het absolute niveau van de eiwit- en vetaanzet te bepalen. Een andere methode om de produktie te karakteriseren is het gebruik van frequent gemeten *vivo* data van varkens. Een voorlopige rekenexercitie gaf aan dat de produktie van varkens met betrekking tot eiwit- en vetaanzet vrij nauwkeurig bepaald kan worden uit gegevens over energieopname en verandering van lichaamsgewicht. Deze methode is gebaseerd op dezelfde aannames als de meeste groeimodellen, maar de berekeningen zijn omgekeerd. In groeimodellen wordt de groei berekend uit de energieopname met een aanname omtrent de samenstelling van de groei. In de alternatieve methode wordt uit de groei en de energieopname de samenstelling van de groei berekend. Daarom is deze methode onafhankelijk van de aannames in het Lineair-Plateau concept.

In het laatste deel van de algemene diskussie is de verdeling van aangezet weefsel in het lichaam bediskussieerd. In het bestudeerde gewichtstrajekt (25-105 kg lichaamsgewicht) werd het grootste deel van het weefsel aangezet in karkas LEAN en DEPOT. Gemiddeld ontwikkelde de weefselgroep LEAN zich met vergelijkbare snelheid als de weefselgroep DEPOT. Deze ontwikkelingssnelheden werden sterk beïnvloed door energieopname en lichaamsgewicht. Een toename van deze factoren had een groter effekt op DEPOT dan op LEAN. Dit betekent dat er een sterke aanleg is om te vervetten als gevolg van voedings-manipulaties, zelfs in de gebruikte beren met een hoge genetische kapaciteit. De verdeling van eiwit en vet was vergelijkbaar met de ontwikkeling van de respectievelijke weefsel-groepen LEAN en DEPOT. De studie naar weefselverdeling liet duidelijk zien dat een toenemend gedeelte van het eiwit in niet-LEAN karkasdelen aangezet wordt met een toename van de energieopname. De sterke neiging hiertoe werd geïllustreerd door het feit dat varkens met een aanzienlijk verlaagde eiwitmassa door een hoge voeropname toch meer eiwit hadden in niet-LEAN karkasdelen. Vervolgens is analytisch afgeleid dat de lineaire relatie tussen energieopname en eiwitaanzet niet uitsluit dat er een lineaire relatie bestaat tussen energieopname en vleesaanzet. Dit ondanks het feit dat een toenemend

gedeelte van het eiwit aangezet wordt in andere weefsels dan LEAN met een toename in energieopname.

Tenslotte is een klassieke visie op groei, zoals gepresenteerd door de Cambridge School of Agriculture in de dertiger en veertiger jaren van deze eeuw, vergeleken met de huidige uitkomsten van onderzoek. Deze vergelijking gaf aan dat een concept omtrent fysiologische leeftijd gerelateerd is aan ontwikkeling van het lichaam. De onverwachte gunstiger weefselaanzet van chronologisch oudere en aanzienlijk vettere dieren in hoofdstuk IV kon worden verklaard door een lagere fysiologische leeftijd. In de visie op fysiologische leeftijd (of mate van ontwikkeling) speelt de hoeveelheid vetvrij weefsel een belangrijke rol. Dit werd geïllustreerd met de robuuste relatie tussen eiwitmassa en watermassa.

De eerste belangrijke konklusie uit de studies was dat het Lineair-Plateau concept, dat geschikt is om een voedingsprincipe te illustreren, een oversimplificatie in kwantitatief opzicht is. De minimale verhouding tussen vet- en eiwitaanzet, die konstant verondersteld was, hangt af van energieopname, lichaamsgewicht en voedingsverleden. Modellen die het Lineair-Plateau concept gebruiken zouden hier rekening mee moeten houden. Een tweede belangrijke konklusie is dat een toename in de eiwitaanzet, veroorzaakt door de voeding, samengaat met een toename in de vetaanzet. Bovendien zal zo'n toename ook resulteren in een verlaging van het percentage eiwit dat aangezet wordt in waardevolle lichaamsdelen.

CURRICULUM VITAE

Karel Hendrik de Greef werd op 11 juli 1963 in Woudenberg geboren. In 1981 behaalde hij het VWO diploma aan het Corderius College te Amersfoort. Dit diploma werd in 1982 aangevuld met de deelcertificaten natuurkunde en scheikunde. In datzelfde jaar werd de studie Zoötechniek aan de toenmalige Landbouwhogeschool aangevangen. Deze studie werd in januari 1988 afgesloten met afstudeervakken op het gebied van de veehouderij en veevoeding. Van augustus 1987 tot en met februari 1988 werkte hij bij de vakgroep Veefokkerij van de Landbouwuniversiteit aan de uitvoering en verwerking van een experiment omtrent het effekt van recombinant somatotropine op produktie- en vleeskwaliteitseigenschappen bij vleesvarkens. Per 1 maart 1988 werd hij als AIO aangesteld bij de vakgroep Veevoeding. In het kader hiervan is onderzoek verricht aan de simulatie van groei van varkens en de beïnvloeding van de lichaamssamenstelling van vleesvarkens.