A REVIEW OF HORMONAL INFLUENCE ON GROWTH AND FATTENING OF LIVESTOCK*

T. G. ALTHEN
Meat Science Research Laboratory, AMRI, ARS, USDA
Beltsville, Maryland

With the trend toward increased emphasis on energy conservation, feeding animals to a high degree of finish has become wasteful. In addition, health reports have suggested that human diets high in animal fat can lead to cardiovascular diseases. Therefore, consumers have become more conscious of lean tissue in their selection of meats. The livestock producer must try to produce an animal that will have a high yield of lean cuts, yet will have acceptable eating qualities. For example, with increased numbers of grass fed cattle, new methods to maintain a high standard of quality must be developed. Therefore, the processes regulating growth and fattening of livestock are of fundamental importance to the producer as well as the consumer.

A number of hormones affect growth generally, and more specifically, gluconeogenesis, lipogenesis, lipolysis and protein synthesis (figure 1). For example, epinephrine and glucagon produce rapid hyperglycemic effects on blood sugar, which are balanced by the increased secretion of a hypoglycemic hormone such as insulin.

In the regulation of fat metabolism, several hormones such as epinephrine, growth hormone, adrenocorticotropic hormone and lipotropins stimulate lipid breakdown and utilization by activating enzymes called "lipases," which catalyze the hydrolysis of triglycerides to release fatty acids. The primary balance in conserving triglycerides is provided by insulin. In promoting glucose uptake by adipose tissue, insulin increases the supply and utilization of carbohydrate, sparing C_2 units, used in the synthesis of triglycerides.

In the regulation of protein synthesis, growth hormone and insulin are the most important; and both lead to a positive nitrogen balance and increased rate of protein synthesis. The protein anabolic effects of androgens are more specific; but muscle, as well as those tissues involved in reproduction, are affected. Also, normal levels of thyroxine are necessary for adequate protein synthesis. In thyroid deficiency, all metabolic processes proceed sluggishly, however, an excess of thyroxine leads to a general catabolic effect and a negative nitrogen balance. The hormones that are most involved in protein degradation are the glucocorticoids, which can cause protein wasting and negative nitrogen balance.

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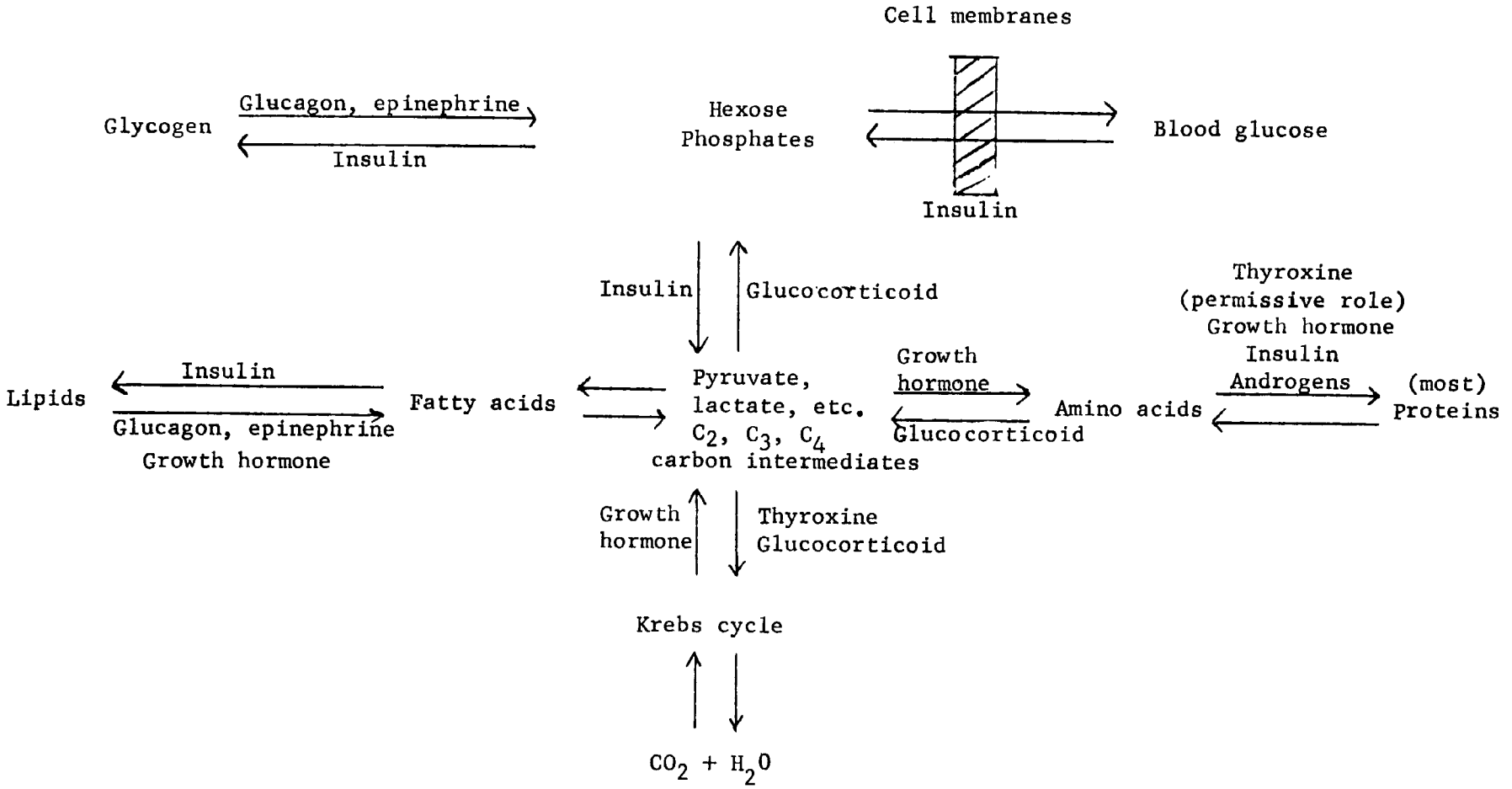


Figure 1. EFFECT OF HORMONES ON METABOLISM.
(Diagram modified from Friedman and Lipner, 1971.

Hormones may be related to each other synergistically or antagonistically in the production or degradation of some tissues. For example, growth hormone and insulin are both protein anabolic and promote positive nitrogen balance; but in adipose tissue, insulin is lipogenic and growth hormone is lipolytic.

The complex interrelationships of the hormones complicate the study of the independent influence of each hormone on the growth and fattening process in farm animals.

Several methods have been used to study the effect and/or influence of specific hormones on growth and development. The methods most commonly used have been:

(1) Administration of exogenous doses of a particular hormone (exogenous treatment with a hormone).

(2) Measurement of endogenous hormone levels and correlation of hormone level with a particular function.

(3) Measurement of the secretion, metabolic clearance and endogenous level of a hormone and correlation of these parameters with a particular function.

I would like to discuss each method and, in particular, some hormone-function relationships that have been identified by use of each procedure.

Through studies utilizing administration of exogenous hormones, various factors which can affect the influence of a particular hormone on growth or fattening have been identified. Using several studies from the literature, I would like to point out some of these factors for several hormones.

Growth hormone causes a general increase in body mass and affects the body composition of treated animals. Growth hormone data illustrate that response may differ with sex (table 1). Treatment with growth hormone enhanced growth of females but not of males (Henricson and Ullberg, 1960). Treatment with growth hormone also enhanced nitrogen retention and reduced fat deposition (Turman and Andrews, 1955, Davis et al., 1969, and Machlin, 1972a).

Even though growth hormone treatment improves growth, increases carcass protein and reduces carcass fat, it has not been used to a significant extent in animal production because the isolation of the natural hormone in sufficient quantities to treat farm animals routinely has not been practical. Also, as pointed out by Machlin at the RMC (1972b), in the case of polypeptide hormones, the species of origin and the purity of the hormone must be questioned. In reference to species of origin, this was originally questioned on the basis of immunological activity. Polypeptide hormones, such as growth hormone, are composed of many amino acids. However, the portion of the molecule

Table 1. Influence of various injected hormones on growth and carcass traits of farm animals

Hormone	Species (age or body weight)	Dose	Effect[a]	Reference
Growth hormone	Swine (24 days)	.05-.2 mg/kg BW daily	Growth females↑ males -	Henricson and Ullberg, 1960
Growth hormone	Swine (50 kg, BW)	.15-.67 mg/kg BW daily	Growth - Feed/gain ↓ Protein ↑ Fat ↓	Turman and Andrews, 1955
Growth hormone	Sheep (35 kg, BW)	10 mg, daily	Nitrogen retention ↑	Davis, et al., 1969
Glucocorticoids	Sheep (50 kg, BW)	50 mg, each 2d day	Growth -	Purchas, 1973
Epinephrine	Swine (65 kg, BW)	.15 mg/kg, BW daily	Nitrogen retention ↑ Fat - Growth ↑	Cunningham, et al., 1963
Epinephrine	Swine (2-8 weeks)	.0625-.2 mg/kg BW, daily	Nitrogen retention ↑ Fat ↓ Growth ↓	Cunningham et al., 1963
Insulin (alloxan-diebetic)	Swine (15 kg)	200 mg Alloxan/ kg, BW	Growth ↓	Romsos et al. (1971)

[a] Increase (↑), Decrease (↓), no effect (-).

(sequence of amino acids) that imparts immunological species specificity may be different from that which imparts biological activity. Thus, even though hormones isolated from two different species may be immunologically dissimilar, their biologically active cores could be similar and, thus, biologically active across species (Evans et al., 1966). Some radio-receptor studies indicate this to be the case (Freychet et al., 1971). Therefore, the next step is to identify the biologically active core of the naturally occurring hormone. After identification of the amino acid sequence of the active core, through synthetic processes, it may be possible to prepare the compound commercially so that it can be used in place of the naturally occurring compound (subject to FDA approval).

Thyroidal hormones (thyroxine and triiodothyronine) are required for normal growth and maturation. Generally, they do not stimulate growth beyond the normal level.

Feeding of thyroprotein (iodinated casin) to swine sometimes increases body weight gain, but the dose is important. Growth was promoted in young swine fed up to 44 mg thyroprotein per kg of diet (Peo and Hudman, 1960) (table 2). However, the growth rate was depressed and the feed-gain ratio was increased in young swine fed 88 mg per kg of diet. In addition, beneficial effects are limited to young growing pigs; after fattening begins and they reach approximately 50 kg, thyroprotein retards the rate of gain and decreases feed efficiency (Carlson, 1969).

The adrenals secrete several hormones which have varying effects on general metabolism and growth. Epinephrine, secreted from the adrenal medulla, has well known glycogenolytic and lipolytic properties. Glucocorticoids secreted from the adrenal cortex increase urinary nitrogen excretion and stimulate gluconeogenesis from amino acids.

As with thyroprotein, age was a factor in the effect of treatment with epinephrine (table 1). Epinephrine treatment retarded growth of young swine but enhanced growth of the heavier swine. Epinephrine increased nitrogen retention in swine of both ages and retarded fat deposition in the younger swine (Cunningham et al., 1963). Glucocorticoid treatment had no effect on 50 kg sheep (Purchas, 1973) (table 1).

The effects of gonadal steroids are variable, depending upon the dose, age, sex and species of the animal. For example, implantation of the synthetic estrogen-like compound, diethylstilbestrol (DES), increases the rate of gain by about 0.1 to 0.25 kg/day in beef cattle (Carlson, 1969). However, DES had no effect when administered to 20 kg swine. (Thrasher et al., 1959) (table 2).

Treatment with exogenous hormones (method 1) to determine hormone-function relationships, has given some useful information. However, usefulness of the method as a research tool is limited by the number of animals that can be treated at a time, the lack of enough hormone in the case of polypeptides, to treat large numbers of animals and, finally, the effect of variables such as species, sex, age and dosage.

Table 2. Influence of feeding various hormones or synthetic analogs on growth and carcass traits of farm animals

Hormone	Species (age or body weight)	Dose	Effect[a]	Reference
Thyroxine	Swine (8 days)	0-44 mg thyroprotein/ kg, diet	Growth ↑ Feed/gain ↓	Peo and Hudman, 1960
Thyroxine	Swine (8 days)	88 mg thyroprotein/ kg, diet	Growth ↓ Feed/gain ↑	Peo and Hudman, 1960
Thyroxine	Swine (20 kg, BW)	.0088-.0176% thyro-protein, diet	Growth ↑ Feed/gain ↑	Perry et al., 1950
Androgenic compounds	Swine (20 kg, BW)	.66-11 mg various analogs/kg diet	Growth - Leanness ↑	Thrasher et al., 1959
Estrogenic compound (DES)	Swine (20 kg, BW)	2.2 mg DES/kg diet	Growth - Carcass characteristics -	Thrasher et al., 1959

[a] Increase (↑), decrease (↓), no effect (-).

The second method used to examine hormone-function relationships has been to correlate the endogenous hormone supply with growth and carcass traits.

In earlier studies, pituitary hormone concentrations were determined by bioassay. For example, Baird et al. (1952) reported that anterior pituitaries from pigs selected for rapid gain contained significantly greater amounts of growth hormone per unit tissue than those from pigs selected for slow rate of gain. Baker et al. (1956) concluded that reduction in growth rate of swine after 227 days of age was due to the dilution of available growth hormone to a level that no longer stimulated rapid growth.

In these earlier studies, only pituitary hormone levels could be measured. The relationship between pituitary hormone and its circulating level was unknown. In other words, did high pituitary levels mean high or low circulating levels? With the development of radioimmuno-assay procedures during the early 1960's quantitation of the low levels of circulating hormones has been possible.

The hormone that has been examined most often in relation to growth and carcass traits is growth hormone. Its various parameters, such as total pituitary contents and pituitary or serum concentrations have been quantitated and then statistically analyzed to determine their correlations with growth rate and with carcass traits. Few significant correlations between growth hormone and growth or carcass traits have been reported. One study utilizing cattle (Hafs et al., 1971) and another utilizing swine (Siers and Hazel, 1970) suggested that high serum growth hormone was associated with a slow growth rate and low ratio of lean to fat. These studies suggested that animals that are growing more rapidly or depositing more lean tissue use serum growth hormone at a more rapid rate and hence have low levels of serum growth hormone. On the other hand, several other studies of both cattle (Trenkle and Irvin, 1970) and swine (Chappel and Dunkin, 1975) reported that a high level of serum growth hormone was associated with a fast growth rate and a high ratio of lean to fat. In Beltsville, studies of swine selected for either high or low backfat thickness also showed that growth hormone tended to be positively related to amount of lean tissue and negatively related to the amount of fat tissue (Althen and Gerrits, 1972). These latter studies agree with the known biological function of growth hormone in that high levels of growth hormone are associated with lean animals. The discrepancies in reported relationships between growth hormone and its functions may have been due to failure of the measurement of serum hormone levels, particularly of a single sample, to reflect accurately the influence of that hormone on the animal under investigation (Siers and Trenkle, 1973). Also, in the measurement of endogenous hormones and their relationships to growth, breed differences must be taken into account. If, within a species, two breeds with different carcass traits differed in their serum hormone levels, an apparent significant correlation could exist between carcass trait and hormone. For example, simple correlation coefficients were calculated for pituitary growth

hormone concentration versus some carcass traits from swine selected for high or low backfat thickness. All the coefficients (r) were highly significant ($p < .01$). When pooled correlation coefficients were calculated to account for carcass differences between selected lines, most r values were not significant (table 3). Therefore, the investigator must be aware of any breed differences that may affect the direct relationship between two variables.

Other hormones such as glucocorticoids and thyroxine (PBI) have been examined in relation to growth and carcass traits with relatively the same results as the growth hormone studies. The correlations between measured levels of endogenous hormones (method 2) and production traits have not always been meaningful, due to variability of hormone level within each individual animal.

Since single measurements of endogenous hormone level may not always reflect the behavior of the hormone in the animal under investigation, a more informative approach to the hormone-function relationship is method 3. By this method, metabolic clearance (MCR) and secretion rates (SR) of a hormone are measured so that utilization rate may be determined. Through measurement of MCR and SR, along with pituitary and serum hormone levels, hopefully, all of the relationships between pituitary, circulating and utilization rates of a particular hormone may be identified (figure 2).

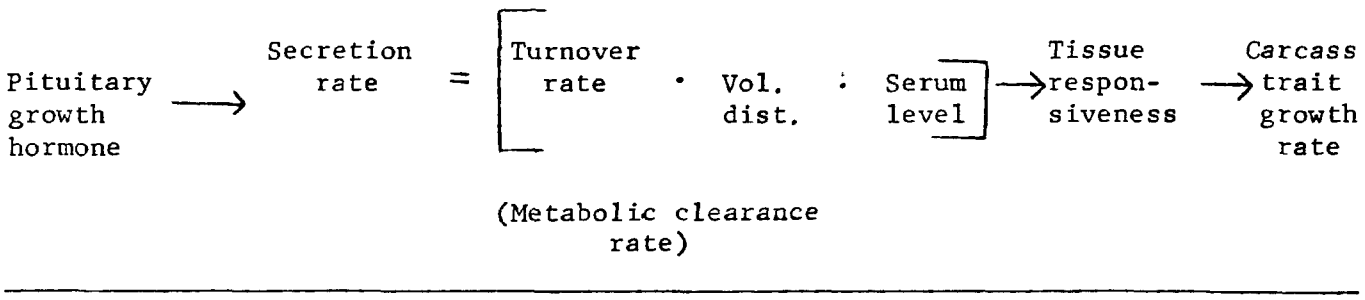
I will discuss this method using data from Duroc swine selected for low (lean) or high (obese) backfat thickness. The pigs used in this study were from the 18th generation of selection for leanness or fatness (Hetzer and Miller, 1973). An unselected control line was also sampled.

Table 3. Simple and pooled correlation coefficients (r) for pituitary growth hormone vs. carcass traits

Carcass trait	r values	
	Simple	Pooled
Fat cuts as % slaughter weight	-.59**	.13
Lean cuts as % slaughter weight	.56**	.15
Backfat thickness	-.59**	-.07
Loin eye area	.57**	-.20*

* $P < .05$.
** $P < .01$.

Figure 2. Relationship between parameters of hormone measurement.



Clearance and secretion rates were determined by a priming dose, continuous infusion technique (Tait and Burstein, 1964). The technique involves the administration of exogenous hormone and the measurement of endogenous hormone and is, therefore, a combination of the two methods discussed earlier. The animals were sampled at two stages of development. The first sampling was at 15 weeks of age, when the accretion of protein and fat occur at about the same rate and the lean to fat ratio is greater than one in all lines. The second sampling was at 90 kg body weight. In swine at this stage of development, rate of protein accretion has decreased and the fraction of weight gain due to fat has increased so that the lean to fat ratio is less than one (Davey et al., 1969).

Carcass data (table 4) shows some differences between the swine at 15 weeks of age; however, the low backfat pigs were significantly lighter than high backfat pigs ($P < .05$). Usually, at this age, growth rates among lines are not consistently different. More typical differences are observed at 90 kg body weight (table 5). The lean pigs were significantly taller, longer and trimmer than the obese pigs.

At 15 weeks of age, the lean swine had lighter anterior pituitaries than the control line, thus, the lean swine had greater concentrations of pituitary growth hormones than the controls ($P < .05$) (table 6). None of the other data for growth hormone were significantly different.

At 90 kg body weight the lean swine again had greater concentrations of pituitary growth hormone than the control swine and, in addition, had slightly higher values for serum growth hormone than the obese swine (table 7). However, as in the younger swine, none of the clearance or secretion data were significantly different.

Since rates of protein and fat deposition, as well as lean to fat ratios, were different in swine at the two stages of development, data for lines at 15 weeks of age and at 90 kg weight were pooled to determine whether growth hormone data were also different (table 8). Since variation in body weight between the two stages (about 35 kg vs. 90 kg) was considerable, MCR and the SR were also calculated on the basis of body weight.

Concentrations of pituitary growth hormone were not significantly different. However, the older pigs had significantly heavier pituitaries, ($P < .01$) hence, significantly higher content of pituitary growth hormone ($P < .01$).

Although the levels of serum growth hormone were not significantly different in pigs at the two stages of development, growth hormones had a shorter half life and greater distribution in the younger pigs. These findings suggest that growth hormone was used more rapidly in the younger swine. This concept is supported by the observation that growth hormone, expressed in units per kg body weight, was secreted at almost a three fold greater rate in the younger as in the older animals (22.1 vs. 8.0 ng/min/kg body weight). Therefore, although levels of serum growth hormone were

Table 4. Carcass data for Duroc swine at 15 weeks of age selected for backfat thickness

Variable	Line[a]			Prob > t		
	High	Control	Low	H vs L	H vs L	H vs L
Slaughter weight, kg	39.3 ± 1.5	34.2 ± 5.8	25.2 ± 4.5	.05	-	-
Body length, cm	52.1 ± 1.0	55.2 ± 1.3	50.1 ± 3.7		-	-
Loin length, cm	43.7 ± 0.8	44.9 ± 3.1	40.8 ± 3.2	-	-	-
Loin eye area, sq cm	12.5 ± 0.5	16.6 ± 2.9	13.4 ± 1.9	-	-	-
Lean cuts, as percent slaughter wt	37.1 ± 1.1	44.1 ± 1.6	40.3 ± 1.1	-	.05	-
Fat cuts, as percent slaughter wt	14.0 ± 0.7	7.9 ± 1.0	4.5 ± 0.5	.01	.01	.05
BF thickness, mm	36.4 ± 0.8	22.1 ± 3.2	7.8 ± 1.2	.01	.05	.05
Femur length, mm	142.0 ± 2.0	141.3 ± 7.6	137.3 ± 8.7	-	-	-

[a] 3 observations per line.

Table 5. Carcass data for Duroc swine at 90 kg body weight selected for backfat thickness

Variable	Line[ab]			Prob > t		
	High	Control	Low	H vs L	H vs C	L vs C
Slaughter age, weeks	31.6 ± 2.2	30.4 ± 1.3	30.6 ± 1.0	-	-	-
Body length, cm	66.0 ± 2.0	72.2 ± 0.5	76.8 ± 0.6	.01	.05	.01
Loin length, cm	54.3 ± 1.2	60.0 ± 0.3	62.7 ± 0.5	.01	.01	.01
Loin eye area, sq cm	18.1 ± 1.0	34.6 ± 0.8	36.3 ± 1.2	.01	.01	-
Lean cuts, as percent slaughter wt	35.0 ± 1.1	45.1 ± 0.6	47.5 ± 0.8	.01	.01	.05
Fat cuts, as percent slaughter wt	22.4 ± 1.6	12.4 ± 0.7	5.6 ± 0.4	.01	.01	.01
BF thickness, mm	66.7 ± 3.8	40.3 ± 2.1	21.6 ± 1.7	.01	.01	.01
Femur length, mm	176.0 ± 2.9	188.6 ± 2.0	210.4 ± 4.8	.01	.01	.01

[a] 5 observations per line.
[b] Mean ± s.e.m.

Table 6. Porcine growth hormone (GH) data for lean, obese and control swine at 15 weeks of age

Line[1]	Total pit. GH (ug)	Pit. GH conc. (ug/mg)	Serum GH (ng/ml)	$t_{\frac{1}{2}}$ (min)	Metabolic clearance rate (ml/min)	Secretion rate (ng/min)
Obese	3551 ± 746[2]	27.5 ± 4.5[a,b]	2.24 ± 0.64	7.4 ± 1.6	341 ± 114	907 ± 413
Control	3386 ± 408	22.9 ± 2.6[a]	2.68 ± 0.31	8.9 ± 1.6	279 ± 153	802 ± 488
Lean	3145 ± 608	36.9 ± 2.3[b]	3.22 ± 0.12	9.8 ± 0.2	158 ± 45	520 ± 167

1 3 observations within each line.
2 Mean ± standard error of the mean.
a,b Means within a column which have different letter superscripts are significantly ($P < .05$) different.

Table 7. Porcine growth hormone (GH) data for lean, obese and control swine at 90 kg body weight (30 weeks of age)

Line[1]	Pit. GH ug	Pit. GH conc. (ug/mg)	Serum GH (ng/ml)	$t_{\frac{1}{2}}$ (min)	Metabolic clearance rate (ml/min)	Secretion rate (ng/min)
Obese	5501 ± 714	34.3 ± 6.3[a,b]	1.70 ± 0.23[a]	11.3 ± 1.5	305 ± 70	535 ± 187
Control	6666 ± 548	29.5 ± 2.9[a]	2.21 ± 0.12[a,b]	12.0 ± 0.8	280 ± 26	626 ± 79
Lean	7190 ± 452	42.3 ± 1.9[b]	2.53 ± 0.17[b]	11.7 ± 0.5	336 ± 38	932 ± 130

1 5 observations within each line.
2 Mean ± standard error of the mean.
a,b Means within a column which have different letter superscripts are significantly ($P < .05$) different.

Table 8. Porcine growth hormone (GH) data pooled across lean, obese and control groups of swine at two stages of development

Stage of development[1]	Total pit. GH (ug)	Pit. GH conc. (ug/mg)	Serum GH (ng/ml)	Volume of distribution (percent)	$t_{\frac{1}{2}}$ (min)	MCR (ml/min)	MCR/ kg body wt.	SR (ng/min)	SR/ kg body wt.
15 weeks of age	3361 ±308	29.1 ±2.6	2.71 ±0.25	9.5 ±1.6	8.7 ±0.7	259 ±63	7.7 ±1.5	743 ±199	22.1 ±5.0
90 kg body weight	6452 ±364	35.4 ±2.6	2.14 ±0.13	5.5 ±0.4	11.6 ±0.6	317 ±27	3.6 ±0.3	698 ±87	8.0 ±1.0
Prob > t	.01	--	--	.05	.01	--	.05	--	.05

[1] 9 observations at 15 weeks of age.
15 observations at 90 kg bodyweight.

about the same, more growth hormone was available per unit of tissue in the younger animal; and growth hormone was turned over more rapidly in the younger animals, as evidenced by the significantly shorter half life.

The significantly greater distribution of growth hormone in the younger animals, increased exposure of body tissue to the circulating hormone. This effect supports the "dilution" theory, which contends that rapid growth can occur only as long as the ratio of circulating growth hormone to unit of body tissue is sufficiently high to stimulate enough protein synthesis to cause muscle growth (Nalbandov, 1963).

The shorter half life may indicate that the target tissues in the younger animals used or cleared growth hormone faster than tissues in older animals. Therefore, the decline in muscle growth with increasing maturity may be due, in part at least, to a combination of the "dilution" effect and failure of target tissue to respond to the growth hormone at the same rate as tissues in young animals.

The results of this study suggest that growth hormone is not a primary cause of obesity in swine genetically selected for backfat thickness. Also, growth hormone (pituitary, serum, MCR or SR) appeared not to be altered with obesity as is sometimes observed in humans (Glick et al., 1965).

Measurements of clearance and secretion rates, for examination of hormone-function relationships, are difficult to make; and, therefore, the number of experimental units which can be used is limited.

In summary, three methods for determining hormone-function relationships have been discussed. Functions of single hormones are difficult to assess because of the many synergistic and antagonistic actions among hormones. In addition, variables such as sex, species and age of the experimental animal must be taken into account. Some hormones, under proper conditions, can enhance growth and increase the ratio of lean to fat. However, use on a production wide basis has been limited by hormone availability or has been restricted, due to harmful residues.

Once we can determine the hormonal mechanisms for controlling growth and fattening, we may be able to develop techniques to prolong the period and the efficiency of protein synthesis and to delay the period of rapid fat accretion often associated with maturity. Development of these techniques might allow continued supply of wholesome, high quality meats and reduce the energy wastage associated with excessively finished slaughter animals.

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