

Somatotropin regulation of protein metabolism in pigs^{1,2}

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ABSTRACT: A primary goal of exogenous somatotropin treatment is to increase lean body mass. This is accomplished, in part, by increasing the efficiency with which dietary amino acids are used for protein deposition. Somatotropin administration also improves protein balance by minimizing the loss of protein during fasting and maximizing the protein gained during meal absorption. Amino acid catabolism is decreased by somatotropin treatment, as indicated by decreases in blood urea nitrogen, urea synthesis, hepatic urea cycle enzyme activity, and amino acid oxidation. Stable isotope tracer/mass transorgan balance studies have recently demonstrated that somatotropin treatment increases protein anabolism in young, growing swine by increasing protein synthesis in the hind limb and por-

tal-drained viscera in the fed state, with little effect on protein degradation. Detailed study of the tissue-specific responses indicates that somatotropin treatment increases protein synthesis in skeletal muscle by increasing the efficiency of the translational process, but only in the fed state. The somatotropin-induced stimulation of skeletal muscle protein synthesis involves mechanisms that enhance the binding of both mRNA and initiator methionyl-tRNA to the 40S ribosomal subunit. Somatotropin increases protein synthesis in the liver in both the fasted and fed states by increasing ribosome number, with no change in translation initiation. Thus, the protein synthetic response to somatotropin treatment is tissue-specific and dependent on nutritional state.

Key Words: Muscle, Pigs, Protein Synthesis, Proteolysis, Somatotropin

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Introduction

Somatotropin is an important regulator of growth. Administration of somatotropin to domestic animals increases protein deposition, decreases fat deposition, and improves the efficiency with which dietary amino

acids are used for growth (Campbell et al., 1990; Caperna et al., 1991; Beermann and Boyd, 1992). Administration of somatotropin also improves nitrogen retention and reduces amino acid catabolism, as indicated by a reduction in blood urea nitrogen, amino acid oxidation, urea synthesis, and hepatic urea cycle enzyme activity (Dunshea et al., 1992; Vann et al., 2000a; Bush et al., 2002). A primary goal of using anabolic agents to stimulate the growth of domestic animals is to increase muscle mass, and a number of studies have demonstrated that this goal is achieved with somatotropin treatment (Smith and Kasson, 1990; Beermann and Boyd, 1992; Seve et al., 1993). However, the increased growth with somatotropin treatment also occurs in tissues other than muscle, including liver, intestine, kidney, skin, and bone (Evock-Clover et al., 1992; Boisclair et al., 1994; Caperna et al., 1995). The purpose of this review is to examine the mechanism by which somatotropin enhances protein deposition in domestic animals.

Effect of Somatotropin on Protein Synthesis

Although it is commonly held that exogenous somatotropin administration increases protein deposition by

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increasing muscle protein synthesis, the evidence for this assumption must be viewed with caution. For example, infusion of somatotropin in adult humans increased protein synthesis in the whole body and reduced protein synthesis in the leg in one study (Cope land and Nair, 1994), but, in another study, the opposite results were obtained (Fryburg and Barrett, 1993). In steers, chronic somatotropin treatment tended to increase hind limb protein synthesis (Boisclair et al., 1994) and significantly increased protein synthesis in some muscles, although not in others (Eisemann et al., 1989). Differences in the findings among the studies may be due to length and/or mode of somatotropin treatment, species differences, tissues analyzed, developmental stages of the subject population, or nutrient status of the subject. Indeed, feeding profoundly stimulates protein synthesis and this response changes with development (Davis et al., 1996). Whether the animals were fasted or fed at the time of study could potentially affect the measured response to somatotropin treatment.

Studies from our laboratory have suggested that 7 d of somatotropin treatment in young, growing swine (approximately 20 kg) enhances protein balance and metabolic efficiency by minimizing protein loss during fasting and maximizing protein gain during meal absorption (Vann et al., 2000a,b). Using leucine as the stable isotopic tracer to measure rates of whole-body protein turnover and amino acid oxidation, we found that somatotropin treatment increased whole-body protein synthesis in the fasting state (Figure 1). Feeding increased protein synthesis in both control and somatotropin-treated pigs, but somatotropin did not further increase whole-body protein synthesis beyond the effect of feeding. Somatotropin treatment did not alter whole-body protein degradation in the fasted state but reduced protein degradation in the fed state. Thus, somatotropin treatment improved protein balance in both the fasted and fed states, but by different mechanisms; that is, the increase in protein balance in the fasting state involved an increase in whole-body protein synthesis and that in the fed state involved a reduction in whole-body protein degradation. However, the reduction in whole-body leucine oxidation with somatotropin treatment was present in both the fasted and fed state.

To identify the tissue-specific responses of protein synthesis and protein degradation to exogenous somatotropin administration, we used a dual stable isotope tracer/mass transorgan balance technique to measure phenylalanine kinetics in vivo in the hindquarters and, for comparison, the portal-drained viscera (Bush et al., 2003a). Studies were performed in young, rapidly growing pigs in which protein intake and somatotropin treatment were rigorously controlled over a 7-d treatment period. An intraduodenal infusion of diet ensured a fed state during the 6-h isotope infusion study. The results showed that 7 d of somatotropin treatment increased protein synthesis in the hindquarters of growing pigs in the postprandial state. The increase in amino acid

extraction and utilization for protein synthesis in the hindquarters was associated with an increase in blood flow to the hindquarters. Protein degradation tended to be reduced in the hindquarters in somatotropin-treated pigs, although this response was not statistically significant. Protein deposition in the hindquarters was enhanced by somatotropin treatment, likely contributing to the increased body mass observed with somatotropin over the 7-d treatment period. In the portal-drained viscera of growing pigs in the fed state, 7 d of somatotropin treatment increased protein synthesis (Bush et al., 2003a). However, in contrast to that in the hindquarters, this increase in protein synthesis was not associated with a change in blood flow. Protein degradation in the portal-drained viscera was unaffected by somatotropin treatment.

Thus, somatotropin treatment increases protein anabolism in young, growing swine by increasing protein synthesis in the hindquarters and portal-drained viscera in the fed state (Bush et al., 2003a). This contrasts with the lack of effect of somatotropin treatment on whole-body protein synthesis in the fed state that we have observed when using either leucine (Vann et al., 2000a,b) or phenylalanine (Bush et al., 2003a) as isotopic tracers. Differences in the effect of somatotropin on protein synthesis in the hindquarters and portal-drained viscera vs. the whole body are likely due to differences in the responses in other tissues, potentially in adipose tissue. In this regard, considerable data suggest that somatotropin induces a repartitioning of nutrients toward lean tissue and away from adipose tissue deposition (Etherton and Bauman, 1998). However, other mechanisms may also be involved. We also found that somatotropin treatment had no effect on protein degradation in the fed state in either the hindquarters or the portal-drained viscera (Bush et al., 2003a). Whether the decrease in whole-body protein degradation with somatotropin treatment in the fed state (Vann et al., 2000a; Bush et al., 2003b) can be accounted for by a reduction in proteolysis in liver and other visceral tissues remains to be determined. Because somatotropin does not alter urea cycle enzyme activity in the intestine but reduces urea cycle activity in the liver (Bush et al., 2002), we postulate that the reduction in whole-body amino acid oxidation that we have observed using both leucine (Vann et al., 2000a,b) and phenylalanine (Bush et al., 2003a) as isotopic tracers is due to a reduction in amino acid oxidation in the liver.

Thus, the effects of somatotropin on protein synthesis, protein degradation, and amino acid oxidation appear to be tissue-specific. Because protein metabolism by the hindquarters is a compilation of that in individual skeletal muscles, skin, bone, and adipose tissue and that by the portal-drained viscera includes the small intestine, large intestine, stomach, spleen, and pancreas, the role of individual tissues in the response to somatotropin required further study. Furthermore, the underlying mechanism by which somatotropin treat-

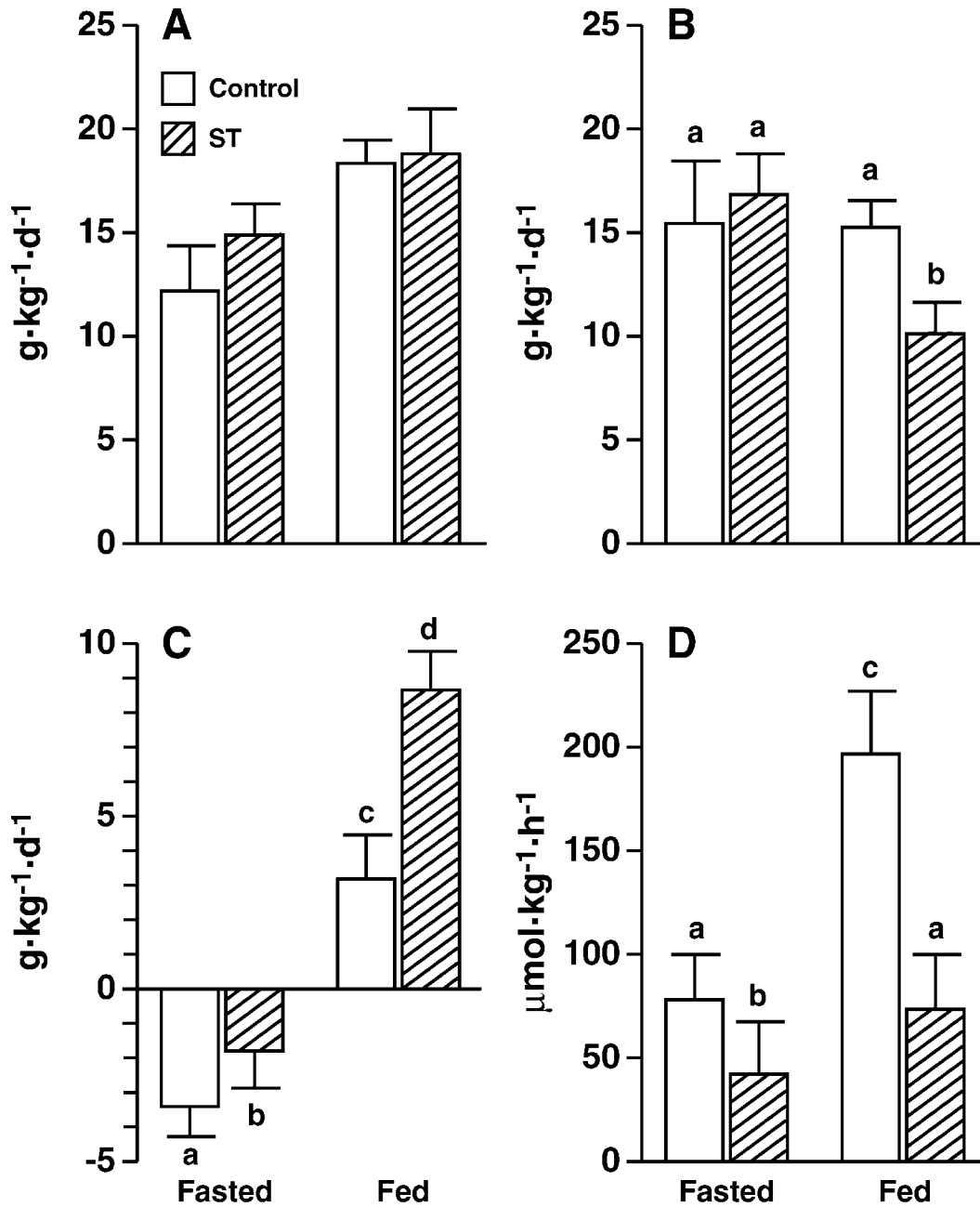


Figure 1. Whole-body protein synthesis (A), protein degradation (B), protein balance (C), and leucine oxidation (D) in control and somatotropin-treated pigs in the fasted and fed states. Results are presented as means \pm SD. Protein synthesis (Panel A) was increased by feeding state and ST treatment (main effect means differ at $P < 0.05$, and no interaction was detected, $P > 0.05$). Because interactions were detected ($P < 0.05$) among treatments for data in Panels B, C, and D, paired t -tests were performed. Different letters above the bars within panels indicate that the simple treatment means differ at $P < 0.05$. Data are from Vann et al. (2000b).

ment enhances protein synthesis and thus protein deposition remained to be defined.

Regulation of Translation Initiation by Somatotropin

The rate of protein synthesis in a tissue is dependent on both the number of ribosomes and the efficiency with which the ribosome translates messenger RNA (mRNA)

(Kimball and Jefferson, 1988). Translational efficiency reflects how well the protein synthetic machinery functions and is dependent on the abundance and activity of the components involved in the translation of mRNA. Translation of mRNA into protein involves the processes of initiation, elongation, and termination, with the primary regulatory site being the initiation process. Studies performed largely in cell culture have shown that translation initiation is regulated at two particu-

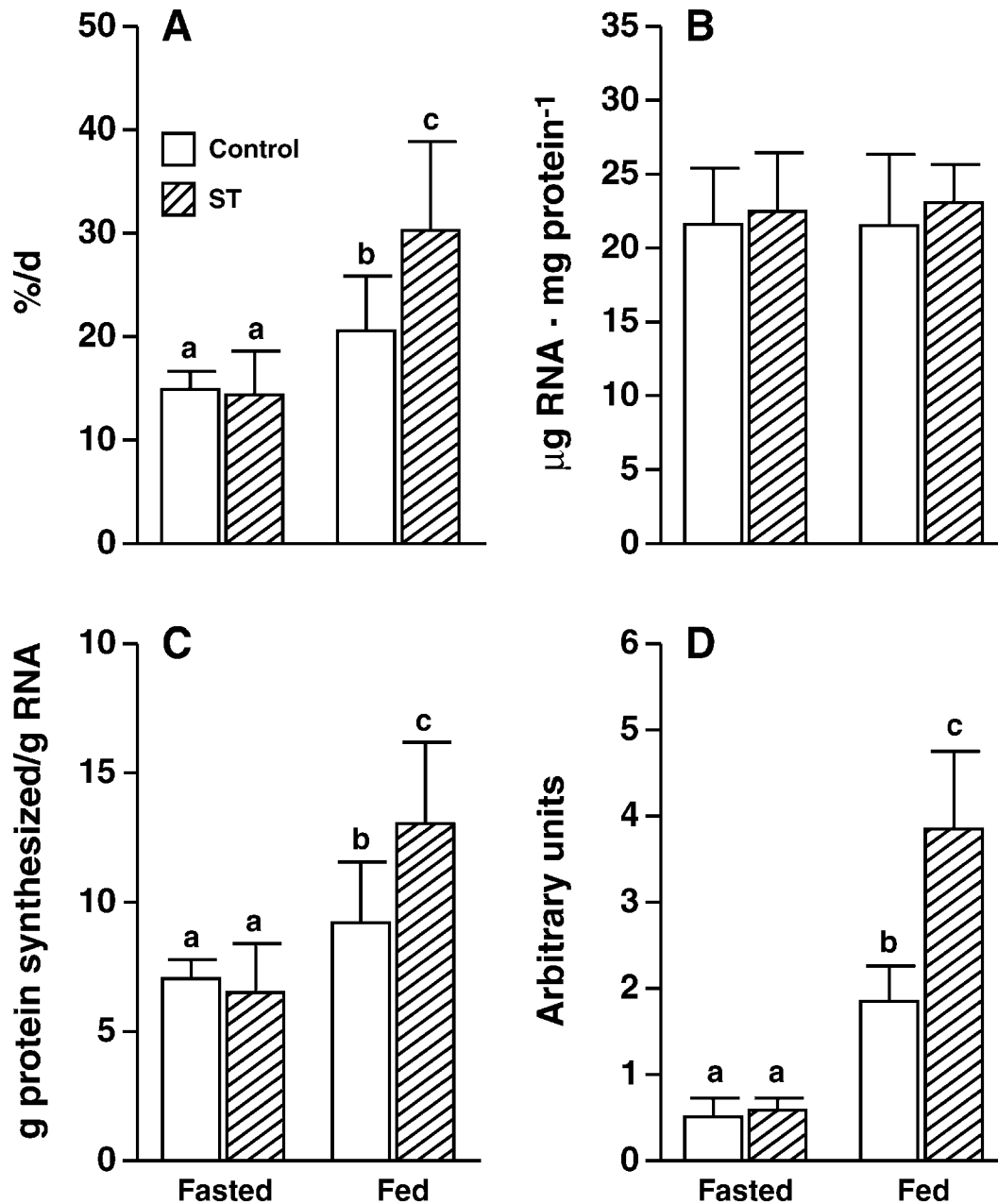


Figure 2. Fractional rates of protein synthesis (A), ribosome number (B), translational efficiency (C), and the association of eukaryotic initiation factor (eIF) 4E with eIF4G (D) in the longissimus dorsi muscle in control and somatotropin-treated pigs in the fasted and fed states. Results are presented as means \pm SD. Because interactions among treatments were detected ($P < 0.05$) for data in Panels A, C, and D, paired t -tests were performed. Different letters above the bars within panels indicate that the simple treatment means differ at $P < 0.05$. Ribosome number (Panel B) was not affected by treatment. Data are from Bush et al. (2003b).

larly important steps. The first is the binding of initiator methionyl-tRNA (**met-tRNA_i**) to the 40S ribosomal subunit, a reaction that is mediated by eukaryotic initiation factor (eIF)2 and results in the formation of the 43S preinitiation complex (Kimball et al., 1996). The eIF2-mediated binding of met-tRNA_i to the 40S subunit is regulated by modulation of the activity of eIF2B, a factor required for the exchange of GDP for GTP on eIF2. The second step critical to the regulation of translation initiation is the binding of mRNA to the 43S

preinitiation complex, which is mediated by the assembly of the eIF4F complex of proteins (Hershey and Merrick, 2000). Phosphorylation and availability of eIF4E regulate the formation of the eIF4F complex. Availability of eIF4E is regulated by its association with the eIF4E binding protein, 4E-BP1, a repressor protein that competes with eIF4G for binding to eIF4E. The phosphorylation of 4E-BP1 in the inactive eIF4E·4E-BP1 complex results in a decreased affinity of eIF4E for 4E-BP1, and the release of eIF4E enhances the binding of

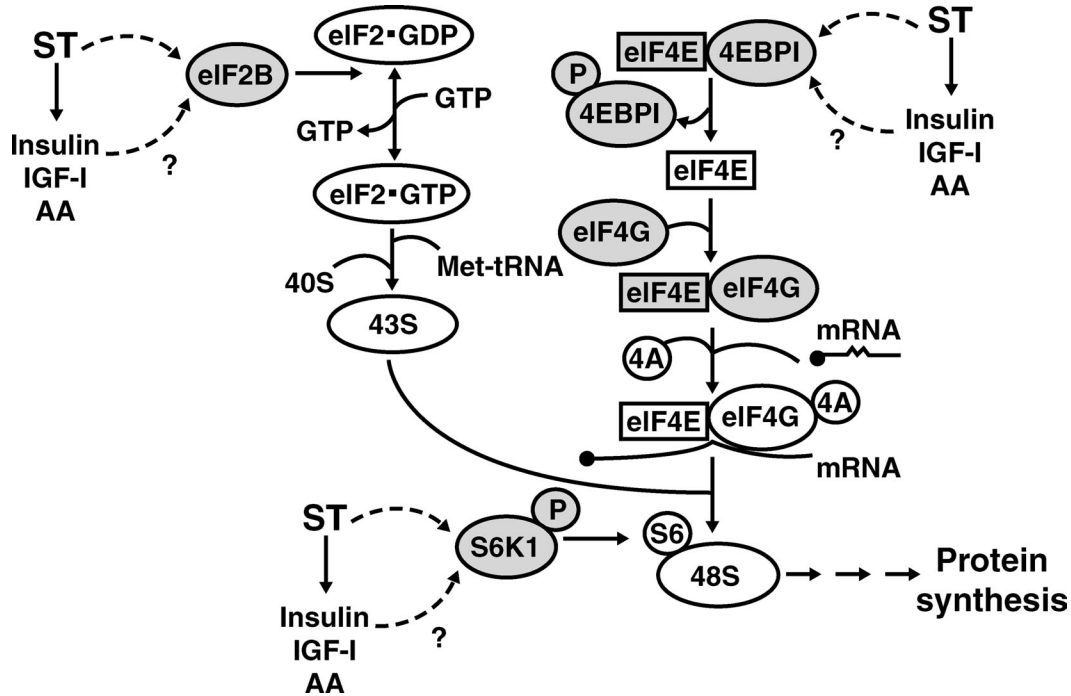


Figure 3. Regulation of translation initiation factors by somatotropin in growing pigs. Key regulatory factors are shown by shadowed symbols. Abbreviations: AA = amino acids; eIF = eukaryotic initiation factor; 4E-BP1 = eIF4E binding protein; met-tRNA_i = initiator methionyl tRNA; IGF-I = insulin-like growth factor-I; S6K1 = S6 kinase 1; ST = somatotropin; 43S = 43S ribosomal subunit; 48S = 48S ribosomal subunit.

eIF4E to eIF4G to form the active eIF4E-eIF4G complex. These events may be regulated by changes in the phosphatidylinositol 3-kinase (**PI3-kinase**)/ribosomal protein S6 kinase 1 (**S6K1**) signaling pathway (Gingras et al., 2001). In vivo studies have demonstrated that feeding (Davis et al., 2000; Kimball et al., 2000, 2002), insulin (O'Connor et al., 2003a,b), IGF-I (Davis et al., 2002b; Shen et al., 2002), and amino acids (Anthony et al., 2000; O'Connor et al., 2003a,b) induce changes in the activation of translation initiation components as well as changes in the overall rates of muscle protein synthesis. However, the effect of somatotropin on the regulation of translation initiation in vivo had not previously been studied.

Therefore, we examined the effect of 7 d of somatotropin treatment on the mechanisms that regulate protein synthesis in skeletal muscle and, for comparison, liver of fasted and fed, young, growing pigs (Bush et al., 2003b). Using leucine as the stable isotopic tracer to measure the fractional rate of incorporation of the labeled amino acid into tissue protein, we found that somatotropin treatment in growing pigs increased protein synthesis in longissimus dorsi muscle, a muscle composed of primarily fast-twitch muscle fibers. However, this increase was only apparent in the fed condition and was due to an increase in the efficiency of translation (Figure 2). The changes in muscle protein synthesis in the fed state was dependent on those translation initiation factors that regulate the binding of both mRNA and initiator met-tRNA_i to the ribosomal

complex such that the phosphorylation of 4E-BP1, association of eIF4E with eIF4G, and eIF2B activity were enhanced. We further found a tissue-specific response to 7 d of somatotropin treatment as somatotropin significantly elevated liver protein synthesis in both the fasted and fed conditions, unlike that of skeletal muscle. This increase in liver protein synthesis was due to an increase in ribosome number and not to a change in the processes that regulate translation initiation.

Whether somatotropin acts directly to increase the activation of specific translation initiation factors in skeletal muscle or indirectly by altering the concentration and/or sensitivity to other anabolic agents has not been determined (Figure 3). In this regard, somatotropin treatment increased circulating insulin levels in the postprandial state (Bush et al., 2003b). Because insulin mediates the feeding-induced increase in skeletal muscle protein synthesis and translation initiation in young, growing swine (Davis et al., 2002a; O'Connor et al., 2003b), it is plausible that the somatotropin-induced stimulation of translation initiation in skeletal muscle of fed pigs (Bush et al., 2003b) may be due to the somatotropin-induced increase in insulin concentrations. Although somatotropin treatment reduces the responsiveness of whole body glucose metabolism to insulin, somatotropin does not alter the insulin responsiveness of whole body protein metabolism (Vann et al., 2000b). However, whole body protein synthesis is a compilation of protein synthesis rates in numerous tissues, most of which are not as responsive as skeletal

muscle to insulin. Somatotropin treatment also increased circulating IGF-I levels, and this was enhanced in the fed condition (Bush et al., 2003b). Because IGF-I increases protein synthesis in muscle, but not liver, of young, growing pigs (Davis et al., 2002b), it is also plausible that the somatotropin-induced increase in muscle protein synthesis is due to the somatotropin-induced increase in IGF-I concentrations. The increase in skeletal muscle protein synthesis by somatotropin treatment occurred only in the fed condition (Bush et al., 2003b). Because muscle protein synthesis is sensitive to amino acid stimulation in young, growing swine (O'Connor et al., 2003a) and amino acids and insulin interact to activate specific translation initiation factors (O'Connor et al., 2003b), it is also plausible that fed levels of amino acids are permissive to the direct (by somatotropin) or indirect (via insulin or IGF-I) stimulation of translation initiation by somatotropin.

Implications

Exogenous administration of somatotropin to domestic animals increases the efficiency with which dietary amino acids are used for protein deposition. From a whole body perspective, this is accomplished by minimizing the loss of protein during fasting and maximizing the protein gained during meal absorption. Although somatotropin treatment increases protein deposition in a number of tissues, including skeletal muscle, the mechanism by which this increase occurs is tissue-specific and dependent on nutritional state. Somatotropin increases protein synthesis in skeletal muscle in the fed state by increasing the efficiency of the translational process and increases protein synthesis in the liver in the fasted and fed states by increasing ribosome number. Further study is needed to determine whether somatotropin acts directly to stimulate protein synthesis in individual tissues or indirectly via changes in the concentration and/or sensitivity to other anabolic agents (i.e., insulin, IGF-I, and amino acids).

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