NUTRIENT REQUIREMENTS OF PIGS TREATED WITH METABOLIC MODIFIERS

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Summary

Recent advances in the biotechnology industries have resulted in two classes of metabolic modifiers, the somatotropins and ß-agonists, which are potentially available to manipulate growth and reduce carcass fat in the pig and other domestic animals. These metabolic modifiers stimulate protein deposition in the pig, particularly during the finishing (fattening) phase of growth. Porcine somatotropin (pST) decreases fat deposition and feed intake whereas the ß-agonists have relatively little effect on fat deposition and feed intake. Increases in protein deposition during pST treatment of young pigs (<60 kg) are due to increased efficiency of use of dietary protein. In finisher pigs, increases in protein deposition during either pST or ß-agonist treatment require a commensurate increase in dietary protein content. Although pST and the ß-agonists increase protein deposition across a wide range in energy intakes, ad libitum feed intakes are necessary to maximise responses.

I. INTRODUCTION

The 1983 National Dietary Survey of Adults indicated that total fat and saturated fatty acids represented 37 and 15% respectively of total energy consumption whereas current recommendations are that only 30 and 10% of total energy should be supplied as total and saturated fat respectively (NH&MRC 1992). As a result of these recommendations and the publicised associations between dietary fat and disease consumers, and the nutrition community are becoming more concerned about fat in the food they eat. Since animal products constitute about 60% of the fat available for consumption in Australia (ABS 1991) there is pressure on animal industries to reduce the fat content of meat and meat products. Simultaneously, there has been a desire on-farm to produce animals which are leaner since it is well recognised that the food conversion efficiency (FCE) is inversely related to the ratio of fat to protein deposited. While genetic selection programs combined with sophisticated feed formulations have resulted in considerable improvement in FCE, progress using current reproductive technology is slow. Recent advances in the genetic engineering and agro-chemical industries have resulted in two classes of metabolic modifiers, recombinant porcine somatotropin (pST) and the ß-agonists, both of which alter the ratio of fat to protein deposition. Animals treated with either of these metabolic modifiers exhibit improved FCE and have leaner carcasses at slaughter. The mechanisms involved are markedly different and have been the subject of recent reviews (Boyd and Bauman 1989; Etherton 1990; Reeds and Mersmann 1991; Dunshea 1993).

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The aim of this paper is to review the effects of these metabolic modifiers on the protein and energy requirements of pigs. Effects of these metabolic modifiers on meat quality, particularly with respect to fat content, will also be discussed. This review is timely since one of these metabolic modifiers, pST, has recently been approved for commercial use in Australia.

II. NUTRITIONAL CONSTRAINTS TO PROTEIN DEPOSITION

To determine the nutrient requirements of pigs treated with metabolic modifiers it is important to understand first of all the interrelationships between nutrient supply (protein and energy in this instance) and protein deposition in the pig. Recent reviews discuss these principles (SCA 1987; Campbell 1988) so they will be reviewed only briefly in this paper.

(a) Interrelationships between dietary protein and energy on protein deposition

Theoretical responses to dietary protein intake are shown in Fig. 1. Fig. 1a demonstrates the two phases of protein deposition: (i) an initial protein-dependent phase where protein deposition increases linearly with protein intake regardless of energy intake and (ii) an energy-dependent phase in which protein deposition only increases if additional energy is provided. Thus, when pigs of a given weight (sex, genotype, etc.) are fed increasing amounts of a protein of constant composition at a set level of energy intake (E_1), protein deposition increases linearly until a maximum (M_1) is reached at a protein intake of P_1. Beyond this protein intake the rate of protein deposition will only increase if additional energy is provided (ie. energy-dependent phase). If extra energy is provided (E_2) protein deposition increases linearly up to a new plateau at a higher protein intake (P_2). If the intrinsic ceiling for protein deposition is M_2, then neither extra energy nor protein will further increase protein deposition. However, the ratio of protein:energy required to maximise protein deposition will decrease. While these theoretical relationships have been confirmed for a very homogeneous group of pigs (see SCA 1987), a normal population of pigs contains individual pigs which exhibit individual linear/plateau responses. When these individual response curves are combined the population response may be curvilinear (Fig. 1b). Fitting a curvilinear rather than linear/plateau model to these data generally has the effect of increasing the estimate of protein requirement (eg. from P1 and P2 to P1'and P2' respectively). While this often raises concerns in the interpretation of experimental data, as often both model types will describe the data equally well, the interpretation of the biology of the system is generally the same.

The slope of the ascending portion of the curves depicted in Fig. 1 is the efficiency with which dietary protein is deposited. This slope is determined by the digestibility of dietary protein and by how well the pattern of absorbed amino acids matches the pattern of requirements for tissue deposition and maintenance. When considering protein requirements of the pig it should be borne in mind that the pig does not have a requirement for protein per se but rather for an appropriate level and balance of individual amino acids, particularly the essential amino acids. In an effort to ease the complexity of diet formulation the ARC

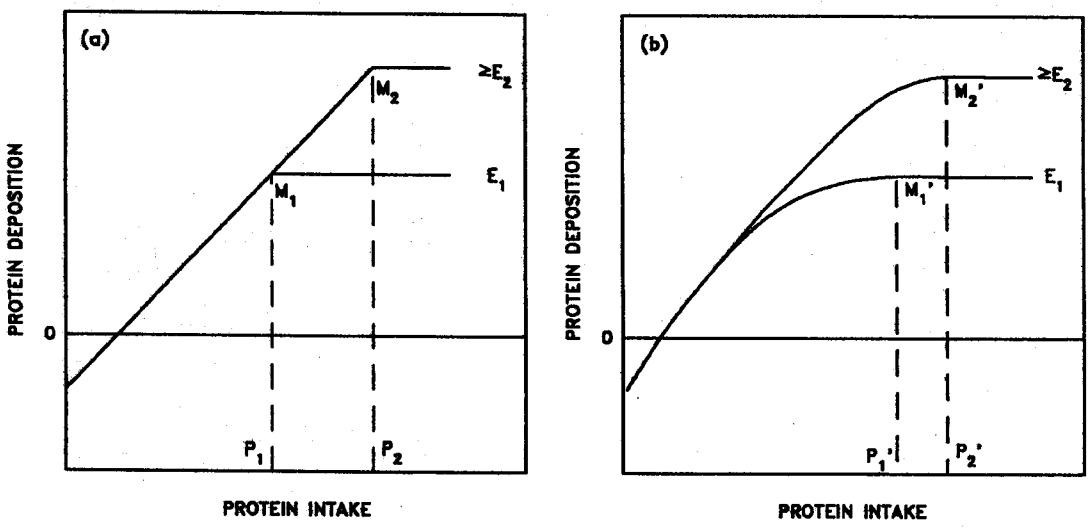

Fig. 1. Relationship between protein deposition and dietary protein intake fitted to either a (a) linear/plateau or (b) curvilinear model.

(1981) adopted the concept of the "ideal" protein. Since amino acids are used predominantly for protein deposition the composition of the ideal protein closely resembles that of muscle (Wang and Fuller 1989). The concept of ideal protein has to some extent successfully shifted the focus of feed manufacturers and producers away from individual amino acid requirements (eg. lysine, threonine) towards thinking in terms of an adequate balance of amino acids in the diet. As an animal grows the composition of the ideal protein also changes as the usage (maintenance versus growth) and source of amino acids change. Metabolic modifiers likely change the ratio of amino acids deposited to amino acids used for maintenance and this will effect the pattern of amino acids required. Nevertheless, these will for the large part be subtle changes and in the experiments outlined below the pattern of amino acids used were similar to that proposed as "ideal" by the ARC (1981) and SCA (1987). The concepts of changing patterns of amino acid requirements have been incorporated into the AUSPIG model (Black et al. 1986).

(b) Effect of energy intake on protein deposition under conditions of protein adequacy

Knowledge of the relationship between protein deposition and energy intake is crucial to determining optimum feeding strategies for different classes of pigs. These relationships are outlined in Fig. 2. In this model, total energy deposition increases linearly with increasing energy intake (Fig. 2a). Energy retained as protein also increases linearly up to a maximum at an energy intake of Q, beyond which further energy has no effect on the rate of protein deposition. Fat deposition also increases linearly until an energy intake of Q after which a sharp increase in the rate of fat deposition occurs. At zero energy balance protein gain is still marginally positive while fat deposition is negative. Fat deposition does not commence until energy intake reaches a somewhat higher level of intake (R). The potential impact of energy intake upon body composition is very much related to what intake, if any, for a particular pig corresponds to Q. This is perhaps more clearly

demonstrated in Fig. 2b which depicts the ratio of fat to protein in tissue gain. While protein deposition increases linearly with energy intake, the ratio of fat to protein deposited increases curvilinearly, rising steeply initially before approaching an asymptote. However, once a plateau in protein deposition is reached there is a sharp linear increase in the ratio of fat to protein deposited (beyond energy intakes of Q). Therefore, whether protein deposition continues to respond linearly up to the limit of appetite or reaches a plateau at an intermediate energy intake can have profound effects upon the composition of weight gain, body composition and FCE.

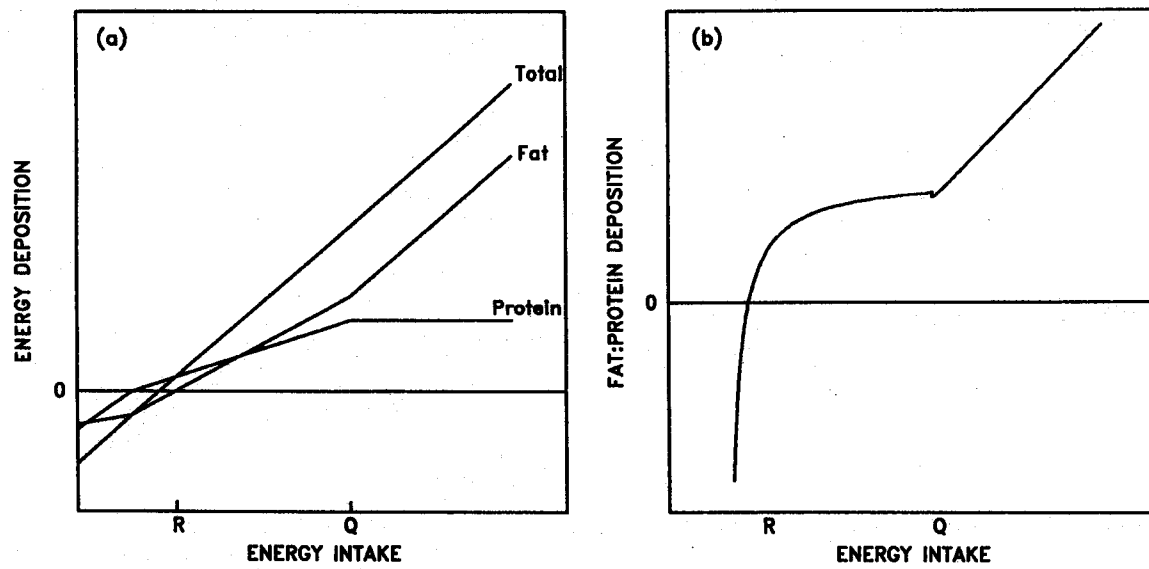

Fig. 2. Interrelationships between energy intake on (a) deposition of protein, fat and total energy, and (b) the corresponding ratio of fat:protein in tissue gain (adapted from SCA, 1987).

III. EFFECT OF METABOLIC MODIFIERS ON NUTRIENT REQUIREMENTS

Metabolic modifiers can have a number of different impacts on the relationships outlined in Fig. 1 and 2. They alter the actual rates of deposition of protein and/or fat and can also alter feed intake, nutrient digestibility, maintenance energy requirements and the efficiency of use of dietary nutrients. In turn, changes in any of these aspects of metabolism can influence both the slopes and response of the relationships outlined earlier.

(a) Porcine somatotropin (pST)

(1) Production responses Exogenous pST treatment consistently improves average daily gain (ADG) and FCE (Etherton et al. 1987; Campbell et al. 1988,1989,1990a,b,1991). In slaughter-balance studies, dose-dependent increases in protein deposition and reductions in fat deposition and carcass fat have been observed (Boyd et al. 1986,1991; Etherton et al. 1987; Evock et al. 1988). Porcine somatotropin increases protein deposition and decreases fat deposition in males, females and castrates (Campbell et al. 1989) of both poor and

improved genotypes (Campbell et al. 1990a,1991). Although the greatest responses occur in finisher pigs (60 to 100 kg), exogenous pST also improves performance in younger pigs (Campbell et al. 1988,1990b; Boyd et al. 1991). As a result of the reduction in fat deposition there is a consistent dose-dependent reduction in feed intake (Boyd and Bauman 1989). This reduction is sometimes associated with improved digestibility (Wray-Cahen et al. 1991) although these differences disappear when pigs are pair-fed (Verstegen et al. 1990).

(2) Effects on meat quality Subcutaneous adipose tissue is the major site of fat synthesis and deposition in the pig. A major effect of exogenous pST treatment is a dramatic reduction in de novo fat synthesis (Dunshea et al. 1992) and consequently subcutaneous fat depth can be decreased by up to 70% (Krick et al 1992). While subcutaneous fat is trimmed from many pork cuts (eg. loin chops), it is still retained in roast portions and skin-on bacon and the nutritive value of these latter products will be enhanced by pST. In addition, these cuts of meat contain more lean since both loin-eye area and ham weight are increased by pST treatment (McKeith 1993). The level of intramuscular fat also decreases during pST treatment. This is important because this type of fat cannot be removed by trimming. Subjective fat marbling scores are consistently reduced by pST while chemically extractable intramuscular fat in the loin is reduced from approximately 3 to 2 g of fat per 100 g meat (Prusa et al. 1989; McKeith 1993). The fatty acid composition of both subcutaneous and intramuscular fat is relatively unchanged by pST treatment although the dramatic reduction in fatty acid synthesis offers the opportunity to manipulate fatty acid composition through dietary manipulation. Slight or no effects of pST on objective and subjective measures of meat tenderness, appearance and shelf life have been reported (McKeith, 1993; Wander et al. 1993).

(3) Responses to dietary protein Since there is relatively little effect of pST upon protein digestibility, the increased protein deposition observed in pST treated pigs must be due to either an increase in the efficiency of use of dietary protein and/or an increase in the requirement of dietary protein to support the increased protein deposition. In the grower pig (30-60 kg) it appears that pST has very little if any effect on dietary protein requirements but there is an increase in the partial efficiency of utilisation of dietary protein (Campbell et al. 1990; Caperna et al. 1990; Krick et al. 1993). For example, Campbell et al (1990b) conducted an experiment where grower pigs were fed a fixed amount (digestibility unchanged) of diets containing varying levels of "ideal" protein and were also treated with pST (Fig. 3). Regardless of the model used to interpret the data, these data suggest only a marginal increase in the dietary protein requirement but a substantial increase in the efficiency of use of dietary protein. Therefore, it appears that conventional grower diets (17-18% crude protein based on the linear/plateau model) may be sufficient to allow the expression of the benefits of pST in grower pigs (25-60 kg). In this class of pigs, energy is more likely to be limiting than protein.

The effects of pST on the protein requirements of finisher pigs (60-90 kg), in which increases in protein deposition are greater, are more equivocal. Campbell et al. (1991) fed finisher male pigs (60-90 kg) diets restricted to 36 MJ DE/d but varying in "ideal" protein from 7 to 23% and treated them with 0 or 100 μg/kg pST. They found that there was no change in the efficiency of use of dietary protein in pST treated-pigs but that the dietary protein requirements increased from 10.6 to 18.1% to support an increase in protein deposition from 119 to 215 g/d (Fig. 4).

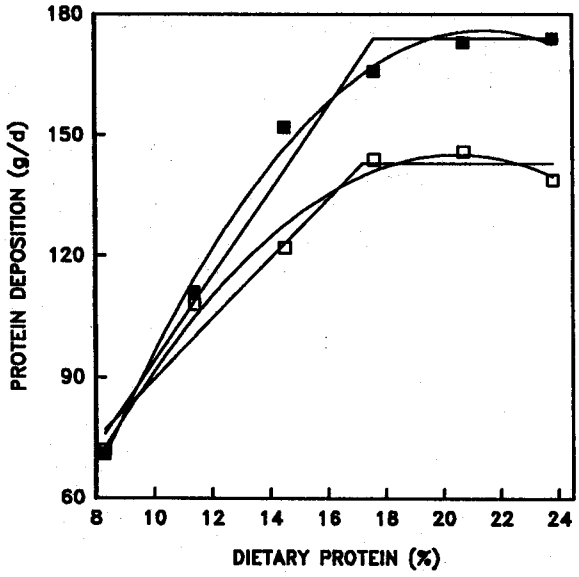

Fig. 3. Relationship between protein deposition and dietary protein content in pigs treated with 0 (□) or 90 (■) μg/kg pST between 30 and 60 kg (Campbell et al. 1990b).

However, in another experiment both the efficiency of use of dietary lysine (formulated to be the first limiting amino acid) and the lysine requirement were increased in castrate male and female pigs treated with pST (Fig. 5; Boyd et al. 1991). Differences in response may reflect the relative efficiencies with which entire male, castrate male and female pigs deposit dietary protein. Thus, in the study by Campbell et al. (1991) conducted in entire males, the control animals were already depositing dietary protein with an efficiency of 62% and pST did not improve it. In the study reported by Boyd et al. (1991), involving castrate males and females, the efficiency of utilisation was lower (40%). Treatment with pST increased efficiency of use of dietary protein to 60%, similar to that found by Campbell et al. (1991). Interestingly, the efficiency of use of dietary protein in the castrate and female pigs of Boyd et al. (1991) were similar to that observed in female pigs of the same genotype used by Campbell et al. (1991) (38%; Dunshea et al. 1993b). Therefore, in improved finishing male pigs which already retain dietary protein with a high efficiency, improvements in protein deposition during pST treatment may only be sustained by increasing dietary protein intake. Conversely, in finisher castrate or female pigs with lower efficiency of use of dietary protein, pST treatment can increase both the efficiency of use of dietary protein and the dietary protein requirement.

(4) Responses to dietary energy It is generally accepted that an increase in protein deposition is associated with increased maintenance energy requirements (MER), and this has been borne out by differences between pigs with higher and lower genetic merit in their capacity to deposit protein and their MER (Campbell and Taverner 1988). Indirect calorimetry confirmed that there is a modest increase (12%) in MER in castrate pigs treated with pST (Verstegen et al. 1990). Given the increased MER and the considerable decrease in feed intake which occur during pST treatment, it is possible that dietary energy intake may limit responses to pST.

The relationship between energy intake and protein deposition is linear in the grower pig fed protein adequate diets (see II (b) above). This was confirmed in the study of Campbell et al. (1988) who demonstrated that protein deposition increased with energy intake in young pigs treated with 0 or 100 μg/kg of pST (Fig. 6). At every level of energy intake, protein deposition was higher, and fat and total energy deposition lower, in the pigs treated with pST. Extrapolation of the relationship between total energy deposition and energy intake to zero energy retention suggests that MER may be increased by 8% by pST. Note also that ad libitum feed intake was reduced by pST. Therefore, in order to gain the maximum benefit from treating grower pigs with pST it is necessary to ensure that feed intake is maximised.

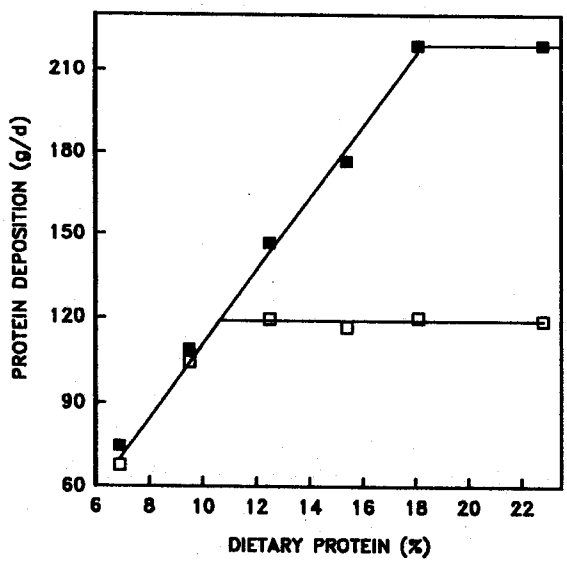

Fig. 4. Relationship between protein deposition and dietary protein content in finisher boars treated daily with 0 (□) or 100 μg/kg pST (■) (after Campbell et al. 1991)

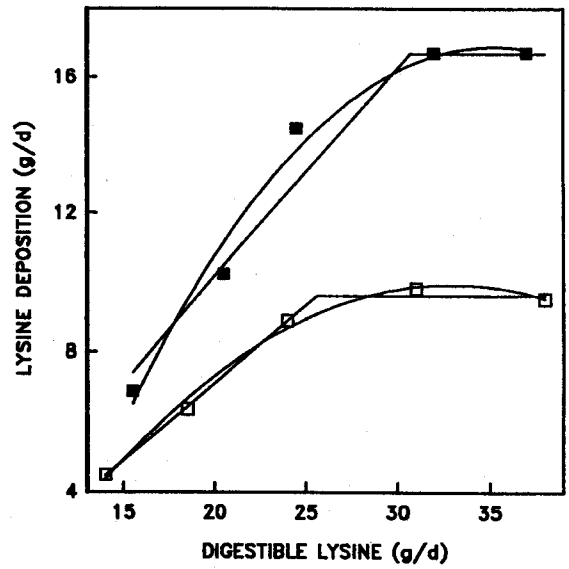

Fig. 5. Relationship between lysine deposition and dietary lysine intake in finisher female and castrate pigs treated daily with 0 (□) or 150 μg/kg pST (■) (after Boyd et al. 1991)

In older pigs the relationship between energy intake and protein deposition is more typically a linear-plateau one (Fig. 2a). Campbell et al. (1991) reported data depicting the relationship between protein deposition and energy intake in entire male and female pigs treated with 0 or 90 μg/kg of pST over the weight range of 60-90 kg. The control female pigs exhibited the expected linear-plateau relationship with protein deposition plateauing at 112 g/d (2.7 MJ/d), at an energy intake of approximately 30 MJ DE/d (Fig. 7). While female pigs treated with pST still exhibited the linear-plateau response in protein deposition to energy intake, they had increased protein deposition at every level of energy intake. Protein deposition plateaued at 203 g/d (4.8 MJ/d) at an energy intake of 33 MJ DE/d. Ad libitum feed intake was decreased by almost 20%. In addition, MER was increased from

12.4 to 15.8 MJ DE/d (+27%). For control male pigs there was the expected linear-plateau relationship between protein deposition and energy intake whereas for pST-treated males the relationship was linear (see Campbell et al. 1991). Therefore, while individually-penned contemporary female and castrate male pigs consume energy in excess of that required to maximise protein deposition, if the full benefits of exogenous pST are to be achieved commercially then feed intake needs to be maximised.

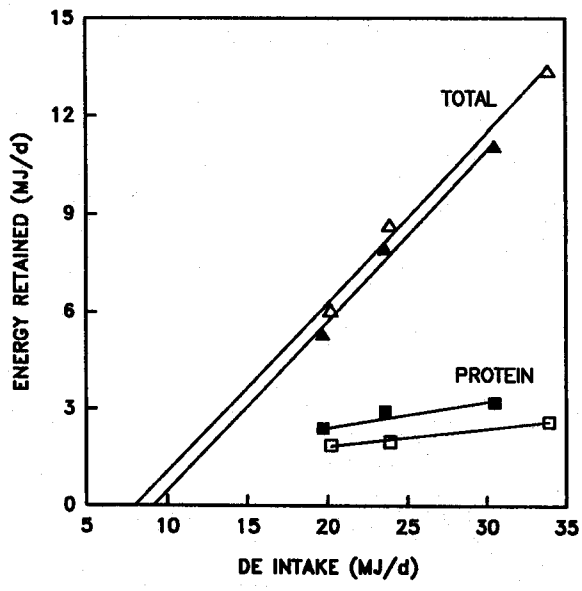

Fig. 6. Relationship between protein energy (□,■) and total energy (▽,▼) retained in pigs treated daily with 0 (open symbols) or 100 µg/kg pST (closed symbols) between 30 and 60 kg (Campbell et al. 1988)

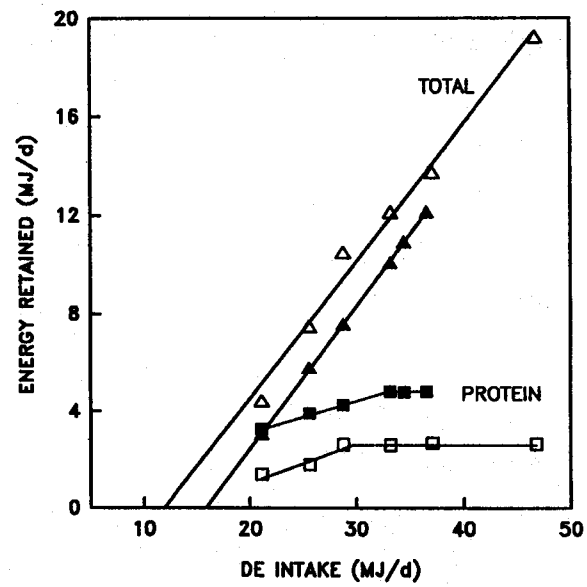

Fig. 7. Relationship between protein energy (□,■) and total energy (▽,▼) retained in female pigs treated daily with 0 (open symbols) or 100 µg/kg pST (closed symbols) between 60 and 90 kg (Campbell et al. 1991).

(b) ß-agonists

(1) Production responses Treatment of pigs with ß-agonists, particularly ractopamine (RAC), generally has resulted in dose-dependent increases in ADG, FCE and carcass lean content (see Dunshea 1991,1993). Feed intake is typically unchanged (Adeola et al. 1990; Gu et al. 1991; Yen et al. 1991) or decreased slightly (Adeola et al. 1990; Watkins et al. 1990; Mitchell et al. 1991) during ß-agonist treatment. While there is general agreement that protein deposition is increased during ß-agonist treatment, effects on fat deposition have been more equivocal. For example, while RAC increased protein deposition in entire male, female and castrate male pigs of an improved genotype by 15, 42 and 41% respectively, there was little effect on fat deposition (Dunshea et al. 1993a). Other ß-agonists which have improved performance in finisher pigs are salbutamol, cimaterol, clenbuterol and Ro 16-8714 (see Dunshea 1991,1993).

(2) Effects on meat quality Dietary RAC and other ß-agonists have little effect on the rate of fat deposition in pigs, presumably due to a rapid down-regulation of adipocyte ß-receptors (Dunshea 1993). Despite this, subcutaneous fat depth is often reduced since there is an increase in lean tissue deposition (ie. dilution effect). Therefore, dietary RAC supplementation decreases the fat content of cuts of pig meat, such as bacon and roasts, which contain subcutaneous fat. The effect of dietary RAC on intramuscular fat are equivocal with increased levels (Aahlus et al. 1990), no change (Perkins et al. 1992) and decreased levels (Engeseth et al. 1992) being reported. The fatty acid composition of both subcutaneous and intramuscular fat is essentially unchanged by dietary RAC (Engeseth et al. 1992; Perkins et al. 1992). Likewise, objective and subjective measures of meat tenderness or appearance are not discernibly affected by dietary ß-agonists (McKeith, 1993).

(3) Responses to dietary protein As for pST-treated pigs, the ß-agonist RAC has no effect on nutrient digestibility (F.R. Dunshea, unpublished) and so effects must be predominantly post-absorptive. The limited information, obtained only in finisher pigs, suggests that the increased protein deposition rates observed in response to ß-agonists increase the dietary requirement for protein since RAC and other ß-agonists are not effective in pigs fed low levels of dietary protein. Thus, Anderson et al. (1987) found that RAC increased nitrogen retention in pigs fed a 16% protein diet, whereas nitrogen retention was decreased in pigs fed a 12% protein diet. Likewise, Bracher-Jacob and Blum (1990) found that the ß-agonist Ro 16-8714 increased protein deposition in pigs receiving diets containing 14% but not 11% dietary protein.

Recently, we investigated the interactions between dietary protein and RAC in female pigs restricted to 30 MJ DE/d (Dunshea et al. 1993b). The relationship between protein deposition and dietary "ideal" protein content (Fig. 8) clearly demonstrates that the rate of protein deposition was influenced by both dietary protein and RAC. The relationship between protein deposition and dietary protein content for both the control and RAC-treated pigs could be described equally well by either a linear-plateau or a curvilinear model (Fig. 8). Regardless of which model was used the interpretation is the same. The efficiency of use of dietary protein was not altered by RAC since protein deposition increased with protein content at a similar rate for both the control and RAC-treated pigs over at least the two lowest levels of dietary protein (< 11% protein). However, at higher dietary protein contents, the plateau or maximal protein deposition rate was 23% higher in the pigs receiving RAC (96 vs 118 g/d for control and RAC-treated pigs, respectively). When the linear/plateau model was fitted to the data, the dietary protein content required to support maximum protein deposition were 12.7 and 15.8% for the control and RAC-treated pigs, respectively. When a quadratic model was fitted to the data the respective estimates of dietary protein required to maximise protein deposition were 17.6 and 20.8%. Regardless of which model is used, it is apparent that RAC-treated female pigs require a diet containing approximately 3% more protein to maximise protein deposition. Also, although the two models gave quite disparate (approximately 5% protein content) estimates of the dietary protein level to maximise protein deposition, it should be noted that estimates of protein deposition predicted by the curvilinear model were within the SE of maximum protein deposition predicted by the linear/plateau model over a range of 8% of dietary protein for both control and RAC-treated pigs.

(4) Responses to dietary energy As noted earlier, it is generally accepted that an increased MER is an inevitable consequence of increased protein deposition and this is certainly the

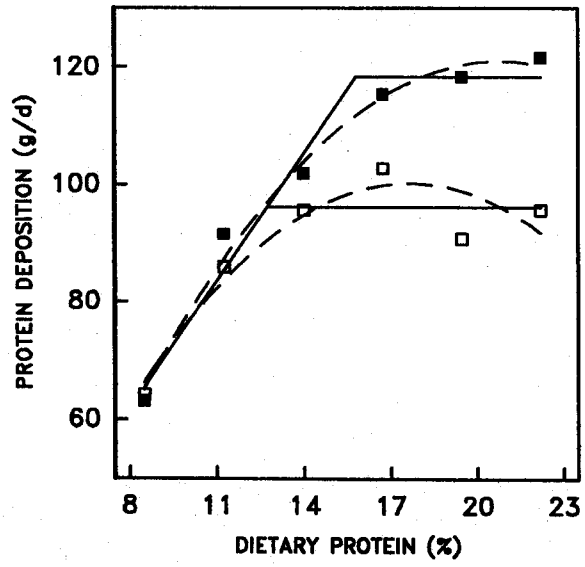

Fig. 8. Relationship between empty body protein deposition and dietary protein content in female pigs restrictively-fed (30 MJ DE/d) receiving 0 (□) and 20 (■) ppm dietary RAC between 60 and 90 kg (Dunshea et al. 1993b).

case for pigs treated with pST. However, the ratio of energy deposited to energy intake shows no change in the efficiency of energy deposition during RAC treatment under both ad libitum and restricted feeding conditions (Dunshea et al. 1993a,b). Conversely, Mitchell et al. (1990) found that the efficiency of utilisation of DE was 19% less in RAC-treated pigs. Subsequent work in the same laboratory demonstrated no difference in the efficiency of use of DE, although regression analysis suggested a small (8%) increase in MER of RAC-treated pigs (Mitchell et al. 1991). Also, fasting heat production was not altered in RAC-treated pigs (Yen et al. 1991).

Our previous research demonstrated that dietary RAC did not increase protein deposition in female pigs restricted to approximately 70% of ad libitum intake (21 g/d; Dunshea et al. 1993b) to as great an extent as it did in ad libitum fed female pigs (51 g/d; Dunshea et al. 1993a) suggesting that energy intake may limit response to RAC. Therefore, we decided to investigate the interactions between dietary energy intake and protein and fat deposition in RAC-treated finisher female and male pigs (Dunshea et al. 1993c). The relationship between protein deposition and DE intake for the control females was of the linear/plateau form with carcass protein deposition reaching a plateau at 140 g/d (3.3 MJ/d) at an energy intake of 36 MJ DE/d (Fig. 9a). However, in RAC-treated females protein deposition increased linearly with increasing energy intake up to a maximum of 191 g/d (4.6 MJ/d) at an ad libitum DE intake of 47.2 MJ DE/d. Supplementation of the diet with RAC increased protein deposition at every level of energy intake. The slope of the linear ascending portions of the curve were not different and the improvement in protein deposition due to dietary RAC was 21 g/d (0.5 MJ/d) up until a DE intake of 36 MJ/d. For males receiving either 0 or 20 ppm of RAC the relationship between protein deposition and energy intake was linear up until ad libitum DE intakes of approximately 45 MJ/d (Fig. 9b). While the slopes of these lines were the same, the benefit to protein deposition in male pigs (19 g/d) was similar to that observed in female pigs. Therefore, dietary RAC increases protein deposition in both sexes at every level of energy intake but ad libitum intakes are necessary to maximise protein deposition in improved genotypes treated with RAC. Also,

the differences in protein deposition between the sexes are still evident during RAC treatment.

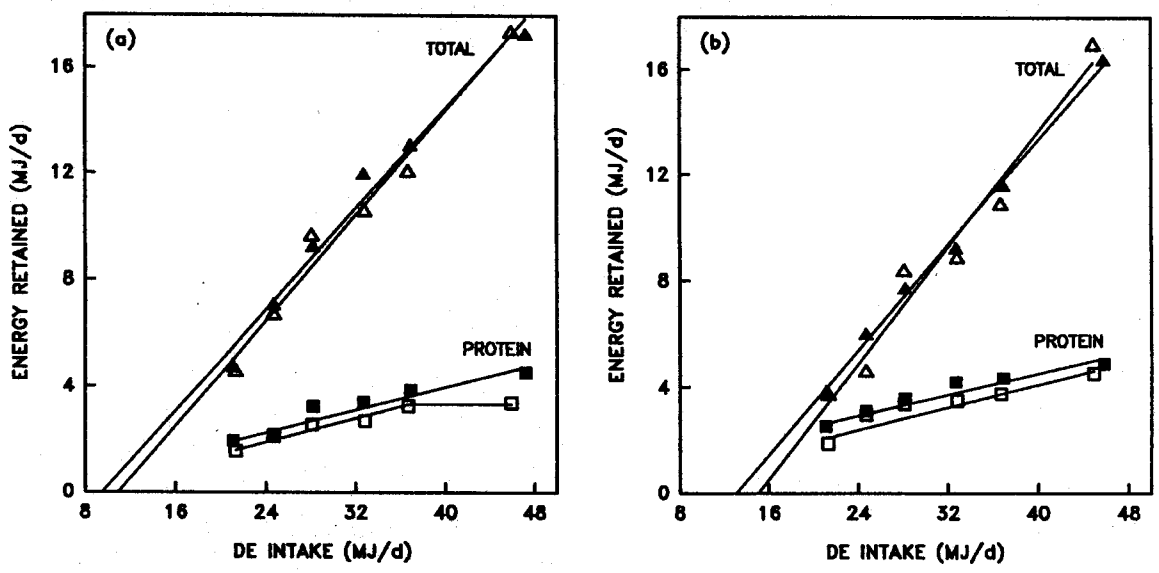

Fig. 9. Relationship between protein energy (□,■) and total energy (▽,▼) retained and DE intake in (a) female or (b) male pigs receiving 0 (open symbols) or 20 (closed symbols) ppm dietary RAC between 60 and 90 kg (Dunshea et al. 1993c).

While RAC increased the amount of energy stored as protein it had no effect on total energy storage or the amount of energy stored as fat (difference between total and protein energy). Unlike with exogenous pST, MER was decreased by RAC treatment in both female (10.9 vs 9.5 MJ DE/d for control and RAC-treated, respectively) and male (15.0 vs 13.1 MJ DE/d for control and RAC-treated, respectively) pigs. These data suggest that the MER of RAC-treated pigs are decreased despite greater rates of protein deposition and increased protein mass.

IV. CONCLUSION

Treatment of pigs with pST and dietary ß-agonists such as RAC increases protein deposition and improves productive performance. These metabolic modifiers are most likely to be used in the finisher pig (>60 kg) where increases in performance are greatest. In this class of pigs, increases in protein deposition will most likely require a commensurate increase in dietary protein although there is some evidence that pST may improve efficiency of use of dietary protein when it is low. Conversely, when dietary protein is adequate, pST and RAC increase protein deposition across a wide range of energy intakes and may raise or eliminate the plateau of protein deposition. In improved genotypes it may be necessary to feed ad libitum to maximise protein deposition in pigs treated with metabolic modifiers.

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