

# LIPID METABOLISM & VITAMINS/MINERAL

## INTRODUCTION

### 1. General

#### A. Lipids:

- 1) Include a group of substances that are insoluble in water, but soluble in ether, chloroform & benzene.
- 2) Include fats, waxes, glycolipids, phospholipids, steroids, prostaglandins, etc.
- 3) "Fats" are by far the most important lipids based on amounts present in the animal body & its food.
- 4) But others also play significant roles in nutrition & physiology - e.g., cholesterol is a precursor of vitamin D and sex hormones, and it is an infamous component of atheromatous plaques of cardiovascular diseases!

#### B. Lipids in diets for nonruminant species:

- 1) Baby pig's diet (milk) consists of 6-8% fat (30-40% on a DM basis). (Others - 80% water, 5-6% protein & 4.5-5% lactose.)
- 2) Lipid content in grains - Corn,  $\approx$  3.6%; milo,  $\approx$  2.8%; barley & wheat, less ( $<$  2%).
- 3) Soybean & other oilseed meals (solvent extracted) are low in lipids ( $<$  2-3%).
- 4) Animal protein sources (fish meal, meat meal, etc.) are relatively high (6-10%).
- 5) Corn-soy-based diets usually contain  $\approx$  2.5-3% fat.

#### C. Some reasons for using feed grade lipids in nonruminant diets:

- 1) To improve growth rate & feed efficiency.
- 2) To reduce dustiness of feed, and also in confinement buildings.
- 3) To  $\uparrow$  energy content of sow's milk,  $\therefore$  increase the survival rate of baby pigs.
- 4) To reduce segregation of smaller particles.
- 5) To facilitate the pelleting process.
- 6) To reduce wear & tear on mixing and handling equipments,

### 2. Classification of Lipids

#### A. Based on the No. of carbon atoms and the degree of unsaturation:

- 1) Saturated fatty acid (SFA) - No double bonds.
- 2) Unsaturated fatty acid (UFA) - One or more double bonds.
- 3) Polyunsaturated fatty acids - Two or more double bonds.

B. Natural lipids (plant & animal origin):

- 1) Made up of triglycerides (glycerol + 3 FA).
- 2) Most FA have 8 to 24 C with 16 to 18 C being common for many feed lipids.
- 3) Short (< 10 C) or medium chain FA - FA with 14 C or less.

3. Physical and Chemical Characteristics of Lipids (Maynard et al., 1979)

	Corn	Soy	Saf- flower	Coco- nut	Past. grass	Butter	Tallow	Lard	Egg
<b>Saturated acids, %</b>									
Butyric C14:0						3.2			
Caproic C6:0				0.2		1.8			
Caprylic C8:0				8.2		0.8			
Capric C10:0				7.4		1.4			
Lauric C12:0				47.5		3.8			
Myristic C14:0			0.2	18.0	1.0	8.3	3.0		0.3
Palmitic C16:0	7.0	8.5	12.3	8.0	16.0	27.0	27.0	32.2	22.1
Stearic C18:0	2.4	3.5	1.8	2.8	2.0	12.5	21.0	7.8	7.7
<b>Total</b>	<b>9.4</b>	<b>12.0</b>	<b>14.3</b>	<b>92.8</b>	<b>21.1</b>	<b>58.8</b>	<b>51.0</b>	<b>40.0</b>	<b>30.1</b>
<b>Unsaturated acids, %</b>									
Palmitoleic C16:1					2.0				3.3
Oleic C18:1	45.6	17.0	11.2	5.6	3.0	35.0	40.0	48.0	36.6
Linoleic C18:2	45.0	54.4	74.3	1.6	13.0	3.0	2.0	11.0	11.1
Linolenic C18:3		7.1			61.0	0.8	0.5	0.6	0.3
Arachidonic C20:4									0.8
<b>Total</b>	<b>90.6</b>	<b>78.5</b>	<b>85.5</b>	<b>7.2</b>	<b>79.0</b>	<b>38.8</b>	<b>42.5</b>	<b>59.6</b>	<b>52.1</b>
Melting point, °C	< 20	< 20	< 20	20-35		28-36	36-45	35-45	
Iodine No.	105-125	130-137		8-10		26-38	46-66	40-70	
Saponification No.	87-93	190-194		250-260		220-241	193-200	193-220	

**ESSENTIAL FATTY ACIDS**

1. Dietary Requirements

A. “Essentiality” of fatty acids:

- 1) Evans & Burr (1926. Proc. Soc. Exp. Biol. Med. 24:740) indicated that “*a component of fat other than fat-soluble vitamins are dietary essential for rats!*”
- 2) Burr & Burr (1929. J. Biol. Chem. 82:345):
  - a) Feeding the diet almost devoid of fat to rats resulted in a poor growth, symptoms of dermatitis, necrosis of tails and death.
  - b) Also observed adverse effects on reproduction & lactation.
  - c) Small amounts of PUFA were effective in preventing/curing those conditions.

∴ they called the PUFA, “Essential Fatty Acids!”

## B. Swine &amp; chicks:

## 1) Demonstration of the essentiality of FA:

- a) For chicks by Reiser in 1950 (J. Nutr. 42:319).
- b) For swine by Whitz and Beeson in 1951 (J. Anim. Sci. 10:112).

## 2) Deficiency symptoms:

- a) Swine - e.g., poor growth, skin lesions, retarded sexual maturity, underdeveloped GI systems, etc.
- b) Birds - e.g., ↓ growth & disease resistance, dermal problems, faulty feathering, fatty livers, ↓ development of secondary sex characteristics, etc.

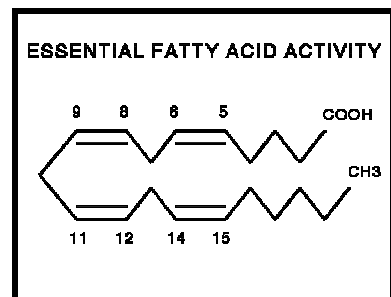
## 2. Essential Fatty Acid Activity

## A. Essential FA “activity,” NOT “essential FA?” - Possible reasons?

- 1) Interconversions among FA, i.e., FA provided in the diet may not be the one that is responsible for alleviating the deficiency symptom(s)!
- 2) Fatty acids are involved in a wide range of metabolic processes in animals:
  - a) May exhibit many manifestations of dietary essential FA deficiencies.
  - b) May respond differently to various FA depending on deficiency symptoms.

## B. Fatty acids to be active:

- 1) Important to have unsaturated bonds between carbons 6-7 and 9-10 from the methyl end of FA chain [ . . . known as omega ( $\omega$ ) carbon], which give FA the correct configuration!
- 2) Activity of various FA:



- a) Linoleic acid (US bonds at 6-7 & 9-10 positions) - Has a 100% activity, and animals can synthesize arachidonic acid from linoleic acid.
- b) Arachidonic acid (US bonds at 6-7, 9-10, 12-13 & 15-16 positions) has a 100% activity.
- c) Oleic acid (an US bond at 9-10 position) has no activity because animals cannot unsaturate the 6-7 bond.
- d) Linolenic acid (US bonds at 3-4, 6-7 & 9-10 positions) - Not effective because the “3-4” bond destroys a critical configuration, and although animals can saturate this bond, not efficiently, ∴ has a limited activity.

## 3) Essential FA:

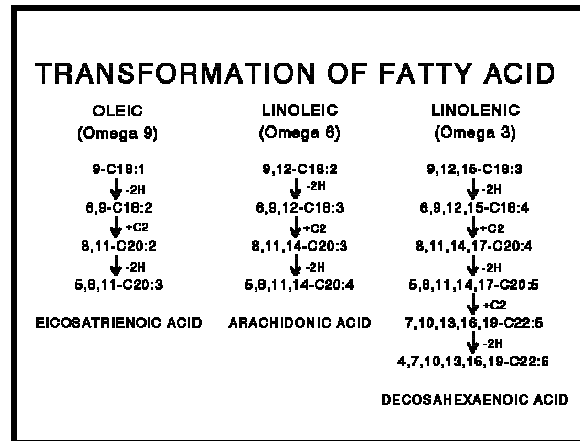
- a) From a metabolic standpoint, “arachidonic acid” is the essential FA.
- b) From a dietary standpoint, “linoleic acid” is the essential FA because of:

- (1) Conversion of linoleic to arachidonic acid.
- (2) Low arachidonic acid contents in feeds.

## C. Metabolic transformation of FA:

- 1) Conversion by microsomal chain elongation or desaturase system.
- 2) Competition among series because of the use of the same enzyme systems:
  - a)  $\omega$ -3- &  $\omega$ -6-family can suppress metabolism of each other.
  - b)  $\omega$ -6 family can suppress formation of PUFA from oleic acid.

- ☞ Affinity for enzymes?  
 Linolenic ( $\omega$ -3) > linoleic ( $\omega$ -6) > oleic ( $\omega$ -9)!



- D. The cat family (e.g., cats & lions) - Unable to desaturate linoleic & linolenic acids (NRC, 1986), ∴ may require specific polyunsaturated FA of animal origin.

## 3. Functions of Essential Fatty Acids

- A. Important components of cellular membranes and subcellular structures (e.g., mitochondria) - Present as phospholipids & provide “fluidity” to the membrane, which is essential for cellular functions.
- B. Involved in the synthesis of arachidonic acid derivatives, which are synthesized and incorporated into the phospholipids of cell membranes - e.g.:
  - 1) Prostaglandins - Involved in vasoconstriction/vasodilation, ♀ reproductive cycles, lipid metabolism, etc.
  - 2) Prostacyclin - Involved in vasodilation, inhibition of platelet aggregation, etc.
  - 3) Thromboxanes - Involved in vasoconstriction, stimulation of platelet aggregation (clotting), etc.
  - 4) Leukotrienes - Mediators of allergic response & inflammation, also potent vasoconstrictors, etc.

4. A Source of Linoleic Acid?

A. Linoleic acid - Sources: [Stahly, 1984. In: Wiseman (Ed.), Cromwell. Pers. Comm, and NRC, 1988]

Source	Percent	(NRC, 1988)
Safflower oil	78	
Sunflower oil	68	
Corn oil	55	(58.0%)
Soybean oil	50	(65.7%)
Cottonseed oil	50	
Peanut oil	27	
Poultry fat	25	(11.8%)
Lard	10	(18.3%)
Fish oil	2.7	
Beef tallow	1.5	(3.1%)
Milk fat	1.5	
Coconut oil	1.5	
Corn	1.8	
Oats	1.5	
Wheat	.6	
Barley	.2	
Soybean meal	.3	

B. Animal fats tend to be low in linoleic acid.

C. Plant oils tend to be high in linoleic acid, especially in forage lipids - e.g., pasture grasses contain ≈ 60% of lipids as linolenic acid.

D. The content and(or) type of animal fats can be influenced by the concentration and type of dietary lipids!

1) Effect of various “oils” on carcass fatty acids in pigs: (Maynard et al., 1979)

	Firm-ness	Melt. pt, °C	Iodine No.	Oleic	Lino-leic	Tot. SFA
Peanut, 4.1%	Medium	34.3	72	47.9	<b>13.8</b>	32.5
Cottonseed, 4.1%	Hard	45.3	64	35.9	15.7	<b>43.0</b>
Soybean, 4.1%	Medium	31.2	76	43.3	18.6	33.8
Corn, 4.1%	Medium	36.3	76	45.0	16.8	33.0
Corn, 11.5%	Oily	24.5	97	41.4	<b>31.4</b>	<b>23.1</b>

\* Tot. SFA = total saturated fatty acids; Lard = ≈ 40% saturated FA & ≈ 11% linoleic acid.

2) “Soft” pork:

a) Oily & difficult to handle.

b) Fats are unstable, ∴ susceptible to rancidity. (Not a major problem today because of refrigeration! But, still . . .?!) )

☞ **The bottom line?** If consumers demand meat products with “less saturated fat or more linoleic acid,” can be done by dietary manipulations!

## 5. Fatty Acid Requirements

### A. Birds (linoleic acid): (NRC, 1994)

- 1) Poultry (chickens, hens & broilers) - 0.83 (hens with 120 g of feed/day) to 1.25% (hens with 80 g of feed/day), with 1.00% for all others.
- 2) Turkeys - 0.8% (8-24 wk & breeders/holding), 1.0% (up to 8 wk), and 1.1% for laying hens.

### B. Swine (linoleic acid):

- 1) ARC, 1981 - 3 & 1.5% of dietary DE for pigs up to 30 kg & from 30-90 kg, respectively.
  - 2) NRC, 1998 - 0.10% for all classes of pigs.
- ☞ These levels are usually present in typical cereal-protein supplement-based diets (e.g., corn, 1.8% & soy, 0.30%).

### C. Fish: (NRC, 1993)

- 1) Fresh water fish generally require either dietary linoleic acid or linolenic acid, or both - 0.5 to 2.5% depending on estimates/species.
- 2) Marine fish require dietary eicosapentaenoic acid [EPA; 20:5 (n-3)] and(or) docosahexaenoic acid [DHA; 22:6(n-3)] - 0.5 to 2% of EPA & DHA depending on estimates/species.

### D. Factors that influence the “essential FA deficiency,” ∴ the requirement:

- 1) Age & carryover effects (e.g., from the egg to chick).
- 2) Growth rate.
- 3) Sex - ♂ may need more (e.g. in rats, 10-20 mg for ♀ vs > 50 mg/d for ♂).
- 4) Humidity & water balance - Related to dermal conditions.

## FATTY ACIDS AND HUMAN HEALTH

### 1. $\omega$ -3 Family (Linolenic) PUFA

#### A. Health benefits (based on epidemiological studies)?

- 1) A low death rate from a coronary heart disease (CHD) among Greenland Eskimos (subsist entirely on a marine diet high in  $\omega$ -3 FA).
- 2) Lower death rate from CHD in Japan (higher fish consumption).

- ☞ The “relationship?” - Originally hypothesized to be via antithrombotic effects, i.e., ↓ platelet adhesion & aggregation!

B. The evidence? (e.g., Wallingford et al., 1991. Nutr. Rev. 49:323)

- 1) “Fish or fish oil?” (Most of fish consumed in the Eskimo studies were not high in  $\omega$ -3 FA!)
  - a) Positive or no response in some studies on fish/fish oil, and marginal effect in other studies.
  - b) Cannot distinguish between effects of fish consumption or fish oil consumption *per se* in studies with a positive response.
- 2) Primary endpoints should be myocardial infarction & death from CHD! - Only one 2-yr prevention study to date, in which reported the ↓ death from CHD but no ↓ in non-fatal myocardial infarction. (Made no comparison of the effects of fish or fish oil consumption in that study, ∴ . . . ?)
- 3) Blood lipids:
  - a) A widespread agreement that fish oil ↓ TG & VLDL in subjects with high initial values.
  - b) The importance of TG level in CHD-risk is still a matter of debate!
  - c) In many studies, observed no effect of fish oil on a total serum cholesterol, LDL or HDL level. (One study with a positive response (i.e., ↓ total cholesterol by feeding 30-40% calories from fish oil) was confounded with PUFA.)
4. Blood pressure:
  - a) Observed ↓ BP with  $\omega$ -3 FA in hypertensive persons.
  - b) Observed ↓ BP with fish oil in a large study with normal healthy subjects, but also observed comparable ↓ with "olive oil" placebo, ∴ the effect was not specific to fish oil.
- 5) Thrombosis:
  - a) Spontaneous platelet aggregation has been reported to be inversely related to an occurrence of myocardial infarction & CHD death in survivors of heart attacks.
  - b) Observed ↓ platelet adhesion & aggregation & ↑ bleeding time with fish oil consumption.
  - ☞ This might be an important line of evidence that would support health claim/message for  $\omega$ -3 FA.

c) Prolonged bleeding time: (McDowell, 1989)

- (1) May ↓ a platelet plug formation in damaged blood vessels.
- (2) May inhibit vessel wall-induced clotting of plasma.

6) Vessel wall effects:

- a) Observed ↓ production of superoxide, interleukin-1 & tumor necrosis factor from leukocytes among "fish oil-supplemented normal subjects."
- b) Also, observed ↓ production of platelet dependent growth factor & endothelium-derived relaxation factor in rats supplemented with fish oil.
- ☞ All these effects may ↓ the progression of early stages of atherosclerosis!

## 2. Linoleic Acid ( $\omega$ -6)

A. Suggested beneficial effects of linoleic acid? (Vergroesen, 1977. Nutr. Rev. 35:1)

- 1) ↓ blood cholesterol & TG levels.
- 2) ↓ thrombotic tendency of platelet.
- 3) Preventive & curative effects in a Na-induced hypertension.
- ... , etc.

B. ↑ linoleic acid intake: (McDowell, 1989)

- 1) Mechanisms of these responses/beneficial effects summarized by Vergroesen (1977) are unknown, or not clearly established.
- 2) Prostaglandins:
  - a) Pharmacological data - Atherosclerosis-promoting factors (hypertension, ↑ thrombotic tendency of platelet) can be counteracted by arterial dilation, and ↑ water & Na diuresis induced by certain prostaglandins.
  - b) Preventive & curative effects of linoleic acid on atherosclerotic syndrome may be explained by ↑ prostaglandin synthesis.

## DIGESTION AND ABSORPTION

### 1. Pre-Duodenal Digestion

- A. Intra-gastric lipolysis has been demonstrated in rats and humans.
- B. Has not been described or demonstrated in the pig or chick, but probably exists.
- C. It is likely that both oral and gastric lipases operate in the stomach, i.e., the initial modification of dietary lipids.

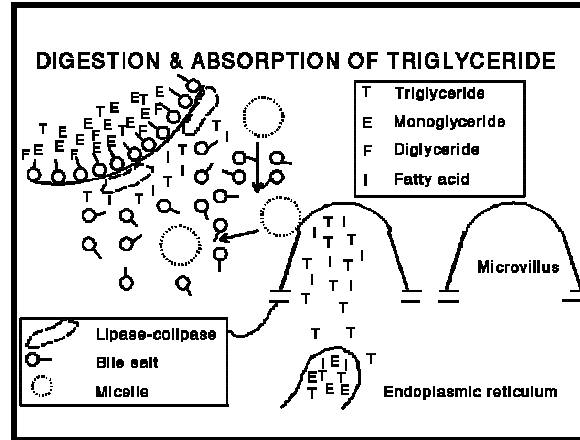
☞ Contribution(s) to the overall digestion - ???

## 2. Digestion & Absorption of Triglyceride

A. Triglyceride (TG) absorption: (Maynard et al., 1979) [Should be E = TG & T = monoglyceride!]

B. Brief Summary

- 1) A coarse emulsion enters the duodenum from the stomach or gizzard.
- 2) Bile salts interact with fat droplets to form emulsion droplets, and along with lipase & colipase, reduce lipids to finer emulsions.
- 3) Lipase and colipase:



- a) Hydrolyze TG droplets into FA and monoglycerides.
- b) Preferentially remove FA in 1 & 3 positions, leaving 2-monoglycerides.
- c) Colipase & bile salt both needed for the lipase activity?

(1) Without colipase or bile salt, lipase would be absorbed and denatured at the interface.

(2) With bile salt but no colipase, lipase remains in the aqueous phase.

- d) Colipase is required for the attachment/function of lipase at the substrate-water interface.

C. Formation of micelles:

- 1) Consist of 2-monoglycerides, FFA & bile salts.
  - 2) Outside, polar (hydrophilic) & center, non-polar (lipophilic).
- ☞ The rate of formation is a critical step in fat digestion/absorption!

D. Migration of micelles to the brush border (lower duodenum):

- 1) Micelles are disrupted.
- 2) FA & monoglycerides are absorbed.
- 3) Bile salts are reused, or eventually absorbed at the lower tract & recirculated via the liver.

- E. Absorbed monoglycerides and FA are resynthesized into TG and phospholipids.
- F. TG are combined with cholesterol & phospholipids to form chylomicron (pig) or very low density lipoprotein (fowl):
- 1) Apoprotein B:
    - a) Synthesized by the rough ER, and being incorporated into lipoproteins in the smooth ER, which is the primary synthetic site of TG.
    - b) Essential for the formation of chylomicron and VLDL.
  - 2) Swine - Absorbed into the general circulation via the lymphatic system . . . The jejunum is a major site.
  - 3) Chicks:
    - a) Via the portal system (the lymphatic system is poorly developed).
    - b) Also, absorbed at the duodenum & ileum.
- G. Glycerol is passively absorbed.
- H. Short-chained FA (< 10 C), which are relatively soluble in water, are absorbed without micelle formation via the portal system.

### 3. Factors Affecting Digestion & Absorption

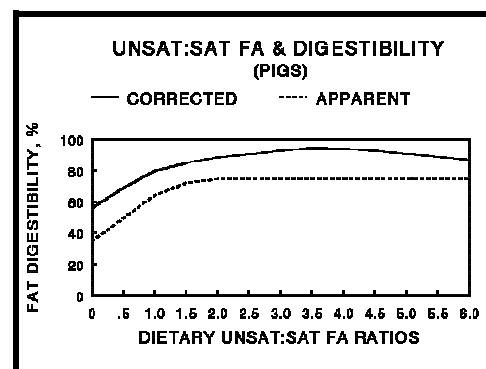
☞ The efficiency of digestion/absorption seems to be associated with the ability to form micelle . . . perhaps, influencing the solubility in bile salt solution!?

- A. Short & medium-chained FA ( $\leq 14$  C) are utilized better vs long-chained FA.
- B. Unsaturated FA are utilized better than saturated FA.
- C. The degree of esterification:

- 1) Removal of FA - > TG > DG > MG.
- 2) Absorption - MG > FA.

D. The ratio of unsaturated/saturated FA:

- 1) "UFA/SFA ratios" & digestibility: [Stahly, 1984. In: Wiseman (Ed.)]



- 2) Effect of dietary fatty acid profile on fat digestion in pigs: [Stahly, 1984. In: Wiseman (Ed.)]

	Supple- mental fat	UFA: SFA ratio	Digesti- bility, %
Corn-soy	Tallow	1.5	85-92
Barley-soy	Tallow	1.0	70-85
Corn-soy	Soy oil	4.8	90-95
Barley-soy	Soy oil	4.0	90-95

☞ When using animal fats, a source of grain may be important, whereas it may not be that important when using plant oils.

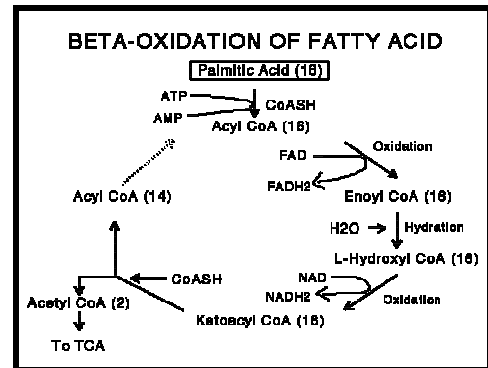
## METABOLISM

### 1. Post Absorption

- A. Enter oxidative pathways for energy production, or
- B. Transported to adipose cells & incorporated into the body fats.

### 2. β-Oxidation

- A. β-Oxidation of palmitic acid: (Adapted & redrawn from Maynard et al., 1979)
- B. Acyl CoA cannot pass into the inner mitochondria membrane alone, thus need a special carrier mechanism, “carnitine transport system (carnitine acyl transferase I & carnitine acyl transferase II).
- B. Remove “2-C unit” at a time & 2-C units enter the citric acid cycle as acetyl CoA.
- C. Glycerol enters the glycolysis pathway through triose sugar.



- 1) 2 glycerol → 2 glycerol-P (- 2 ATP).
- 2) 2 glycerol-P → 2 dihydroxyacetone-P (+ 4 ATP).
- 3) 2 dihydroxyacetone-P → 1 glucose (spontaneous).
- 4) 1 glucose → 6CO<sub>2</sub> + 6H<sub>2</sub>O (+ 36 ATP)

☞ Net 38 ATP (or 19 ATP/mole of glycerol).

### 3. The Energy Content of Lipids

- A. A complete oxidation of fat yields 2.25 x more energy vs carbohydrates!

B. e.g., Tripalmitin vs starch:

1) Tripalmitin (806 g/mole):

$C_{51}H_{98}O_6 (+3 H_2O) + 72.5 O_2 \rightarrow 51 CO_2 + 52 H_2O + \underline{7,657 Kcal}$	
Glycerol	19 ATP
Palmitate	
Phosphorylation	- 2 ATP
7 cleavages x 5 ATP	+ 35 ATP
8 acetyl CoA x 12 ATP	+ 96 ATP
Net (129 ATP x 3)	387 ATP
<b>Total</b>	<b><u>406 ATP</u></b>

2) Starch (162 g/mole, glucose basis):

$C_6H_{10}O_5 (+H_2O) + 6 O_2 \rightarrow 6 CO_2 + 6 H_2O + \underline{680 Kcal}$	
Glycolysis	10 ATP
Phosphorylation	- 2 ATP
NADH $\rightarrow$ mitochondria	- 2 ATP
Net	6 ATP
Oxidation of 2 pyruvate	6 ATP
2 acetyl CoA x 12	24 ATP
<b>Total</b>	<b><u>36 ATP</u></b>

3) Based on “gross energy:”

- a)  $7,657 Kcal \div 806 g = 9.5 Kcal/g$  of fat.  
 b)  $680 Kcal \div 162 g = 4.20 Kcal/g$  of carbohydrate.

$$\therefore 9.5 Kcal \div 4.20 Kcal = \mathbf{2.26}$$

4) Based on “ATP production:”

- a)  $406 ATP \div 806 g = .504 ATP/g$  of fat.  
 b)  $36 ATP \div 162 g = .222 ATP/g$  of carbohydrate.

$$\therefore .504 ATP \div .222 ATP = \mathbf{2.27}$$

#### 4. Fatty Acid Synthesis

☞ See some references on the subject [e.g., Martin et al. (1983) & Mayes (2000) in “Harper’s Biochemistry”]

## A. Elongation pathways (2-C unit at a time):

- 1) Uses acetyl-CoA & NADH or NADPH for reduction in the mitochondria.
- 2) Uses malonyl-CoA & NADPH in the microsome.

☞ Both are modifications of the  $\beta$ -oxidation sequence. (Also, can go through shortening by a sequential removal of 2-C units!]

## B. Some dietary lipids:

- 1) Are directly incorporated into the body fat with no loss of heat . . . Assuming adequate energy intake, i.e., dietary lipids are not necessary as a source of energy.
- 2)  $\therefore$  more efficient vs synthesis from  $\text{CH}_2\text{O}$  or others via acetyl-CoA, which results in a loss of some heat.

## C. Extramitochondrial system for de novo synthesis:

- 1) Found in a soluble (cytosol) fraction of many tissues, e.g., liver, kidney, brain, lung, mammary gland, adipose tissue, etc.
- 2) Major products:
  - a) Palmitic acid in liver & adipose tissues.
  - b) Short-chained FA in the mammary gland.

**BROWN ADIPOSE TISSUE**

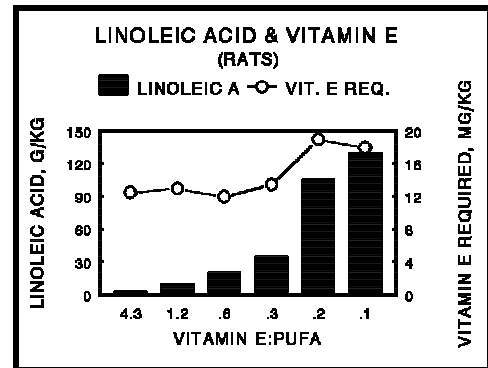
1. A unique adipose tissue located near & around the spinal cord, thoracic organs and kidneys - Localized around some important/vital organs?!
2. Characteristics? - A reddish-brown in color & has a well developed blood supply & high contents of mitochondria and cytochromes.
3. Metabolized within the brown fat tissue itself:
  - A. A poor coupling of oxidative phosphorylation.
  - B. FA oxidation with little ATP formation, and much of energy is released as "heat."
  - C. e.g., "Exposure to cold environment?" - Nerve impulses can lead to release of norepinephrine  $\rightarrow$  activate lipase present in adipose cells  $\rightarrow$  hydrolyze TG to FA and glycerol  $\rightarrow$   $\uparrow$  oxygen consumption and  $\uparrow$  the temperature of the tissue, and warming the blood passing through!
4. May be important when generating heat is necessary/very important, e.g., newborn animals, exposure to cold, arousal from hibernation, etc., i.e., "Non-shivering Thermogenesis!?"

VITAMIN E AND SELENIUM INTERRELATIONSHIPS

1. Introduction

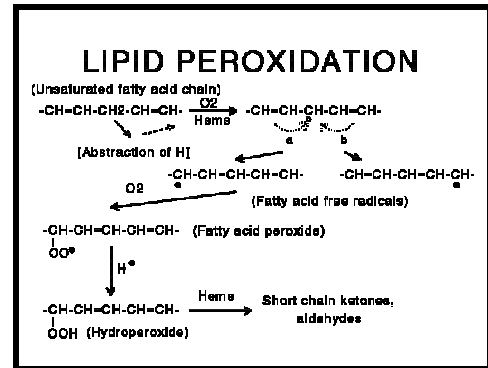
A. Over the years, researchers observed a strong relationship between polyunsaturated FA & vitamin E, i.e., ↑ vitamin E requirement with ↑ dietary PUFA.

- e.g., Effect of linoleic acid intake on vitamin E requirement (rats): (Adapted & redrawn from Jagar, 1972. Ann. NY Acad. Sci. 203:199.)



B. Lipid peroxidation:

- 1) Polyunsaturated FA are highly susceptible to an attack by free radicals and others generated during the metabolic process!
- 2) Lipid peroxidation either by abstraction of H or by addition of OH• (hydroxy radicals).
- 3) Highly reactive intermediates can attack other fatty acids, leading to chain reactions!



C. Polyunsaturated lipids can exacerbate many vitamin E deficiency symptoms.

D. Selenium & synthetic antioxidants have been implicated in the “PUFA-vitamin E relationships:” (Mcdowell, 1989; AO = antioxidant)

Disease	Animal	Tissue	Prevented by			
			E	Se	AO	S-AA
Reproductive failure:						
Embryonic degeneration						
Type A	Rat, hamster, hen, mouse, turkey	Vascular system	X		X	
Type B	Cow, ewe		a	X <sup>b</sup>		
Sterility (♂)	Rat, guinea pig, hamster, dog, rabbit, monkey	♂ gonads	X			
Neuropathy	Chick, human	Brain	X		X	
Liver, blood, brain, capillaries, pancreas:						
Necrosis	Rat, pig	Liver	X	X		X
Fibrosis	Chick, mouse	Pancreas		X		
Erythrocyte hemolysis	Rat, chick, human, dog, monkey	Erythrocytes	X		X	
Plasma protein loss	Chick, turkey	Serum albumen	X	X		
Anemia	Monkey	Bone marrow	X		X	
Encephalomalacia	Chick	Cerebellum	X		X	

- Cont. -

Disease	Animal	Tissue	Prevented by			
			E	Se	AO	S-AA
Cont.						
Exudative diathesis	Chick, turkey	Vascular system	X	X		
Kidney degeneration	Rat, mouse, mink monkey	Kidney tubular epithelium	X	X		
Steatitis (ceroid)	Mink, pig, chick	Adipose tissue	X		X	
Depigmentation	Rat	Incisors	X		X	
Nutritional myopathies						
Muscular dystrophy	Rabbit, monkey, guinea pig, duck, mouse, mink, dog	Skeletal muscle	X		?	
White muscle disease	Lamb, calf, kid, foal	Skeletal & heart muscles	<sup>a</sup>	X <sup>b</sup>		
Type C	Turkey	Gizzard, heart	<sup>a</sup>	X		
Type D	Chicken	Skeletal muscle	X			X
Retinopathy	Dog, monkey, rat	Epithelium	X			
Dermatosis	Dog	Skin				
Immunodeficiency	Dog, chick, pig, mouse, sheep	Reticulo endothelial	X	X		

<sup>a</sup>Not effective if severely deficient in Se; <sup>b</sup>When added to diets low in vitamin E.

## 2. Vitamin E & Selenium as Biological Antioxidants

- A. Vitamin E & Se are both important as biological antioxidants.
- B. Oxidative metabolism produces highly reactive form of oxygen that are highly toxic to organisms - e.g., Superoxide ion, hydroxy radical, hydrogen peroxide and lipid hydroperoxides (UFA + O<sub>2</sub>).
- C. Vitamin E prevents formation of free radicals, which stimulate production of highly-reactive products, within the membrane of cells and organelles.

Thus, **the First line of defense** against peroxidation!

- D. Glutathione peroxidase, which contains Se, eliminates and(or) and prevents formation of peroxides within the cellular interior or cytosol of cells.

Thus, **the Second line of defense** (i.e., destroys peroxides after formation)!

## 3. Interdependence of Vitamin E & Selenium

- A. Se spares Vitamin E at least in 3 ways:
  - 1) Maintains the integrity of pancreas & allows normal lipid metabolism (including metabolism of vitamin E).
  - 2) As an integral part of glutathione peroxidase, reduces vitamin E required to maintain the integrity of cell membranes.
  - 3) May aid in retention of vitamin E in plasma (mechanism, unknown!).

B. Vitamin E reduces the Se requirement by:

- 1) Maintaining body Se in an active form, or preventing its loss.
- 2) Preventing a chain-reactive autoxidation of the membrane, thus inhibiting formation of hydroperoxides, which ↓ the needs for Se-containing glutathione.

### VITAMIN E

#### 1. General

A. Alpha-tocopherol is the most widely distributed vitamin E-compounds in nature & has a greatest biological activity.

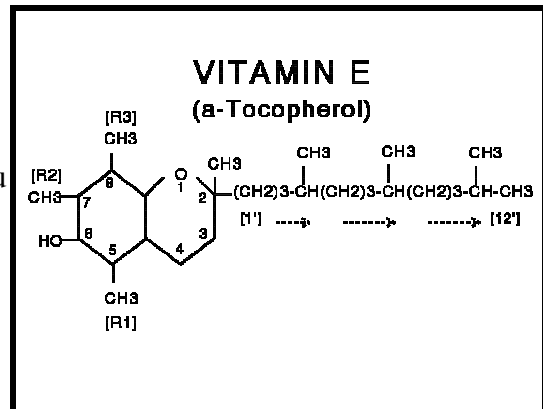
☞ “Tocopherol” - From the Greek words *tokos* (“offspring or childbirth”) and *pherein* (“to bring forth”)!

B. Unit of activity:

- 1) 1 IU of vitamin E = 1 mg of dl- $\alpha$ -tocopherol acetate (synthesized).
- 2) 1 IU of vitamin E = 0.67 mg of d- $\alpha$ -tocopherol acetate (extracted).

C. Natural vitamin E:

- 1) Easily destroyed by oxidation.
- 2) Oxidation is accelerated by heat, moisture, rancid fat & trace minerals (especially by Cu & Fe).



D. Chemical structures:

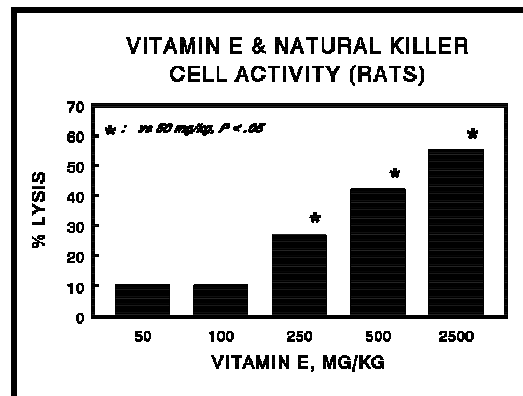
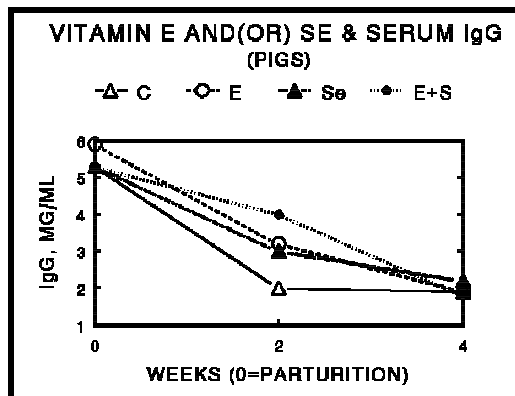
- 1) Structure: (Adapted & redrawn from McDowell, 1989)
- 2) Tocopherols: (McDowell, 1989)

Tocopherol	R <sub>1</sub> (5)	R <sub>2</sub> (7)	R <sub>3</sub> (8)
Alpha	CH <sub>3</sub>	CH <sub>3</sub>	CH <sub>3</sub>
Beta	CH <sub>3</sub>	H	CH <sub>3</sub>
Gamma	H	CH <sub>3</sub>	CH <sub>3</sub>
Delta	H	H	CH <sub>3</sub>
Alpha tocotrienol <sup>a</sup>	CH <sub>3</sub>	CH <sub>3</sub>	CH <sub>3</sub>
Beta tocotrienol <sup>a</sup>	CH <sub>3</sub>	H	CH <sub>3</sub>
Gamma tocotrienol <sup>a</sup>	H	CH <sub>3</sub>	CH <sub>3</sub>
Delta tocotrienol <sup>a</sup>	H	H	CH <sub>3</sub>

<sup>a</sup>Side chain double bonds at 3', 7' & 11' positions.

## 2. Other Functions

- A. May be involved in formation of structural components of membranes, and perhaps increasing the stability.
- B. May stimulate prostaglandin synthesis by increasing the conversion of linoleic to arachidonic acid, and preventing the peroxidation of arachidonic acid.
- C. Inhibit platelet aggregation by increasing the synthesis of PGI<sub>2</sub>. (“Platelet aggregation” & vascular diseases?)
- D. May optimize the immune system:
  - 1) Involved in the protection of leukocytes and macrophages during phagocytosis.
  - 2) Stimulates antibody production.
  - 3) Some examples?
    - a) Effects of vitamin E and(or) Se injections on serum IgG in piglets - Figure on the left: (Hayek et al., 1989. J. Anim. Sci. 67:1299)
    - b) Vitamin E natural killer cell activity in rats - Figure on the right: (Moriguchi et al., 1990. J. Nutr. 120:1096) (Also, high dietary vitamin E ↑ phagocytic activity of alveolar macrophages!



### 4) High-vitamin E:

- a) ↑ activity of natural killer cells.
  - b) Augment responses of splenocytes to mitogens.
  - c) ↑ alveolar macrophage function via macrophage-activating factor produced by lymphocytes.
- ☞ Many investigations with other species demonstrated similar results, even though the magnitude & type of responses were different.

- E. May be involved in oxidation-reduction reactions - Possibly a cofactor in cytochrome reductase portion of NAD oxidase & succinate oxidase systems?
- F. May be involved in others such as:
  - 1) Normal phosphorylation reactions.
  - 2) Synthesis of ascorbic acid.
  - 3) Sulfur AA metabolism.

### 3. Effect of Vitamin E on Animal Performance?

☞ Example in pigs - Dietary vitamin E & sows/piglets<sup>a</sup> (Mahan, 1991. J. Anim. Sci. 69:2904.)

#### A. Data:

Item	Vitamin E, IU/kg				
	0	16	33	66	
No. of pigs/litter:					
Birth	9.85	10.87	11.20	10.04	NS
Weaning	6.73	7.00	7.88	8.14	NS
$\alpha$ -tocopherol, $\mu\text{g/ml}$ :					
Sow serum (d 28)	.40	.78	1.45	1.83	Ln
Colostrum	2.72	4.34	7.75	7.01	Ln
Milk (d 28)	.44	.77	1.29	1.67	Ln
Pig serum (birth)	.05	.10	.15	.16	Ln
Pig serum (d 28)	.65	1.15	1.33	2.36	Ln
Post-weaning pig weight gain (0-28 d), g/d	375	346	332	329	

<sup>a</sup>NS = not significant; Ln = linear.

#### B. Conclusions/implications:

- 1) < 16 IU/kg supplemental vitamin E is inadequate, which may result in a small litter size, sow agalactia, and also increase in pig mortality during the first week after birth.
- 2) Vitamin E can transverse placental tissue, but rate is low.
  - ☞ 0.142, 0.224, 0.241 & 0.305  $\mu\text{g/g}$  for 0, 16, 33 & 66 IU supplemental vitamin E/kg, respectively.
- 3) Mammary transfer, therefore, seems to be more effective means to provide  $\alpha$ -tocopherol to nursing pigs.
  - ☞ But, the importance of  $\uparrow$  vitamin E status of the fetus before birth (or even after birth) has not been evaluated.

4. **Vitamin E Requirements**

Animal	Requirement, IU/kg
Swine: (NRC, 1998)	
3-10 kg	16
10-120 kg	11
Sows/boars	44
Poultry: (NRC, 1994)	
Immature chickens	4.7-10
Laying hens	4-6
Broilers	10
Turkeys, growing	10-12
Turkeys, breeding	25
Horses (NRC, 1978)	233 µg/kg BW
Fish: (NRC, 1993)	
Channel cat fish, rainbow trout, pacific salmon & tilapia	50
Common carp	100

5. **Sources** (McDowell, 1989)

Feedstuff	α, ppm	β, ppm	γ, ppm	δ, ppm
Barley	4	3	.5	.1
Corn	6	-	38	Trace
Oats	7	2	3	-
Rye	8	4	6	-
Wheat	10	9	-	.8
Corn oil	112	50	602	18
Cottonseed oil	389	-	387	-
Palm oil	256	-	316	70
Safflower oil	387	-	174	240
Soybean oil	101	-	593	264
Wheat germ oil	1330	710	260	271

6. **Possible Reasons for ↑ needs for Vitamin E (& also Se) in Recent Years**

- “Naturally occurring” vitamin E-Se deficiencies in swine were not reported until the late 60's, but became widespread in the 70's!
- A. ↑ use of confinement facilities - no access to other sources of the vitamin.
- B. Low-Se content of Midwestern feeds - Midwest is a major supplier of grains to many states/countries.
- C. ↑ use of solvent-extracted protein supplements.
- D. Limit feeding programs for sows during the gestation phase.
- E. Loss of vitamin E & Se via processing/storage of grains (e.g., drying, high moisture grains, etc.)
- F. Selection/production of meatier-type pigs.
- ☞ Some of these factors may be applicable to other species as well!

**VITAMIN E AND HUMAN HEALTH**

- *References - Various VERIS (Vitamin E Research & Information Service, LaGrange, IL) publications.*

**1. Antioxidants & Aging/Cancer**

- A. "Free radicals" - Highly unstable substances produced via metabolism & also from exposure to certain environmental factors (dietary components, smog, radiation, etc.).
- B. One suggestion for aging - Free radicals damage body cells and cause pathological changes associated with aging, and this process is gradual & irreversible!?
- C. Cancer:
  - 1) "Cancer" - Probably the result of external factors combined with a hereditary disposition for cancer.
  - 2) Normally, worn-out/injured tissues are replaced and(or) repaired.
  - 3) Often, cells change to a precancerous stage, but body's immune system detects & destroys abnormal cells.
  - 4) Occasionally, certain cells undergo changes without detection by the immune system, which can lead to "uncontrolled" growth & spread.
  - 5) Vitamin & Se Recommendations?

<b>Vitamin &amp; Se Recommendations for Cancer Prevention</b>				
<i>Antioxidant</i>	<i>RDA</i>	<i>Recommendations</i>	<i>Toxic levels</i>	<i>Factors &amp; requirements</i>
Vitamin E	10-20 IU	200-800 IU	Negligible at < 1200 IU	High PUFA intake, smog, smoking
Vitamin A	5,000 IU	12,500 IU	Limited: 25,000 IU, chronic; > 300,000 IU, acute	Smoking
Vitamin C	60 mg	1,000 mg	Negligible at 1-2 g	Stress, oral contraceptives, smoking
Selenium	None	50-200 µg	Potentially toxic at > 200 µg	Aging, high PUFA intake, smog, heavy metal contamination

- D. Free radical related damages:
  - 1) Oxygen containing free radicals readily attack PUFA in cell membranes via peroxidation (a chain reaction).
  - 2) Unless free radicals are neutralized, they can cause considerable damage to the structure & functions of cell membranes.

**E. Vitamin E:**

- 1) Inhibits accumulation of damaging free radicals. (Vitamin A,  $\beta$ -carotene & vitamin C are also antioxidants.)
- 2) Enhances the body's immune response (defence against cancer).
- 3) Protects vitamin A & spares Se.
- 4) Inhibits conversion of nitrites (present in smoked, pickled & cured food) to nitrosamines (strong tumor promoters) in the stomach.

**2. Protection Against Air Pollution Damages**

- A. Nitrogen dioxide & ozone (most damaging!) can generate unstable free radicals.
- B. Vitamin E traps & neutralizes free radicals more effectively than others in the lung.

**3. Optimal Immune System**

- A. Immune response initiation is considered to take place at the cell membrane level.
- B. Has stabilizing & regulatory effects on cell membranes to maintain optimal cell function. (via effects on free radicals!)
- C. Vitamin E supplementation -  $\uparrow$  immune response to antigen, stimulates production of antibody-producing lymphocytes, and  $\uparrow$  antibody production.
- D. Modulates synthesis of prostaglandins - "PG" are important regulators of immune responses and other host defenses, i.e.,  $\uparrow$  PG is immunosuppressive, and vitamin E may prevent infection-induced  $\uparrow$  in PG.

☞ Optimal concentrations for the immune function in most animal studies range from 180 to 360 mg/kg, which are at least 3 to 6 times higher than those concentrations found in animal diets!

**4. Neurological Role**

- A. Detrimental effects of vitamin E deficiency on nervous & cardiac systems & skeletal muscle have been known for years.
- B. Identification of a chronic deficiency in progressive neurological syndromes in children & adults is much more recent. (Mechanisms are unknown, but probably associated with free radical damages to cell membrane of nerve & muscle tissues.)
- C. Beneficial effects of vitamin E supplementation on some neurological disorders - Tardive dyskinesia, Alzheimer-like dementia, Parkinson's disease, etc.

**5. Exercise**

- A. The body takes in & utilize oxygen at a higher rate during exercise.

- B. A higher rate of lipid peroxidation with higher degrees of exercise? Thus, vitamin E needed to prevent free radical-related tissue damage may increase during strenuous exercise!

## SELENIUM

### 1. Introduction

- A. The early interest in Se was in its role as a toxic element:

#### 1) Did You Know?

"The battle of "Little Big Horn" in 1876. The U.S cavalry commanded by General George A. Custer had a 3-day forced march before reaching the Little Big Horn in Montana. Forages in that region contain toxic levels of Se. The horses were hungry since they had little grazing time during the march, so they avidly consumed the toxic plants and became sick. Obviously, they were unfit for ensuing battle, and thus were a factor in defeat. Also, a relief expedition failed to reach the beleaguered troops of General Custer in time to provide needed support. The officer in command of that expedition wrote in his official report that a peculiar sickness affected his horses, and was responsible for the delay." (McDowell, 1989)

#### 2) Marco Polo's journal:

*" . . . a poisonous plant . . . which if eaten by horses has the effect of causing the hoofs . . . to drop off . . ."*

- 3) An Army surgeon in the Nebraska Territory described similar signs in horses in 1857, and he termed it "Alkali" disease!
- 4) Three types of toxicity:
- Blind staggers type (with as low as 2-5 ppm?) - Wandering aimlessly, stumbling, impaired vision & signs of respiratory failure.
  - Alkali disease type (with as low as 2-5 ppm?) - Lmeness, hoof malformation, loss of hair & impaired reproduction.
  - High levels (over 40 ppm) - Sudden death or severe distress (labored breathing, ataxia, abnormal posture, diarrhea, etc.).

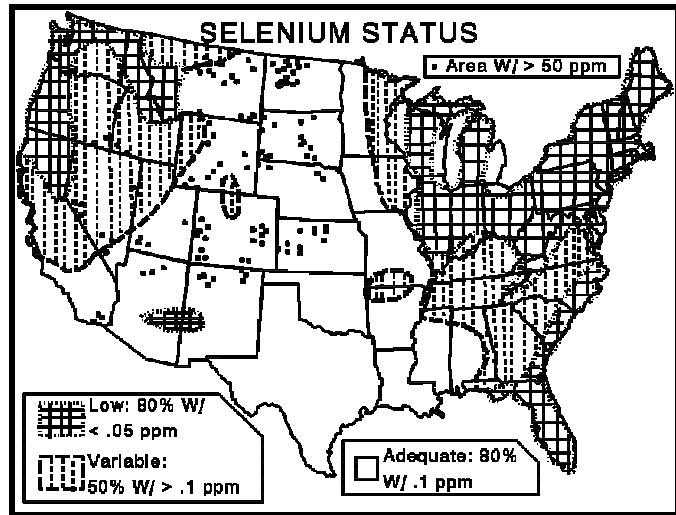
- B. Selenium as a nutrient:

- 1) Established as an essential nutrient in the late 1950's:
- Prevented liver necrosis in rats.
  - Prevented exudative diathesis in chicks.

- c) Prevented white muscle disease in ruminants, etc.
- 2) In the early 70s, Rotruck et al. (1973; Science 179:588) discovered that Se was an integral part of glutathione peroxidase.

**2. Selenium Deficient Areas**

- A. Selenium deficient areas of the US are much larger than those areas that are Se-toxic.
- B. Selenium in crops in relation to animal needs: (Adapted & redrawn from Maynard et al., 1979 & McDowell, 1992)



**3. Other Functions?**

- A. May play a role in electron transport - e.g., isolated a selenoprotein that resembles cytochrome C) in heart & muscle, which was absent in Se-deficient animals.
- B. A specific selenoprotein in spermatozoa may serve as a structural protein for mitochondria, or as an enzyme.
- C. May play a role in RNA because Se can be incorporated into purine or pyrimidine bases.
- D. May have a role in prostaglandin synthesis & EFA metabolism.
- E. May be involved in the immune response.

e.g., Effect of dietary Se on IgG concentration in yearling & older ponies (mg/100 ml): (Knight & Tyznik, 1990. J. Anim. Sci. 68:1311)

Week	0.02 ppm	0.22 ppm
0	1,170	1,250
1	1,420	1,475
2	1,280	1,740
3	1,700	2,365
4	1,380	2,090
5	1,310	1,915

\* Observed similar responses in whole blood Se & GSH-Px activity.

**4. Absorption/Excretion**

- A. Dietary concentrations/Se status of animals and the “form” affect the rate of absorption:
  - 1) Greater absorption in a deficient state.

- 2) Organic compounds, selenide (- 2) & elemental Se (0) are absorbed less efficiently.
- 3) Selenite (+ 4), selenate (+ 6) & selenomethionine are highly available sources.

B. Absorption rate:

1) Swine:

- a) No absorption at the stomach and first part of the SI.
- b) The greatest absorption at the last part of the SI, cecum & colon.

☞ About 77% of oral Se was retained in one study!

- 2) In rats, soluble Se compounds are efficiently absorbed from the GI tract (e.g., 92 & 91% for selenite & selenomethionine, respectively).
- 3) In humans, apparent absorption of dietary Se ranges from 55 to 70%.
- 4) Ruminants - Less efficient vs nonruminants (e.g., ≈ only 30% in sheep) because Se may be reduced to insoluble compounds in the rumen .

C. There seems to be no homeostatic control of Se absorption.

D. Excretion:

- 1) Via urine, feces & exhalation (1° route in the Se toxicity).
- 2) Urinary excretion - 1° route in nonruminants & humans (excretion rate is closely related to dietary intake).
- 3) Fecal excretions - Contain unabsorbed dietary Se, small amounts of Se excreted via bile, pancreatic and intestinal secretions.
- 4) In general, ruminants excrete Se in the feces possibly because rumen microbes reduce Se to unavailable form, ∴ ↑ excretion in the feces.

## 5. Deficiency

☞ Signs often observed in young pigs, and perhaps similar symptoms in poultry?

A. A sudden death - A prominent feature of the deficiency).

B. Based on necropsy:

- 1) Massive hepatic necrosis.
- 2) Edema in lungs, stomach submucosa, etc.
- 3) Paleness & dystrophy of the skeletal muscle (white muscle).
- 4) Mottling and dystrophy of the myocardium (mulberry heart).

C. Impaired immune response.

D. Impaired reproductive performance & milk production.

6. **Assessing Se Status** (Ullrey, 1987. J. Anim. Sci. 65:1712)

A. Analysis of plasma or serum Se:

- 1) Plasma or serum Se ↑ directly with ↑ dietary inorganic Se from deficient to adequate (0.1 to 0.3 ppm).
- 2) From > 0.3 to 0.5 ppm, plasma or serum Se ↑ until reaching a dietary level that ↓ feed intake.

B. Analysis of whole blood Se:

- 1) Whole blood Se levels are ≈ 10 to 50% higher vs plasma or serum because of higher Se contents in erythrocytes.
- 2) Whole blood Se levels tend to follow a pattern in plasma or serum.
- 3) 1° difference? - A tendency for a lag period in the Se response in whole blood vs serum or plasma possibly because of a relatively long half-life of erythrocytes.

☞ Most of Se in red cells is incorporated during erythropoiesis.

C. Assay of plasma or serum GSH-Px activity:

- 1) Relatively low proportion of Se is associated with GSH-Px in rats.
- 2) Also, a very low GSH-Px activity in plasma of sheep (∴ usually not recommended to use as a response criterion!).
- 3) The GSH-Px activity provides conclusions similar to plasma Se in a deficient to adequate region in rats.
- 4) But above adequate levels, there are poor correlations with dietary or plasma Se in rats, swine & cattle.

D. Assay of whole blood GSH-Px activity:

- 1) The GSH-Px activity in erythrocytes is higher than plasma in all species examined, ∴ consistently measurable.
- 2) The GSH-Px activity has a high correlation with plasma Se in low-Se animals, but correlations may be poor in adequate to high dietary Se.

E. Measurement of urinary Se excretion - Urinary Se as a proportion of intake ↑ remarkably when dietary levels exceed an apparent requirement.

F. Analysis of Se in skeletal muscle:

- 1) Dietary & skeletal muscle Se levels are directly related in animals consuming diets that are low to adequate.
- 2) Samples obtained by biopsy, at necropsy or at slaughter can be used to assess Se status in cattle, sheep or swine.

G. Se contents in animals (wet basis):

- 1) Skeletal muscle (i animals apparently fed adequate diets) - Swine, 0.05 to 0.10 ppm; cattle, 0.04 to 0.14 ppm (0.50 ppm in one report); sheep, 0.06 to 0.24 ppm (0.85 & 1.56 ppm reported in two reports).
- 2) Poultry - Chicks & poult fed deficient diets =  $\approx$  0.05 ppm (Scott & Thompson, 1971: Poult. Sci. 50:1742) & whole egg =  $\approx$  0.3 ppm (Latshaw, 1975: J. Nutr. 105:32).
- 3) Plasma - 0.80 to 0.91 ppm in swine & 0.42 ppm in rats.

7. Requirements

A. Must consider:

- 1) Variations in the Se content of feedstuffs (i.e., geographic areas).
- 2) Antioxidant levels in the diet (including vitamin E).
- 3) In swine & poultry - Se status of dam influences the requirement for nursing/weaning pigs & chicks. (Se is readily transmissible through placental & mammary barriers, and also from hens to eggs!)
4. The amount of supplemental Se permissible is regulated in the US [maximum of .3 ppm (FDA, 1987)] and also in Canada.

B Se requirements - Plasma glutathione peroxidase level is a reliable index of the Se status of pigs (also for poultry?):

Animal	mg/kg or ppm
Swine (NRC, 1998):	
3-10 kg	0.30
10-20 kg	0.25
20-120 kg	0.15
Sows/boars	0.15
Poultry (NRC, 1994):	
Immature chickens	0.10-0.15
Laying hens	0.05-0.08
Broilers	0.15
Turkeys	0.20
Horses, all classes (NRC,1989)	0.10
Fish (NRC, 1993):	
Channel catfish	0.25
Rainbow trout	0.30
Pacific salmon	Required, but not determined
Common carp & tilapia	Not tested

## 8. Amelioration of Se Toxicity?

- A. e.g., Effects of arsenicals & cysteine on chicks fed diets supplemented with a toxic level of inorganic Se: (Lowry & Baker, 1989. J. Anim. Sci. 67:959)

	Gain, g/d	Gain:feed, g/kg	Liver Se, µg/g DM
Basal	269	662	0.8
Basal + 15 mg/kg Se	109	458	14.9
Basal + Se + 14 mg/kg As	256	657	17.2
Basal + Se + 0.4% L-Cys	149	520	18.5
Basal + Se + As + Cys	254	671	17.4

- B. L-Cys (& its derivatives, which are commonly used to treat heavy metal toxicity) showed ameliorative activity.
- C. “As” compound totally corrected performance depressions, but it did not lower liver Se.
- ☞ Not in this research, but has been demonstrated in the earlier research that “As” increased biliary excretion of Se into the intestine.

## CHOLINE

### 1. Functions

- A. In lipid metabolism:

- 1) A component of phospholipids (important in building & maintaining the cell structure):
  - a) Phosphatidylcholine (lecithin) - A part of cell membrane, and also lipid transport moieties.
  - b) Sphingomyelin - Found in brain & nerve tissues.
- 2) Involved in phosphorylation and mobilization of long-chained FA from the liver, and in oxidation of FA in the liver.
 

☞ Hastening a removal of lipids or ↓ deposition of lipids in the liver, ∴ referred to as a “lipotropic” factor!

- B. Other functions:

- 1) Involved in formation of acetylcholine, which is released at the termination of parasympathetic nerves.
- 2) Serving as a source of labile methyl groups, which are important in: (e.g.)

- a) Formation of methionine from homocystine.
- b) Formation of creatine from guanidoacetic acid.

**2. Is Choline Vitamin?**

- A. Tentatively classified as a B-complex vitamin.
- B. But, does not satisfy a strict definition of the vitamin, which is:

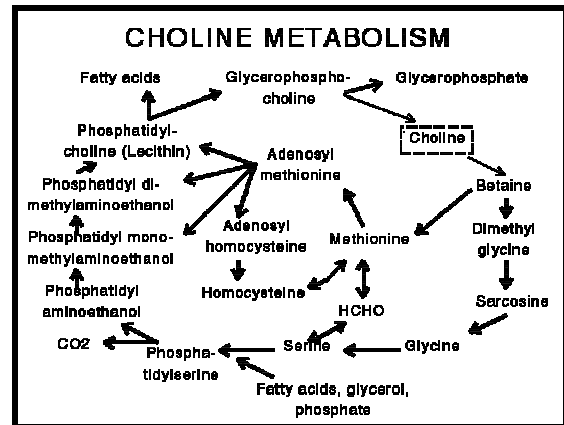
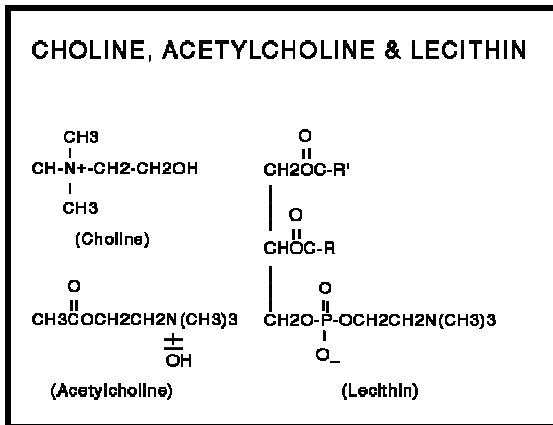
*“An organic substance of nutritional nature present in low concentration as a component of enzymes. It catalyzes reactions and may be derived externally to the tissue or intrinsic biosynthesis.”*

C. Choline:

- 1) Required by animals in greater amounts vs others.
- 2) Functions as a structural constituent rather than as a coenzyme.

**3. Structure and Metabolic Pathway**

- A. Free choline, acetylcholine & lecithin - Figure on the left.
- B. Metabolic pathway - Figure on the right: (Adapted & redrawn from McDowell, 1989)



**4 Deficiency**

A. Signs in poultry:

- 1) Poor growth.
- 2) Perosis (slipped tendon):
  - a) Hemorrhages & puffiness of a hock joint.

- b) Flattening of a joint.
- c) Achilles tendon slips from its condyles.
- d) Immobility.
- e) Effect of choline on perosis in chicks? (Ketola & Nesheim, 1974. J. Nutr. 104:1484)

Choline, mg/kg	Incidence, %
0	54
300	0
600	0

- ▷ Perhaps, need phospholipids for a normal maturation of cartilage matrix of bones, thus choline has some beneficial effects?
- ▷ In mature birds, their synthetic rate might be sufficient to meet the requirement, but may have to supplement for a maximum egg production.

#### B. Signs in swine:

- 1) Poor growth & unthriftiness.
- 2) Poor conformation - Short-legged & pot bellied.
- 3) Lack of coordination - e.g., "Spraddled legs" in newborn pigs:
  - a) Can be prevented by supplementation of choline to the sow diet.
  - b) But, also genetics, folacin and(or) B<sub>12</sub> may be involved!
- 4) Fatty infiltration of liver.

### 5. Requirements

#### A. In general:

- 1) Requirements can be met by: a) dietary supplemental choline or from typical feedstuffs, and b) choline synthesis in the body.
- 2) Affected by:
  - a) Dietary methionine (an other principal methyl donor) level.
  - b) Folacin level - Folic acid is involved in formation of a labile methyl group from a formate C.
  - c) Vitamin B<sub>12</sub> level - B<sub>12</sub> is involved in transfer of a methyl group to tetrahydrofolate.
  - d) Others - Dietary protein, lipids & carbohydrate, sex, growth rate, etc.

#### B. Choline supplementation in swine diets:

- 1) Research with starter, grower and finisher pigs showed no beneficial effect of supplementation.
- 2) For sow diets - May or may not have beneficial effects:

a) Effects of supplemental choline (188 litters): Stockland & Blaylock, 1974. J. Anim. Sci. 39:1113:

Added choline (ppm):	0	400	800
Farrowing rate, %	62	80	76
No. born	9.3	10.4	9.8
Live pigs born	8.0	9.2	9.8
No. weaned	6.6	7.6	6.9
Piglets w/spraddled legs	17	7	10

b) Supplemental choline on sows: (Maxwell et al., 1987. J. Anim. Sci. 64:1044)

Item	No suppl	882 or 551 ppm
Litter size:		
At birth	10.65	10.69
At d 21	7.76	8.22
Litter weight (kg):		
At birth	13.17	13.33
At d 21	36.53	39.68
Spraddle legs:		
Litter w/one or more	8	10
No of pigs	11	12

\* Total = 1,350 & 1,800 ppm for 551- & 882-ppm groups, respectively.

- (1) Some improvements in sow reproductive performance with choline supplementation.
  - (2) No effect of choline on the incidence of spraddle legs.
- 3) No signs of toxicity reported in swine, but may reduce weight gain of pigs with 2,000 mg/kg!

e.g., Excess supplemental choline (ppm) on weight gain of pigs (g/d): (Southern et al., 1986. J. Anim. Sci. 62:992)

Item	0	500	1,000	2,000	4,000	6,000
Weanling pigs:						
Exp. 1 & 2	721	696	720	684	-	-
Exp. 3	649	-	-	636	664	601
Weanling-finisher pigs:						
Exp. 4	757	-	-	701	-	-
Exp. 5	763	-	-	734	-	-
Grower-finisher pigs:						
Exp. 6	882	-	-	845	-	-
Exp. 7	777	-	-	802	-	-

## C. Choline requirements:

Animal	mg/kg
Poultry (NRC, 1994):	
Immature chickens	470-1,300
Laying hens	875-1,310
Broilers	750-1,300
Turkeys, growing	800-1,600
Turkeys, breeding	1,000
Swine (NRC, 1998):	
3-5 kg	600
5-10 kg	500
10-20 kg	400
20-120 kg	300
Gestating sow & boar	1,250
Lactating	1,300
Horses (NRC 1978)	Microbial synthesis
Fish (NRC, 1993):	
Channel catfish	400
Rainbow trout	1,000
Pacific salmon	800
Common carp	500
Tilapia	Not tested

## 6. Sources: (McDowell, 1989)

Source	ppm (dry basis)
Alfalfa, dehy	1,370
Barley	1,177
Corn, yellow	567
Cottonseed meal	2,965
Fish meal, anchovy	4,036
Oats	1,116
Milo	737
Soybean meal	2,916
Wheat	1,053

A. Not enough information on the availability of choline in natural feedstuffs, but based on a chick assay, soybean meal & whole soybeans may contain 60 to 75% available choline.

B. Supplemental choline:

- 1) Choline chloride contains 86.8% choline (a 70% liquid or 25-60% dry powder).
- 2) Choline bitartrate contains 48% choline.

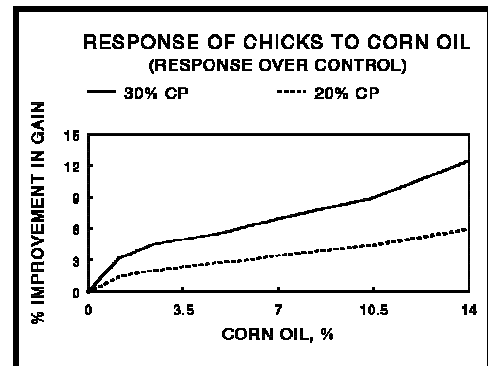
## SUPPLEMENTAL DIETARY LIPIDS

### 1. Dietary Lipids in General

#### A. High-energy diets for poultry:

- 1) About 50-60 years ago, researchers (Univ. of Connecticut) observed that feed efficiency was improved by ↑ energy content of broiler diets.
- 2) Intensive research at many universities followed, and found that:
  - a) There is a limit to the level of dietary energy that can be ↑.
  - b) Beyond a certain level:
    - (1) Produced poor growth & feathering, and also reduced feed efficiency.
    - (2) Believed by many that high energy ingredients such as fats contained "toxic" factors.
- 3) Around mid 50's, researchers discovered an importance of "quality & quantity" of dietary protein:
  - a) "Toxic effects" of high energy were overcome by simultaneous increase in dietary protein content (Donaldson et al., 1955, 1956, 1958. Poul. Sci. 34:1190, 35:1100, and 37:614.)
  - b) e.g., See Rand et al. (1958). Response of chicks to corn oil at two levels of protein. Poul. Sci. 37:1075.

☞ At the lower protein concentration, performance ↓ as dietary lipid ↑, but not at the higher protein concentration.



- 4) Subsequently, they found that growth rate, carcass fat, feed efficiency & feathering were influenced by manipulations of protein and energy ratios.

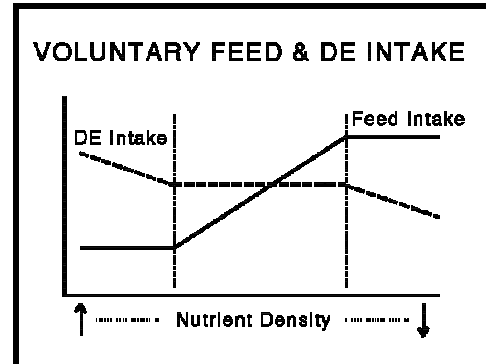
☞ Led to the concept of "**Calorie to Protein Ratio!**"

#### B. Energy density & energy intake:

- 1) Feed intake is largely determined by dietary energy content, i.e., animals generally adjust their voluntary feed intake to achieve a constant energy intake!

- 2) Voluntary feed and energy intakes with varying nutrient (energy) density: [Cole et al., 1972. In: Cole (Ed.) Pig Production]

☞ The idea that “Animals eat to satisfy their energy needs?” The concept may work within a certain range of energy densities . . . But, outside of that range - ???

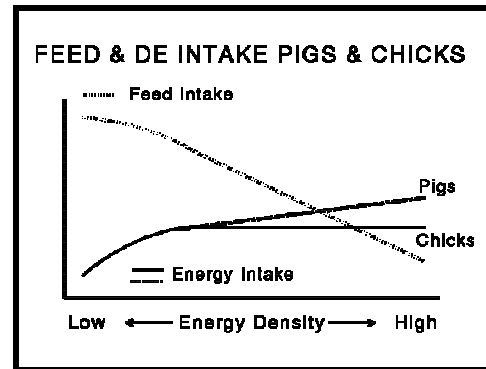


- 3) Feed and energy intakes in the pig and chick:

- a) Chicks have a well controlled system for energy intake.
- b) But energy intake tends to increase with dietary addition of lipids in pigs:

(1) This pig's propensity to over-consume energy may lead to its characteristic, “obesity?!” (The same is true for humans!?)

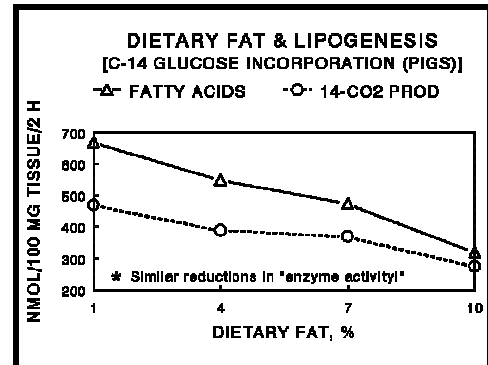
(2) Also, this contributes to an unclear energy-protein relationship in swine.



### C. Dietary lipids for swine:

- 1) Addition of lipids to swine diets:
  - a) Extensively investigated over the years (started in the early 1950's).
  - b) Generally, improved feed efficiency, but inconsistent responses in weight gain and carcass fat.
  - c) The necessity of adjusting dietary protein in accordance with supplemental fat is still a matter of debate!
- 2) Possible reasons for conflicting results (. . . regarding the necessity of adjusting protein concentrations):
  - a) Differences in the age of pigs.
  - b) Differences in the type & levels of lipids used.
  - c) “Protein sparing effect” of dietary lipids:
    - (1) Lipids are highly available energy source, and have beneficial effects on protein metabolism.

- (2) Thus, less deamination of protein as a source of energy, i.e., lipids (& CH<sub>2</sub>O) may provide enough substrates for the TCA cycle.
- (3) Greater release of insulin (anabolic), which has beneficial effects on protein metabolism, with dietary lipids?
- d) Improve protein digestibility with dietary lipids.
- e) “Quantity and quality of protein used.
- f) Depressions in the rate of lipogenesis:  
(Allee et al., 1971. J. Nutr. 101:1415)



- (1) Also, decrease the activity of malic enzyme, citrate cleavage enzyme, etc.
- (2) A possible reason? - Free-FA or their CoA derivatives may inhibit acetyl CoA carboxylase, which is a limiting factor in lipogenesis!

g) Alterations in the body composition.

D. Extra caloric effect:

- 1) The addition of fat improves the utilization of energy, and this increase in efficiency is referred to as “extra caloric” effect.
- 2) Quite often, greatly exceed its gross energy value:
  - o e.g., Estimated ME value of fat: (Jensen et al., 1970. Poul. Sci. 49:1697)

Age, wk	ME, Mcal/kg
8-12	9.92
12-16	8.96
16-20	10.11
20-24	11.69

\* vs GE = 9.4 to 9.5 Mcal/kg & assumed ME value = 7.72 Mcal/kg.

- 3) May be explained partly by:
  - a) Synergism between saturated and unsaturated fatty acids.
  - b) Lower rate of passage of food, which may enhance nutrient digestion/ absorption.
  - c) ↓ energy expenditure for FA synthesis from CH<sub>2</sub>O, i.e., direct deposition of lipids.
  - d) ↑ vitamin absorption (fat-soluble vitamins), which would have positive effects on digestive/metabolic processes of other nutrients.

2. Supplemental Dietary Lipids? (e.g., Swine)

A. Baby pigs:

1) For nursing piglets, diets mainly consist of sows' milk:

- a) 18-20% solids & 6-8% fat (30-40% fat on DM basis).
- b) Milk fat droplets are very small, i.e., relatively high in short- and medium-chained FA!
- c) Highly digestible - Digestibility, 95-100%.

2) Weanling pigs:

- a) One of the major interests in swine research over the years.
- b) Early investigations indicated:

- (1) Early weaned pigs can not utilize lipids efficiently.
- (2) Investigations on a source of fats, emulsifying agents, form of diets (liquid vs dry), etc. had no effect on performance! - e.g., Frobish et al., 1969 & 1970. J. Anim. Sci. 29:320 & 30:197.

c) More recent data (based on weekly or daily performance) indicated that pigs are inefficient in utilization of lipids during the first 2 weeks or so after weaning:

- ▷ e.g., Effect of fat source and combinations on starter pig performance: (Li et al., 1990. J. Anim. Sci. 68:3694)

	None	Soy	Coconut	Soy+ Coconut	Grease	Grease + coconut
0-2 wk (10% fat)						
ADG, g	281	263	254	272	277	249
ADFI, g <sup>a</sup>	322	286	281	295	304	281
F:G	1.15	1.11	1.13	1.08	1.11	1.14
2-5 wk (5% fat)						
ADG, g <sup>a</sup>	450	490	481	522	490	499
ADFI, g	745	749	717	767	754	722
F:G <sup>a</sup>	1.68	1.52	1.50	1.47	1.55	1.46
0-5 wk						
ADG, g <sup>b,c</sup>	381	400	390	422	404	400
ADFI, g <sup>c,d</sup>	577	563	540	581	577	540
F:G <sup>a</sup>	1.52	1.41	1.39	1.37	1.43	1.37

<sup>a</sup>Control vs fat ( $P < .01$ ); <sup>b</sup>Control vs fat ( $P = .06$ ); <sup>c</sup>Soy + coconut vs coconut ( $P < .05$ ); <sup>d</sup>Soy + coconut vs grease ( $P < .05$ ).

- d) Effects of dried whey & corn oil on lipase activity: (Cera et al., 1990. J. Anim. Sci. 68:384)

Day	Whey, % Oil, %	0 0	0 6	25 0	25 6
Total lipase units					
7		1,440	1,445	1,787	1,658
14 <sup>a</sup>		2,575	2,509	3,048	4,082
21 <sup>b</sup>		7,542	5,577	6,149	5,294
28 <sup>b</sup>		8,720	11,685	7,991	10,482
Lipase units/g pancreas					
7		193	196	201	204
14		200	224	210	255
21 <sup>b</sup>		328	265	286	267
28		318	358	335	332

<sup>a</sup>Dried whey effect,  $P < .01$ ; <sup>b</sup>Oil effect,  $P < .05$ .

- e) Effects of a source of fats on apparent digestibility: (Cera et al., 1988; J. Anim. Sci. 66:1430)

Item <sup>a</sup>	Corn oil	Lard	Tallow
Apparent absorption, g/d			
Wk 1 <sup>b</sup>	16.0	13.1	12.0
Wk 2	26.6	25.6	27.6
Wk 3 <sup>c</sup>	47.0	43.8	50.7
Wk 4	63.0	60.7	61.6
Apparent digestibility, %			
Wk 1 <sup>d</sup>	79.0	68.1	64.8
Wk 2 <sup>d</sup>	80.5	71.8	72.4
Wk 3 <sup>d</sup>	88.8	83.6	81.8
Wk 4 <sup>d</sup>	88.8	84.9	82.5

<sup>a</sup>Linear response to week,  $P < .01$  (all sources); <sup>b</sup>Corn oil vs animal fats,  $P < .01$ ; <sup>c</sup>Lard vs tallow,  $P < .05$ ; <sup>d</sup>Corn oil vs animal fats,  $P < .05$ .

- f) Effects of medium- and short-chained fatty acids on apparent digestibility: (Cera et al., 1989. J. Anim. Sci. 67:2040)

Item <sup>a</sup>	Tallow	Corn oil	Coconut oil
Apparent absorption, g/d			
Wk 1	17.7	22.2	18.6
Wk 2	34.7	39.7	38.8
Wk 3	54.0	52.3	64.9
Wk 4	75.8	72.9	77.4
Avg	45.5	46.8	49.9
Apparent digestibility, %			
Wk 1	75.4	76.5	81.7
Wk 2 <sup>b</sup>	76.0	79.7	83.3
Wk 3 <sup>c,d</sup>	81.5	86.3	89.2
Wk 4 <sup>c</sup>	86.6	89.3	89.7
Avg <sup>e,e</sup>	81.8	84.8	87.3

<sup>a</sup>Linear response to week,  $P < .01$  (all sources); <sup>b</sup>Animal vs vegetable,  $P < .05$ ; <sup>c</sup>Animal vs vegetable,  $P < .01$ ; <sup>d</sup>Corn vs coconut,  $P < .01$ ; <sup>e</sup>Corn vs coconut,  $P < .01$ .

- ▷ Also, there were trends for improved weight gain and N utilization (↑ N retention & lower serum urea) with coconut oil.

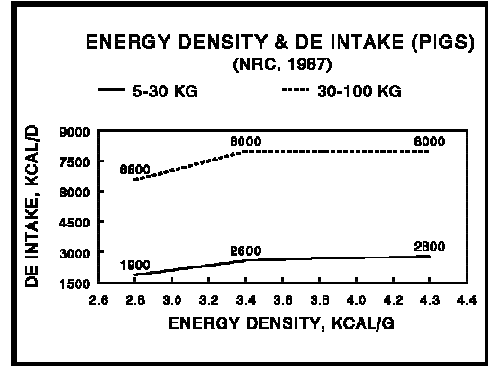
☞ The bottom line (baby pigs):

- a) First two wk or so after weaning, pigs can not utilize lipids efficiently possibly because of insufficient lipase concentrations or activity.
- b) The ability of weanling pigs to utilize lipids improves with age.
- c) Lipids containing higher proportions of short- and medium-chain FA (e.g., coconut oil) may be utilized better by young pigs.

B. Grower-finisher pig:

1) The relationship between energy density and voluntary feed intake is very important:

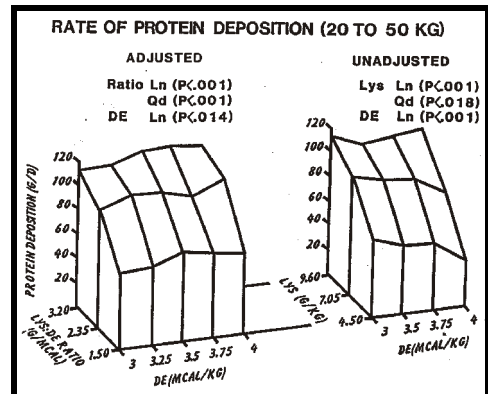
- a) Pigs generally consume feed to meet their energy requirements.
- b) With reduced energy density, animals increase feed intake.
- c) With increased energy density, animals reduce feed intake.
- d) Within a limit, energy intake remains relatively constant:



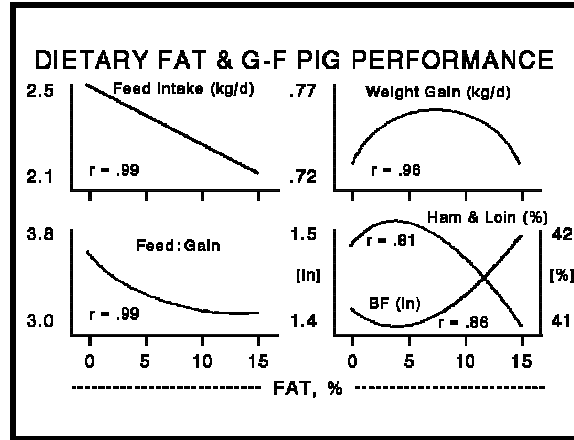
- (a) Energy density and DE intake: See NRC (1987).
- (b) Performance of pigs fed graded levels of sand: (Baker et al., 1968. J. Anim. Sci. 27:1332)

Item	% Sand:	0	20	40
Gain, kg/d		.85	.84	.73
Feed, kg/d		2.89	3.46	4.13
Feed (- sand), kg/d		2.89	2.77	2.48
G:F		.294	.243	.177
G:F (- sand)		.294	.303	.294

2) Because of this relationship, it is important to balance other nutrients. Otherwise, may see reduced performance: See Chiba et al. (1991a,b). J. Anim. Sci. 69:694 & 708.



- 3) Typical responses to supplemental fat in growing-finishing pigs - Summary [“Effect of dietary lipids. Adapted & redrawn from Moser, 1977. Feedstuffs 49(15):20].



- 4) Dietary lipids/thermal environment & relative performance: [Stahly, 1984. In: Wiseman (Ed.)]

Temp (°C)	10			22.5			35			
	Fat (%)	0	5	Δ	0	5	Δ	0	5	Δ
ME intake		114	112	-2	100	103	+3	72	77	+5
Gain		99	98	-1	100	109	+9	66	75	+9
ME:gain		116	116	0	100	94	-6	114	106	-8
backfat		93	97	+4	100	106	+6	85	92	+7

- ▶ Utilized better in warm/hot environments than in a cold environment!

- 5) Dietary lipids & apparent digestibility (%): [Stahly, 1984. In: Wiseman (Ed.)]

Fat (%)	Ileal			Fecal		
	4.5	17.0	26.8	4.5	17.0	26.8
CP	74	73	76	82	81	85
Amino acid:						
Lys	83	84	85	83	84	87
Trp	77	79	81	89	89	92
Thr	69	70	73	84	85	88
Met	83	82	84	83	85	86
<b>Avg</b>	<b>78</b>	<b>79</b>	<b>81</b>	<b>87</b>	<b>88</b>	<b>90</b>

- ▶ Dietary lipids generally improves digestibility of protein & amino acids!

☞ The bottom line (grower-finisher pigs):

- a) To a certain extent, the pig can adjust voluntary feed intake to achieve constant energy intake.

- b) Because of this, nutrient levels (especially, amino acids) have to be adjusted in concert with dietary energy densities.
- c) Dietary lipids - Improve feed efficiency, may or may not improve weight gain, and little adverse effects on carcass with  $\approx \leq 5\%$ .
- d) Lipids are utilized better in a warm or hot environment vs cold environment (associated with a low heat production rate in lipid metabolism).
- e) Dietary lipids may improve nutrient digestibility.

C. Gestating & lactating sows:

- 1) On the average, producers lose about 25-30% of piglets born before weaning (. . . even though there has been some improvement in recent years):

- a) Most of the losses occur during the first few days, and  $\approx \frac{1}{2}$  due to starvation & crushing!
- b) Smaller, weaker pigs: [Speer, 1970. Unpublished data (based on 1948 litters)]

Birth wt., lb	Survival, %
Under 2.0	42
2.0-2.4	68
2.5-2.9	75
3.0-3.4	82
3.5-3.9	86
4.0 and over	88
<b>Avg</b>	<b>77</b>

- c) Baby pigs:

- (1) Only  $\approx 2\%$  body fat (mostly structural),  $\therefore$  low energy reserves.
- (2) Liver glycogen depletes rapidly within 12-24 h.
- (3) Develop a hypoglycemia  $\rightarrow$   $\uparrow$  chance of being crushed.
- (4) Little insulation (hair & fat).

☞ Because of all these factors (lack of insulation & low energy reserves), pigs cannot maintain a proper body temperature.

- d) To increase survival rate of baby pigs:

- (1) Must increase body reserves of pigs and(or)
- (2) Improve the quality of their diet (i.e., milk).
- (. . . + other management practices, obviously!)

- 2) Dietary lipids for sows - Dietary fat can improve the baby pig survival rate!

- a) Summary of effects of dietary fat: [Based on 677-938 litters; Moser & Lewis, 1980. Feedstuffs 52(9):36]

Item	Contr.	Fat	Diff.
Born alive	10.0	9.9	-.1
No. weaned	8.1	8.4	.3
Survival, %	82.0	84.6	2.6

- b) The degree of response to dietary lipids depends on the status of herds & birth weight of pigs: (Pettigrew, 1981. J. Anim. Sci. 53:107)

- (1) Herds having < 80% survival rate - 4.1 % ↑.
- (2) Herds having > 80% survival rate - .6% ↑.
- (3) Pigs weighing < 1 kg at birth - 17.1% ↑.

- 3) Possible reasons for improved baby pig survivability:

- a) Increased fat content of milk - "Effect of dietary fat on milk fat (%)"<sup>a</sup>:

	Fat in diet:	-	+
Moser & Lewis, 1980		7.3	9.1
Coffey et al., 1987		5.14	6.07
Schoenherr et al, 1989		5.37	6.85
Newcomb et al., 1991 (DM basis)		20.6	23.6

<sup>a</sup>Moser & Lewis. Feedstuffs 52(9):36; Coffey et al. J. Anim. Sci. 65:1249; Schoenherr et al. J. Anim. Sci. 67:482; Newcomb et al. J. Anim. Sci. 69:230.

- b) Increased milk production - "Effect of dietary fat on milk yield (kg/d)"<sup>a</sup>:

Reference	Contr.	Fat
Kruse et al., 1977	4.60	5.33
Pettigrew, 1978	3.82	4.48
Boyd, 1979	8.72	9.44
Coffey et al., 1987	8.93	11.06

<sup>a</sup>Kruse et al. Acta Agric. Scand. 27:289; Pettigrew. Proc. Pacific NW Pork Exposition, WSU; Boyd. Ph.D. Dissertation. Univ. of Nebraska; Coffey et al. J. Anim. Sci. 65:1249.

- c) A slight increase in energy reserves of "newborn" piglets<sup>a</sup>:

	Contr.	Fat
Coffey et al. (1987):		
Blood FFA, mmol/l	3.23	4.12
Blood TG, mg/dl	31.1	33.9¶
Newcomb et al. (1991):		
Plasma FFA, µeq/l	120.0	136.4¶
Liver glycogen, % wet tissue	13.9	18.3¶

<sup>a</sup>Coffey et al. J. Anim. Sci. 65:1249; Newcomb et al. J. Anim. Sci. 69:230; ¶ = nonsignificant.

4) Effects of dietary fat at various phases<sup>a</sup>: [Moser, 1985. In: Cole & Haresign (Ed.)]

	Gestation	Lactation	Gest + Lact
Pigs weaned/sow	+7 (3)	+7 (2)	0 (17)
Survival (%)	+4.7 (4)	+1.2 (3)	+3.4 (22)

<sup>a</sup>( ): No. of experiments.

- a) To observe beneficial effects of fat, sows may have to consume 1 kg of fat before parturition. (Pettigrew, 1981. J. Anim. Sci. 53:107)
- b) Can be done by:
  - (1) Feeding a diet containing 10% fat for a week, or
  - (2) A diet containing 5% fat for 2 weeks before farrowing.

### FEED GRADE LIPIDS

#### 1. Terminology (See AFIA, 1986. Proc. AFIA 46th Annu. Meeting)

##### A. Total fatty acids (TFA):

- 1) Include both free FA and those combined with glycerol.
- 2) In general, fats contain  $\approx$  90% FA (9.4 Cal/g) & 10% glycerol (4.2 Cal/g).
- 3) A good index of the potential energy value of fat.

##### B. Free fatty acids (FFA):

- 1) FA not attached to glycerol.
- 2) High levels of FFA were once thought undesirable:
  - a) High because of extensive bacterial & enzymatic actions, i.e., a reflection of careless handling prior to rendering.
  - b) Also, may be due to  $\uparrow$  oxidation rate,  $\therefore$   $\uparrow$  rancidity?
- 3) But with the use of antioxidants, high levels pose no problem!

##### C. Moisture has adverse effects on fat - Corrosion of handling equipment/facilities $\rightarrow$ rust (a powerful promotor of rancidity).

##### D. Insoluble impurities:

- 1) Include minute particles of fiber, hair, hide, bones, minerals, etc. - insoluble in kerosene or petroleum ether.
- 2) Can cause problems in handling & storage (plus may  $\downarrow$  nutritional quality).

## E. Unsaponifiable matters:

- 1) Include fat soluble vitamins, pigments, sterols, fatty alcohols, etc., which are not split into glycerol and alkali salt of FA (soap) by alkaline hydrolysis (KOH).
- 2) All natural fats & oils contain small amounts.

## F. Iodine value:

- 1) Measure the degree of unsaturation (each double bond takes up 2 atoms of iodine).
- 2) Expressed as grams of I absorbed/100 g of oil or fat.

## G. Fat stability:

## 1) Oxidative rancidity:

- a) Can lower the quality of fat.
- b) Can destroy fat-soluble vitamins in feeds.

## 2) Measured by:

- a) "Peroxide value" - Measures mEq of peroxide/kg lipids, and considered "not" rancid if  $< 5$  meq/kg.
- b) AOM test:

- (1) A measure of peroxide value after 20 h of bubbling air through samples.
- (2) Determine the ability of fat to resist oxidative rancidity.

## H. Titer - An indication of the degree of hardness (or unsaturation):

- 1) Determined by melting FA after fat hydrolysis & cooling slowly, and measuring "congealing" temperature.
- 2) Titer:
  - a) Over 40°C - Classified as "Tallow."
  - b) Under 40°C - Classified as "Grease."

## I. Color:

- 1) Variations - From pure white (refined beef tallow) & yellow color (grease) to very dark color (acidulated soapstock).
- 2) Generally, differences in color have no effect on the nutritional value of fat, but may be an important consideration in "pet" foods.

2. **Use of Feed Fats by Various Species** (million lb; 1990 = estimates; Rouse, 1987. Feed Management. Feb.):

Animal	1986	1990	% †
Veal	100	125	25
Pet	400	450	13
<b>Hog</b>	<b>100</b>	<b>250</b>	<b>150</b>
Cattle	200	225	13
<b>Broiler</b>	<b>650</b>	<b>750</b>	<b>15</b>
<b>Turkey</b>	<b>500</b>	<b>700</b>	<b>40</b>
<b>Layer</b>	<b>30</b>	<b>50</b>	<b>67</b>
Fish	30	50	67
Dairy	90	250	178
<b>Total</b>	<b>2,100</b>	<b>2,850</b>	<b>35</b>

3. **Production of Animal Fats** (Rouse Marketing Inc., 1983)

Source	Mil. lb.
Beef tallow	4,387
Restaurant grease	1,350
Pork grease	537
Dead stock	447
Poultry	253
Other	200
<b>Total</b>	<b>7,222</b>

4. **Feed Fat Categories**

A. "Animal fat:"

1) Mainly from packing house offal or supermarket trimmings.

2) "Tallow" - Titer = > 40°C & 4-20% FFA:

a) Fancy - Mainly for pet foods (maximum of 4% FFA).

b) No. 1 - Maximum of 15% FFA.

c) No. 2 - Maximum of 20% FFA.

3) "Grease" - Titer = < 40°C & 4-50% FFA:

a) Choice white - Maximum of 4% FFA.

b) Yellow - Maximum of 15% FFA.

c) Brown - Maximum of 50% FFA.

B. "Poultry fat" - From poultry offal, and mostly used by the poultry industry as a feed ingredient.

C. "Blended feed grade animal fats" - A blend of tallow, grease, poultry and restaurant grease.

D. "Blended animal and vegetable fats" - A blend of animal fats + vegetable fats.

- E. “Feed grade vegetable fat” - Includes vegetable oils, acidulated vegetable soapstock and other refinery by-products.
5. **Rancidity** - lipids are subject to two types of rancidity:
- A. Oxidative rancidity:
- 1) Light, heat & other factors can lead to formation of free radicals in unsaturated fats.
  - 2) Free radicals react with oxygen to form peroxides.
  - 3) Peroxides react with another unsaturated fat molecule.
- ... **Chain reaction!**
- ☞ Products? - Ketones, aldehydes, organic acids, etc. - have unpleasant odor/off-flavors!
- B. Hydrolytic rancidity:
- 1) At high temperatures (with a presence of water), FA are hydrolyzed from TG (certain minerals can catalyze reactions), which lowers pH of fat.
  - 2) Reduces the ME value of lipids.
- ☞ “High FFA” concentrations in a “low” grade tallow or grease are clear indication that they have undergone hydrolytic rancidity.
- C. Antioxidants - e.g., Ethoxyquin (Santoquin), Butylated hydroxyanisole (BHA), Butylated hydroxytoluene (BHT), . . . , etc.