Fat deposition results in an increase in the mass of adipose tissue within the body. This fatty tissue was long regarded as metabolically inert until the classical work of Rittenberg and Schoenheimer (1937) demonstrated that deuterium labelled fatty acids had a half life of only seven days in the mouse. It is now known that adipose tissue has a number of enzyme systems involved in the deposition, synthesis and mobilization of fat, a comparatively dense capillary network and is innervated by the sympathetic nerve fibers (Tepperman, 1962).

The deposition and accumulation of large quantities of fat in humans is known as "obesity", whereas in meat animals a similar condition is referred to as "overfinished" or "wasty". I point this out to remind you that these are really synonymous and while the concern in these problems may be different, the processes involved in producing the conditions are most likely very similar. At the present time there is more in-depth information available on the basic causes of fat accumulation in humans and experimental animals (Kaunitz, 1966; Reichard, 1966) than is available for meat animals (Byerly, 1962). With this in mind we can now approach the question of the causes of fat deposition relating whenever possible specifically to the porcine animal.

Perhaps initially we should understand why the deposition of fat in the porcine animal is important. As in all red meat animals, we are faced with a paradox in that the site of fat deposition determines whether or not the fat is desirable or undesirable. For example a certain degree of intramuscular fat is desirable for optimum organoleptic properties (Carpenter et al., 1963; Kauffman et al., 1964) whereas the quantity of fat in other depots must be at a minimum for optimum cutability. These concepts are reflected in the standards for the Federal Pork Grades. With this in mind the following slides depict recent changes in fat deposition that have occurred in the swine population of the United States and the economic implications of these changes.

As shown in slide 1, the average lard production per pig marketed in the U. S. has decreased from about 50 pounds in 1948 to about 25 pounds in 1967. This decrease in average lard production per pig has occurred while average market weight has remained quite constant. Therefore these figures reflect a net decrease in the ability of pigs to deposit fat in their carcasses. It is also probable that the cuts are trimmed more closely today and more of the fat is rendered than was true 20 years ago.

The economic significance of this decreased fat deposition is illustrated in slide 2 by comparing the improvement in pigs that has taken place between 1958 and 1967. During this period of time an average of 6.7 pounds of lard or about 8.9 pounds of adipose tissue was replaced by other tissues, namely muscle. Based on 1967 prices, the 8.9 pounds of adipose
tissue would have cost $1.70 on the live animal basis, but in the form of lard would have been worth only 52 cents, representing a $1.18 loss in value. Applied to all pigs marketed in 1967 this amounts to a savings of over $98 million.

While this represents a very substantial decrease in fatness of the pork carcass in less than a decade, indications (personal communication) are that a small percentage of the pork carcasses today qualify as U. S. No. 1 Grade carcasses because of excess backfat. Studies with a limited number of pork carcasses (Allen, 1965; Kauffman and St. Clair, 1965) indicate that U. S. No. 1 Grade carcasses contain greater than one-third of their carcass weight in extractable lipid.

This brings us to the topic of when and how fat is deposited in the porcine animal. Slide 3 shows daily growth rate of muscle and fat at different live weights for the Landrace pig as reported by Clausen, 1953. As pointed out by Hammond (1962) the declining rate of growth in muscle may begin at different times depending upon the breed, but for these particular data it would appear to be at about 200 pounds. Thus the average market weight of about 240 pounds shown in the first slide for pigs marketed in the U. S. indicates that they are probably well into the fattening phase of growth.

What are the factors responsible for the deposition and accumulation of lipid or fat in the animal? Deposition of lipid in adipose tissue is the result of two processes: incorporation of preformed lipid from the circulatory system, and de novo synthesis of lipid from precursors directly in the adipose cell itself (Steiner and Cahill, 1963). The preformed lipid may be of dietary origin or synthesized in the liver and then transported to the adipose tissue. Particularly appropriate to this discussion is the fact that Longnecker (1941) cites the classical work of Lawes and Gilbert (1853) with porcine animals as being the first proof that carbohydrate is converted into fat by the animal. There is now evidence to indicate that adipose cells are the main site of fatty acid synthesis (Kaunitz, 1966). Thus to accomplish the synthesis of fatty acids, a number of enzyme systems are involved. Gibson (1965) has depicted the role of some of the adaptive enzyme systems in slide 4. The numbers in this scheme refer to enzymatic steps which respond to adaptive enzyme formation. These include: 1) glucokinase, 2) glucose-6-phosphate dehydrogenase, 3) citrate cleavage enzyme, 4) acetyl CoA carboxylase, 5) fatty acid synthetase and 6) the oxidative desaturase system. You will note that citrate activates the acetyl CoA carboxylase system which in turn is inhibited by the product of these reactions, free fatty acids. The majority of the fat present in the adipose cell is in the form of triglycerides. An essential requirement for triglyceride synthesis is alpha glycerol phosphate which is formed from glycerol and ATP by glycerokinase. Since the adipose tissues that have been studied in experimental animals indicate that there is little, if any glycerokinase activity, all of the alpha glycerol phosphate must originate from metabolism of glucose in other tissues (Tepperman, 1962). As shown in this slide, the presence of alpha glycerol phosphate in adipose tissue can serve to remove the inhibition of fatty acid synthesis by promoting triglyceride synthesis (Masoro, 1962). It does not seem unreasonable to suggest that differences in enzymatic mechanisms such as these could be responsible for the breed and within animal variations for fat deposition that are found in meat animals.
Adipose tissue contains at least two lipolytic enzyme systems. Both of these systems are shown in a schematic illustration in slide 5. The first of these, lipoprotein lipase, is located at or near the capillary endothelium (Robinson and French, 1960), and is involved in the deposition of preformed lipid in the adipose cell. Its function is to hydrolyze the free fatty acids (FFA) from the triglyceride protein complex, which may be either lipoproteins or chylomicrons, and thereby enable the FFA to enter the adipose cell. The activity of lipoprotein lipase is elevated by insulin or consumption of a fatty meal (Tepperman, 1962). Its activity could be a controlling factor in the deposition of fat at a particular site. After entering the adipose cell the FFA are esterified with alpha glycerol phosphate and deposited as triglyceride. During periods of energy need the adipose tissue triglyceride is broken down into glycerol and FFA which are mobilized into the blood stream for transport to the tissues where energy is needed. The lipase enzyme which mobilizes FFA from adipose tissue is influenced by a number of hormones and is normally quite active during periods of fasting or starvation. The sympathetic nerve fibers which innervate adipose tissue and release norepinephrine upon stimulation are believed to play a key role in the physiological monitoring of this lipolytic system (Jeanrenaud, 1961). Kaunitz (1966) in reviewing the causes of human obesity makes reference to a number of articles which have demonstrated that 1) the action of epinephrine and insulin on the adipose tissue of genetically obese mice is reduced; 2) the response of different fat depots to reducing diets is quite different and 3) there are certain adaptive enzyme changes which lead to suppressed mobilization of fatty acids during starvation. Kaunitz (1966) also suggests that the structural arrangement of the fatty acids on the triglycerides may be a factor contributing to differences in lipolysis rates of different fat depots. In the pig Bowland and Standish (1966) have shown that backfat thickness was significantly reduced by a 48-hour fast. However, Davidson et al. (1968) found no difference in backfat thickness for pigs fasted 68 or 70 hours but they did report a significant reduction in leaf fat weight of fasted pigs in one experiment. These reports suggest that there is some variation between groups of pigs in the manner that their fat depots respond to fasting. Kauffman (1959) and Zessin et al. (1961) have reported that pigs fasted or placed on a severe dietary restriction for extended periods of time have a reduction in backfat thickness and intramuscular fat.

Hertelendy et al. (1966) and Cunningham and Friend (1965) have demonstrated that the adipose tissue of porcine animals is quite responsive to epinephrine administration as evidenced by elevated plasma free fatty acid levels. It has also been reported that epinephrine administered in vivo to the pig exerts a potent inhibitory effect on insulin release even though blood glucose levels were high (Hertelendy et al., 1966). Cunningham (1965) demonstrated that the oral feeding of nicotine, which stimulates the release of epinephrine, causes a decrease in the quantity of fat deposited in the pork carcass. These reports serve as examples of changes in fat accumulation that probably involve the lipase enzyme in the adipose tissue. Since it is known that growth hormone (Rabinowitz et al., 1965) causes an increase in the mobilization of fatty acids from adipose tissue, it is interesting to speculate what influence the selection of meatier pigs has had on growth hormone levels during the last decade or two. Perhaps Dr. Gerrits can shed some light on that subject.
Another particularly appropriate example of ways that the rates of fat accumulation can be influenced or altered is found in the feeding frequency experiments conducted at a number of stations. The initial results reported by Allen et al. (1963) are shown in slide 6. These results were obtained on one litter of pigs (two barrows and two gilts/treatment) fed the same amount of feed either in a single 2-hour feeding or equally divided into 6 feedings over a ten-hour period. The trial was initiated at an average live weight of 20 kg and terminated at an average live weight of 100 kg. The total test period gains for both lots were within 3 kg. The results show that single feeding as compared to multiple feeding significantly reduced the quantity of leaf fat and total extractable fat in the carcass. There was also a tendency for haemsean fat scores and longissimus dorsi marbling scores to be lower. These findings have since been verified in studies reported by Anderson et al. (1965) and Friend and Cunningham (1967). These results for the porcine animal are the opposite of the response obtained in the rat (Tepperman and Tepperman, 1958; Hollifield and Parson, 1962; Cohn and Joseph, 1960) and obese humans (Gordon et al., 1963) where it has been shown that multiple rather than single feedings cause a reduction in fat accumulation. This response in the rat has been attributed to an increase in glucose-6-phosphate dehydrogenase activity in "single feeders" resulting in an accelerated rate of lipogenesis. Cunningham (1967) has recently reported that porcine animals fasted for 24 hours have a greatly increased rate of fatty acid oxidation. This finding suggests that "single fed" pigs mobilize fat for energy with the net result being a smaller accumulation of fat in their bodies than in "multiple fed" pigs. This apparently does not happen in some forms of human obesity (Gordon et al., 1963). These findings with rats, humans and pigs serve to illustrate that what is true for one species may not apply in the same manner to another.

There are a limited number of studies which have employed tracer techniques to study the deposition of fat in porcine animals. Sink et al. (1965) fed an oral dose of radioactive chlorinated mono-oleate to animals which had been fasted 24 hours and ranged in weight from 75 to 190 pounds. Animals were slaughtered 3½ hours after administration of the tracer and the panniculus adiposus (backfat) was assayed for radioactivity over the shoulder, loin and rump areas. Results indicate that 1) greater radioactivity was present in the outside layer of the backfat and 2) activity of the three areas decreased according to loin shoulder rump except for the 190 pound animals where the activity in the shoulder sample was higher than in the loin sample. Cunningham (1967) used carbon 14 labelled glucose, palmitate and valine to study the effect of various fasting periods ranging from one to 24 hours. The results indicate that six times as much C14 palmitate was oxidized when injected after a 24-hour fast as compared to a one-hour fast. An interesting result of these experiments is that very little C14O2 was expired by pigs given an oral dose of C14 palmitate. This is in keeping with the findings of Schoenheimer and Rittenberg (1935) showing that dietary fat is first deposited in the adipose tissue before it is used as metabolic fuel. The results of an additional experiment conducted by Cunningham (1967) to study the distribution of orally administered radioactivity in various tissues are shown in slide 7. These data show that there is a considerable difference in the specific activity of the intramuscular lipid present in the gracilis and longissimus dorsi muscles. It should also be noted that the specific activity of the backfat was greater than the perirenal fat when C14 palmitate was administered. It is important
to keep in mind that these values were determined from animals at the end of a 24-hour fast. Thus, one cannot say from these data whether a high or low count is due to factors associated with deposition or factors associated with lipolysis (mobilization).

No discussion of fat deposition in meat animals should fail to mention the influence of sex. Numerous studies (Cahill et al., 1960; Teague et al., 1964; Allen and Bray, 1964; Doornenbal, 1967; Plimpton et al., 1967; Bruner and Swiger, 1968) have established that the influence of sex upon the quantity of fat present in different depots usually results in boars, gilts and barrows. Recent studies by Baker et al. (1967) have shown that a dietary combination of diethylstilbestrol and methyltestosterone given to barrows and gilts caused a significant decrease in backfat thickness and fat trim plus leaf fat. The response tended to be greater in barrows than in gilts. The systems within the porcine animal responsible for effecting these changes have not been defined, but it seems probable that one or more of the enzymatic mechanisms associated with fat deposition and mobilization would be involved. Studies reported by Allen et al. (1967) suggest that lower intramuscular lipid levels in boars as compared to gilts and barrows are associated with an increased number of muscle fibers capable of mobilizing and oxidizing lipid. This is in keeping with the known catabolic action of testosterone toward fat deposition (Kochakian, 1966).

Because of the importance of marbling or intramuscular lipid to the palatability characteristics of meat, it is important that we consider other factors which pertain specifically to the accumulation of lipid in muscle. Studies by Kelly (1955), Kropf (1956) and Allen (1965) have generally shown that the intramuscular lipid levels in the longissimus dorsi muscle do not increase between about 70 and 205 pounds live weight in the porcine animal. However, Lawrie et al. (1963) have found that the intramuscular lipid level increases progressively between 150 and 260 pounds. Kauffman and Safanie (1967) report that the fascicular organization of the muscle generally parallels the quantity of interfascicular lipid, with the looser fascicular organization being associated with higher lipid levels. It was also shown that the loosely organized muscles accumulated more lipid as maturity of the animal increased. Except for the intrafibrillar lipid, which is probably associated with membranous structures such as mitochondria, the fat that is deposited in muscles is normally between the muscle fasciculi (Kauffman and Safanie, 1967). However, Allen et al. (1967) have reported finding two instances in porcine longissimus dorsi muscle where the muscle cell appeared to be completely replaced by lipid. Such a condition is common in dystrophic muscle (Adams et al., 1962). Other studies by Blumer et al. (1962) and Moody and Cassens (1968) have shown that large intramuscular lipid deposits are associated with the circulatory system. Furthermore, it has been reported (Blumer et al., 1962) that the coarseness of the marbling deposit in beef longissimus dorsi muscle varies between ribs according to the distribution and size of the blood vessels. Helander (1958) has suggested that lipomorphosis (intramuscular lipid accumulation) in human muscle is associated with changes in the blood vessels, since lipomorphosis occurs more frequently in patients with arteriosclerosis.

Another aspect of muscle which may influence its rate of lipid accumulation is its ability to oxidize fatty acids as a source of energy. A number of studies (Fritz et al., 1957; Friedberg and
Estes, 1962; Masoro et al., 1965) have shown that muscles can readily utilize free fatty acids. Other studies with dogs (Issekutz et al., 1964; Spitzer and Gold, 1964) and humans (Havel et al., 1963) suggest that intramuscular lipid is an important source of metabolic energy particularly in the postabsorptive state. Thus these studies show that muscles do utilize lipid as an energy source and furthermore that the source of lipid may be derived from within the muscles themselves.

In conclusion, it is apparent from studies on porcine animals that more information is needed concerning the metabolism of adipose tissue at different sites within the pork carcass. Numerous studies conducted with experimental animals and humans have defined a number of adaptive enzyme steps in the synthesis, deposition and accumulation of lipid. In the porcine animal, as well as other red meat animals, these processes have economic significance and a more thorough knowledge of the controlling mechanisms should lead to further carcass improvement. For an extensive review of adipose tissue I recommend the Handbook of Physiology text entitled "Adipose Tissue."

**BIBLIOGRAPHY**


Clausen, H. 1953. The Improvement of Pigs. Queen's University, Belfast.


---

SLIDE #1

**LARD PRODUCTION IN U.S.**

<table>
<thead>
<tr>
<th>Year</th>
<th>Average Lard Production/Pig (pounds)</th>
<th>Average Weight Market Pigs (pounds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1948</td>
<td>49.8</td>
<td>253</td>
</tr>
<tr>
<td>1958</td>
<td>31.6</td>
<td>236</td>
</tr>
<tr>
<td>1960</td>
<td>30.4</td>
<td>236</td>
</tr>
<tr>
<td>1962</td>
<td>29.7</td>
<td>239</td>
</tr>
<tr>
<td>1964</td>
<td>28.6</td>
<td>241</td>
</tr>
<tr>
<td>1965</td>
<td>26.8</td>
<td>239</td>
</tr>
<tr>
<td>1966</td>
<td>25.6</td>
<td>242</td>
</tr>
<tr>
<td>1967</td>
<td>24.9</td>
<td>241</td>
</tr>
</tbody>
</table>

USDA Livestock and Meat Statistics, Statistical Bulletin No. 333
SLIDE #2

VALUE OF DECREASED FAT DEPOSITION

1958 U.S. Lard Production/Pig 31.6#
1967 " " " " 24.3#

6.7# less lard/pig

6.7# lard is equivalent to about 8.9# adipose tissue

Average price/pound for market pigs 1967 = $0.1907
" " " " " lard 1967 = $0.078

Therefore: (8.9#)($0.1907) = $1.70 paid for fat on live basis
(6.7#)($0.078) = .52 received for lard

Difference $1.18

Value to Swine Industry in U.S.

83,421,000 pigs marketed 1967
$1.18

$98,436,780 saved from pigs marketed in 1967 as compared to those marketed in 1958 by removing 6.7# lard/pig.

SLIDE #3

GROWTH CURVES

Clausen, H. The Improvement of Pigs, 1953
Queens University, Belfast

Also see: Nutrition of Pigs & Poultry, 1962 J. T. Morgan and D. Lewis, Editors
Adaptive Enzyme Systems in Fatty Acid Synthesis

GLUCOSE

TPNH → GLUCOSE-6-PHOSPHATE

α GLYCEROL PHOSPHATE

PYRUVATE

ACETYL-CoA

ACETYL-CARNITINE

CITRATE

CARNITINE

ACETYL-CoA

MALONYL-CoA

SATURATED FATTY ACIDS

TPNH

UNSATURATED FATTY ACIDS

EXOGENOUS LINOLEIC ACID

TRIGLYCERIDES


ADIPOSE TISSUE LIPASES

TRIGLYCERIDE → PROTEIN

Insulin

Fatty meal

+ +

LIPASE

FFA

ALPHA GLYCEROL PHOSPHATE

FFA + GLYCEROL

Epinephrine, Norepinephrine, ACTH, TSH, Glucagon, GH

Glycerol
INFLUENCE OF FEEDING REGIME ON FAT DEPOSITION

<table>
<thead>
<tr>
<th></th>
<th>MULTIPLE FED</th>
<th>SINGLE FED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average Backfat Thickness (inches)</td>
<td>1.21</td>
<td>1.16</td>
</tr>
<tr>
<td>Total Leaf Fat Weight (lbs.)</td>
<td>3.45</td>
<td>2.70 *</td>
</tr>
<tr>
<td>Ham Seam Fat Score (1-5)</td>
<td>2.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Long. dorsi Marbling Score (1-5)</td>
<td>1.9</td>
<td>1.0</td>
</tr>
<tr>
<td>Fat Extracted From Right Side (%)</td>
<td>36.88</td>
<td>33.72*</td>
</tr>
</tbody>
</table>

* P<.05

Allen, E. et al. JAS 22:825, 1963

SPECIFIC ACTIVITY OF FAT IN TISSUES OF PIGS 24 HR. AFTER RECEIVING 0.1 mc. OF C14-LABELED GLUCOSE, PALMITATE OR VALINE IN THEIR FEED

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Item 1</td>
<td>2</td>
<td>Item 1</td>
</tr>
<tr>
<td></td>
<td>d.p.m./gm. live bodyweight</td>
<td>3,513</td>
<td>3,724</td>
</tr>
<tr>
<td></td>
<td>% of dose expired in 24 hr.</td>
<td>40.2</td>
<td>37.2</td>
</tr>
<tr>
<td></td>
<td>d.p.m./gm. tissue fat</td>
<td>Backfat</td>
<td>2,084</td>
</tr>
<tr>
<td></td>
<td>Perirenal fat</td>
<td>3,210</td>
<td>1,350</td>
</tr>
<tr>
<td></td>
<td>Gracilis m.</td>
<td>3,567</td>
<td>2,315</td>
</tr>
<tr>
<td></td>
<td>L. dorsi m.</td>
<td>2,000</td>
<td>1,794</td>
</tr>
<tr>
<td></td>
<td>Liver</td>
<td>20,031</td>
<td>20,954</td>
</tr>
</tbody>
</table>

Adapted from Cunningham, H. M. JAS 26:1332, 1967
D. A. CRAMER: Gene has done something besides working with chicken fat. There is one reference he made to sex differences, differences between intramuscular fats. Gene reported this work in the paper today, but he very modestly did not refer to the authors who were, in fact, Allen and Cassens, both of whom are with us. I said he wasn't a meats man, but I think we could let him join us now.

Our next speaker, Dr. Ewan, is also not a meats man. He is an Illinois boy who went on to Wisconsin, the same as Gene did, to get his PhD. and he got his PhD. in ruminant nutrition. I asked him what ruminant nutrition had to do with electrolyte and water balance in pigs, and he didn't have a ready answer for that, but he did say, "Well, now, in the last two years, since going to Iowa State, I have been in swine nutrition work," so I guess that does qualify him. I'll turn the program over to Dr. Ewan.