

*Animal Welfare in Practice: Mink Farming*  
*OCTOBER 3-4, 2014*



*Mink Health and  
Management-Related Diseases*

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# Nutritional Management of Mink Health and Welfare

- Mink is a **strict carnivore**

- ▶ Makes blood sugar from dietary amino acids
- ▶ Needs a high protein, high energy (fat) diet
- ▶ Needs a diet based on animal products



- Mink is a **seasonal breeder**

- ▶ Body fat deposition in preparation for winter
- ▶ Slimming down, nutritional flushing prior breeding
- ▶ Body condition is important for the health and welfare of mink



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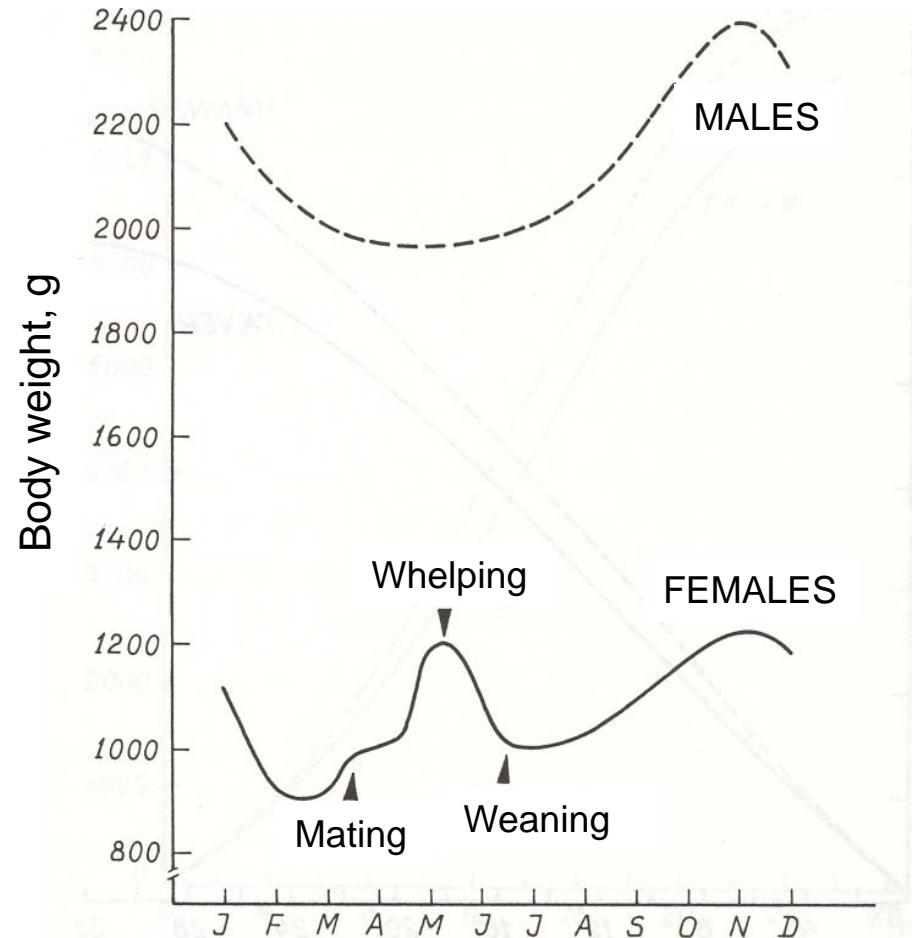
# Mink Is a Seasonal Breeder

- Body weight and body condition
  - ▶ Responsive to changes in energy supply
  - ▶ Seasonal changes in hormonal status
- Wintertime food scarcity
- Reduced physical activity
- Successful reproduction depends on nutritional status and body condition



# Body Weight Shows Large Seasonal Fluctuations

- Sexual dimorphism
- Body fat deposition and mobilization
  - ▶ Autumnal weight gain
  - ▶ Loss prior to breeding
  - ▶ Loss during lactation



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# Practical Problems on Mink Farms

- Large pelts are paid premium price
  - ▶ Producers want to raise big mink
  - ▶ Genetic selection for large body size
- Heavy feeding results in obese animals
  - ▶ Need drastic slimming prior to breeding
  - ▶ Poor health
  - ▶ Poor reproduction



# Practical Problems on Mink Farms

- The production of large pelts contradicts with the goals of successful reproduction and (metabolic) health
  - ▶ Nursing sickness
  - ▶ Fatty liver disease
  - ▶ Bladder infections and stones



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# Nursing Sickness in Mink

- Nursing disease, nursing anaemia, June blues
- Leading cause of death in adult females
- Common cause for culling in high producing breeder ♀♀ and their kits
- Represent a large economic and genetic loss to industry



# Clinical Symptoms of Nursing Sickness

- Loss of appetite, emaciation; >30% BW loss
- Lethargy, staggering gait
- High blood glucose, protein, creatinine
- Severe dehydration
  - ▶ Extremely low plasma and urine sodium concentrations
- Rapid deterioration: Coma, death within 5 d
- Fatty liver common finding post mortem





# What Causes Nursing Sickness?

- Complex metabolic disorder
  - ▶ Develops when demands for lactation require extensive mobilization of body energy reserves
- Incidence may vary greatly between years
  - ▶ Morbidity up to 15%; Mortality up to 8%
- Old females with large litters most often affected
- Seen typically around 42 days after whelping
  - ▶ Stress has been shown to trigger the onset
  - ▶ Post-weaning cases also



# What Causes Nursing Sickness?

- **Linked to poor blood sugar regulation**
  - ▶ During pregnancy and lactation a degree of insulin resistance develops in the female
  - ▶ During these times the mink dam is particularly susceptible to problems in glycemic regulation
- Mink in non-ideal body condition have more problems with blood sugar regulation
  - ➔ Poor reproductive performance
  - ➔ Increased mortality of dams



Mink females in non-ideal body condition show higher gestational glucose levels and greater glycemic variation during breeding, gestation and lactation than dams in ideal condition

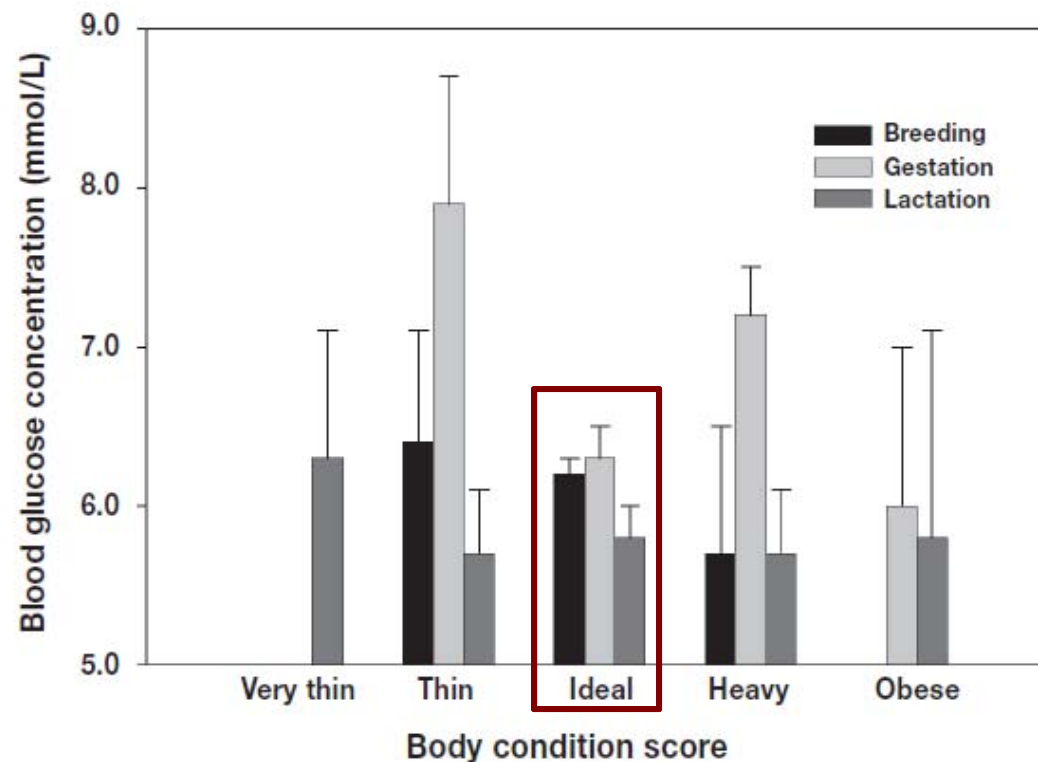


Figure 1. Mean blood glucose concentrations in mink females at breeding, gestation, and lactation according to body condition score: very thin ( $n = 0, 0, 3$ ), thin ( $n = 4, 6, 13$ ), ideal ( $n = 100, 78, 70$ ), heavy ( $n = 3, 15, 11$ ), and obese ( $n = 0, 3, 1$ ).

# Anti-inflammatory (HerO, ASA), insulin sensitizing (CrPic) and antioxidant (ASA) compounds restore glycemic regulation in mink dams with hyperglycemia during late lactation

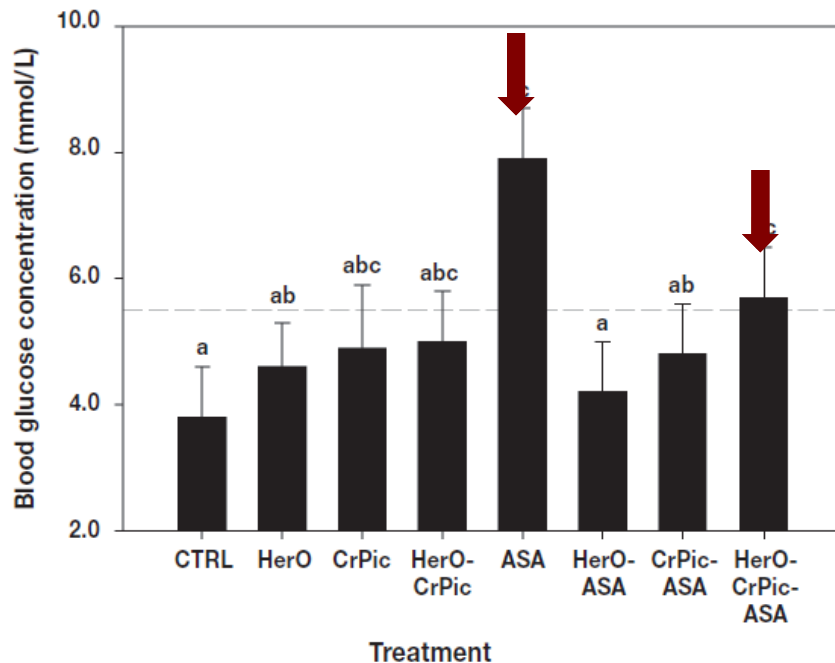


Figure 1. Mean (least-squares) blood glucose levels, with standard errors, averaged over days 28 to 42 of lactation, for normoglycemic mink females fed the basal, control diet (CTRL) or the basal diet plus various combinations of herring oil (HerO), chromium picolinate (CrPic), and acetylsalicylic acid (ASA) for 1 wk from day 21. Means with the same letter(s) are not significantly different, as determined with transformed data ( $P < 0.05$ ). Broken line indicates the cutoff point for normoglycemia (5.5 mmol/L).

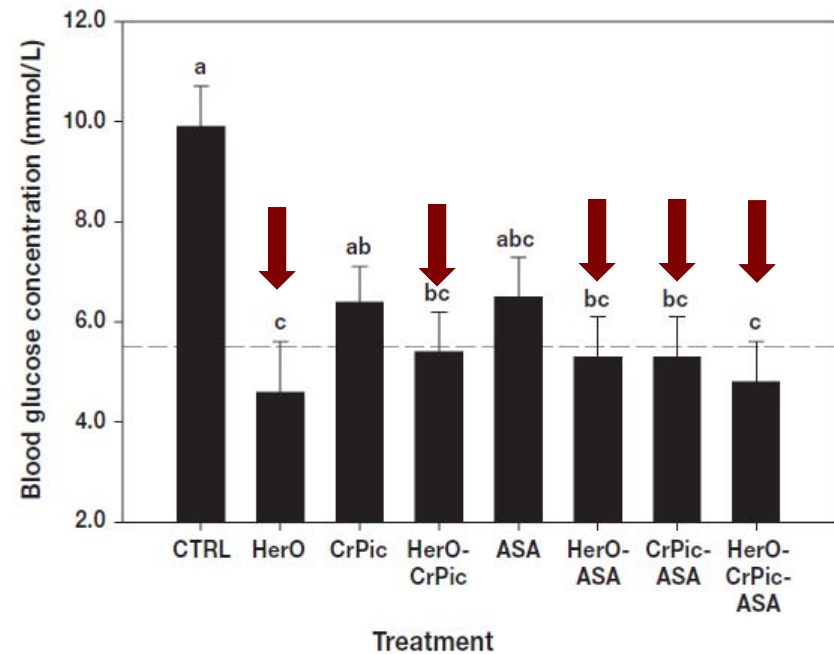


Figure 2. Mean blood glucose levels for hyperglycemic mink females receiving the same experimental dietary treatments. Details as for Figure 1.

# What Causes Nursing Sickness?

- The lactating mink dam may become deficient in n-3 PUFA
  - ▶ These fatty acids are not synthesized and are secreted in milk fat
  - ▶ Lost selectively during mobilization of body fat reserves
- Rapid BW loss may result in the development of fatty liver
  - ▶ Especially a concern for obese mink
  - ▶ Loss of n-3 PUFA in the liver
- Low n-3/n-6 PUFA associated with insulin resistance



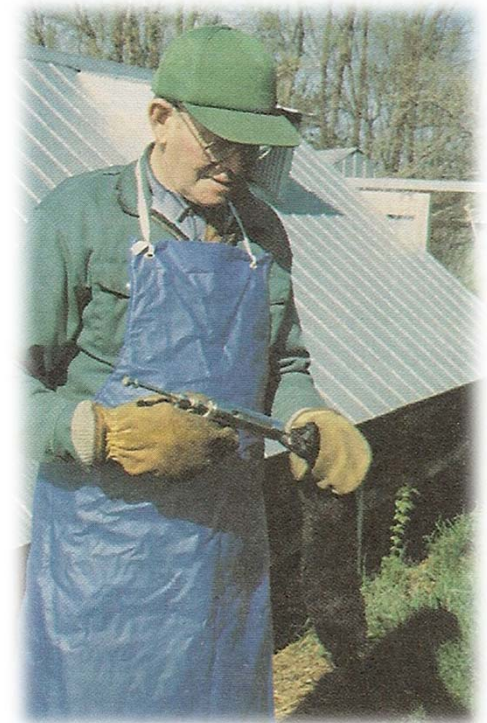
# Does Stress Make Insulin Resistance Worse?

- Stress hormones increase the mobilization of nutrients
  - ▶ Blood glucose, lipid levels ↑
- Epinephrine (adrenaline)
  - ▶ Induces insulin resistance in liver, muscle, and adipose tissue
- Glucocorticoids
  - ▶ Increase gluconeogenesis



# Can We Cure Nursing Sickness?

- There is no effective treatment
- When clinical symptoms detected early, some females respond well to electrolyte therapy (oral, subcutaneous)
- Dietary salt supplementation of 0.42-0.50 NaCl/100 kcal during lactation is beneficial in reducing incidence of nursing sickness



# Can We Cure Nursing Sickness?

- On-farm practices related to breeder selection, animal management, and feeding management which promote better glycemic control reduce the incidence
- Risk increased by large fluctuations in body condition
  - ▶ Excessive fattening during the fall
  - ▶ Rapid slimming prior to breeding
  - ▶ Rapid mobilization of body fat reserves for milk production
  - ▶ Stress during pregnancy and lactation
- Proper conditioning of breeder females throughout the production cycle



# Fatty Liver Disease

- Hepatic lipidosis, liver steatosis, hepatic fatty degeneration
  - ▶ Mortality an increasing problem in mink
  - ▶ Overweight and obese males during September-December
  - ▶ Breeders during slimming down period
- Often asymptomatic and there is no effective treatment
  - ▶ Increased liver weight, liver fat content, plasma ALT
  - ▶ Terminal phases reduced feed intake
  - ▶ Melaena and gastric bleeding



# Liver Lipid Metabolism

- The liver plays a central role in fat metabolism
- Hepatic lipidoses / steatosis = Fatty liver
  - ▶ Characterized by excessive fat (TAG) accumulation
  - ▶ Increased influx of fatty acids (NEFA)
  - ▶ Reduced secretion of fat (TAG) in VLDL
  - ▶ Reduced mitochondrial  $\beta$ -oxidation of fatty acids
  - ▶ Increased fat synthesis (*de novo* lipogenesis)



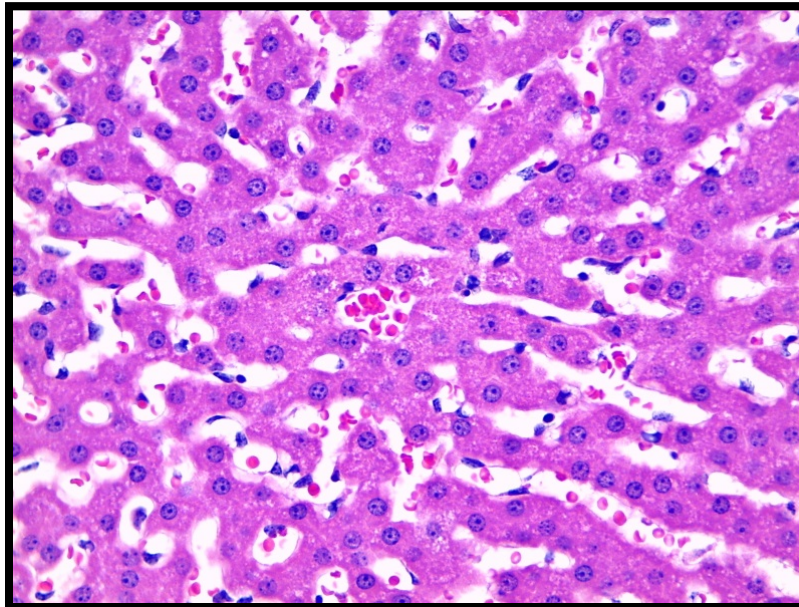
# How Does Fatty Liver Look Like?

- Normal liver fat content about 5%;  
Obese animals about 9%
  - ▶ Mink diagnosed with extreme hepatic fatty infiltration 40-55%
- Liver extremely enlarged
  - ▶ Bright yellow in colour
  - ▶ Fragile texture
  - ▶ May spontaneously rupture causing death

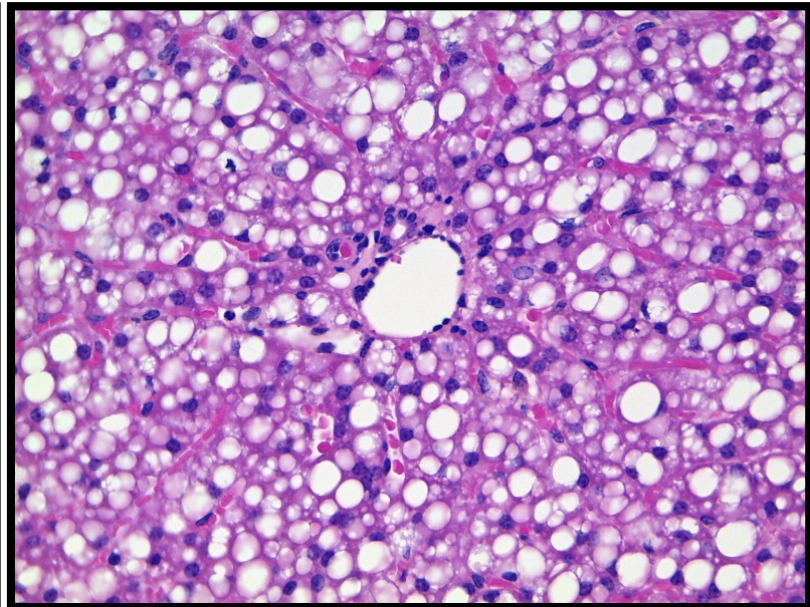


# How Does Fatty Liver Look Like?

Healthy Liver



Macrovesicular Steatosis

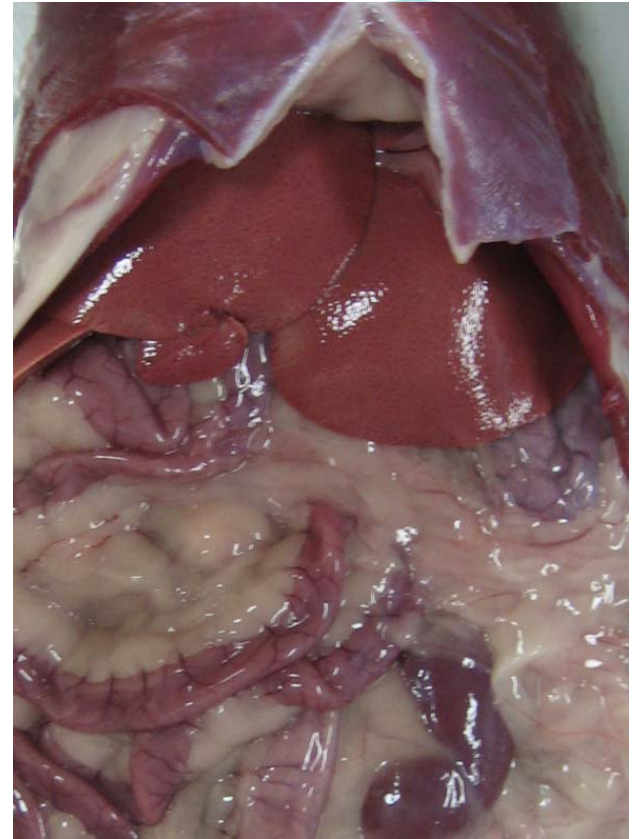


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# What Causes Fatty Liver in Carnivores?

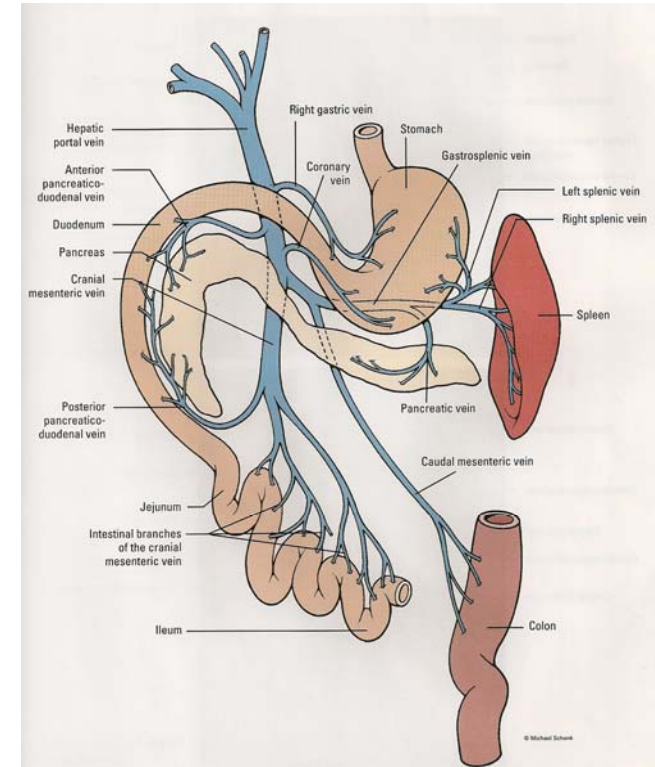
- Protein-calorie malnutrition
  - ▶ Low dietary protein; Amino acid imbalance
  - ▶ Excess dietary fat; Fatty acid imbalance
  - ▶ Excess dietary carbohydrate
- Severe negative energy balance
  - ▶ Loss of appetite, restricted feeding
- Nutritional deficiency
  - ▶ Choline, Vitamin B
- Poor feed quality



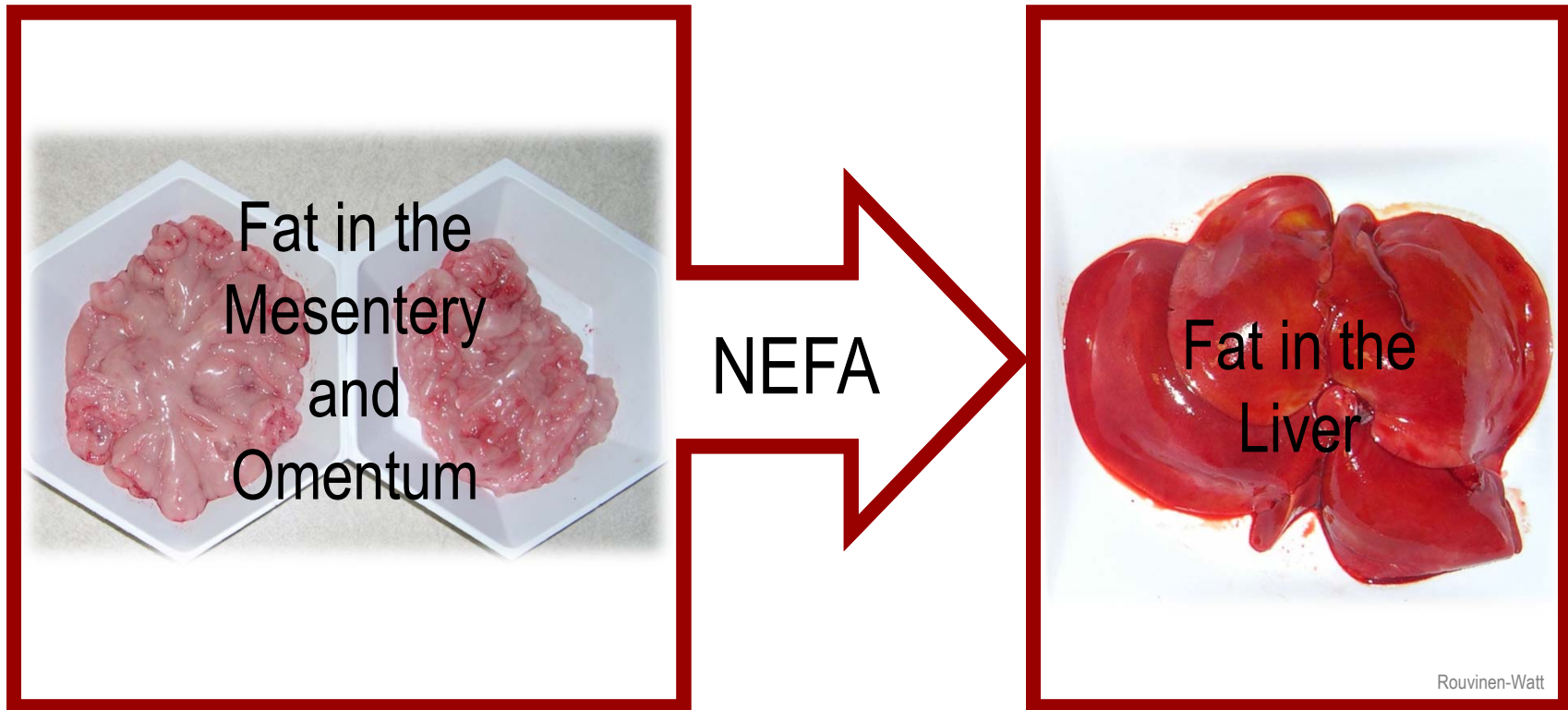


*Fatty liver in mink during food deprivation is caused by the increased hydrolysis of fat depots*

- Visceral fat is a problem
  - ▶ Drains into hepatic portal vein
  - ▶ Blood lipid levels ↑
  - ▶ Liver infiltrated by fat
- Similar situation in obesity
  - ▶ Fat storage in adipose ↓
  - ▶ Release of fatty acids ↑



When mink are not eating enough they mobilize body fat to meet energy demands



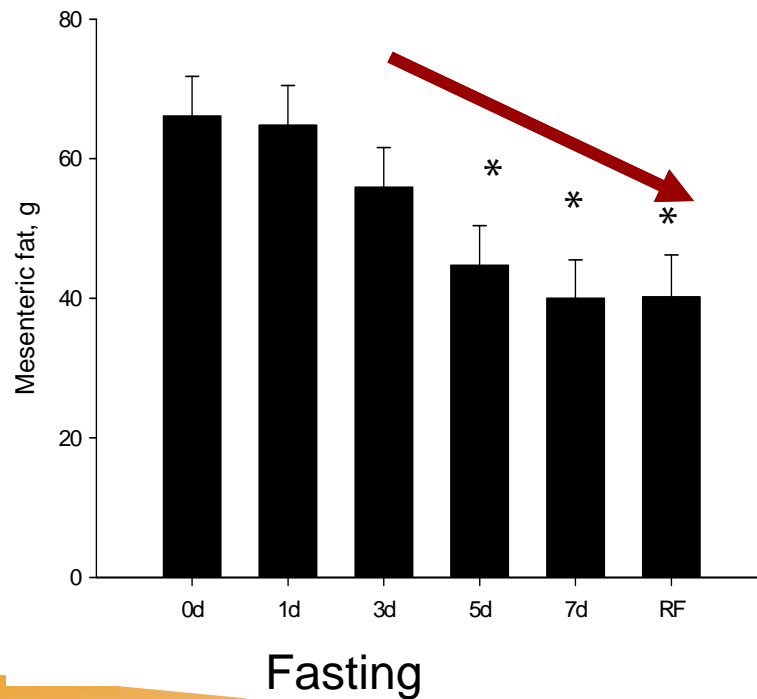
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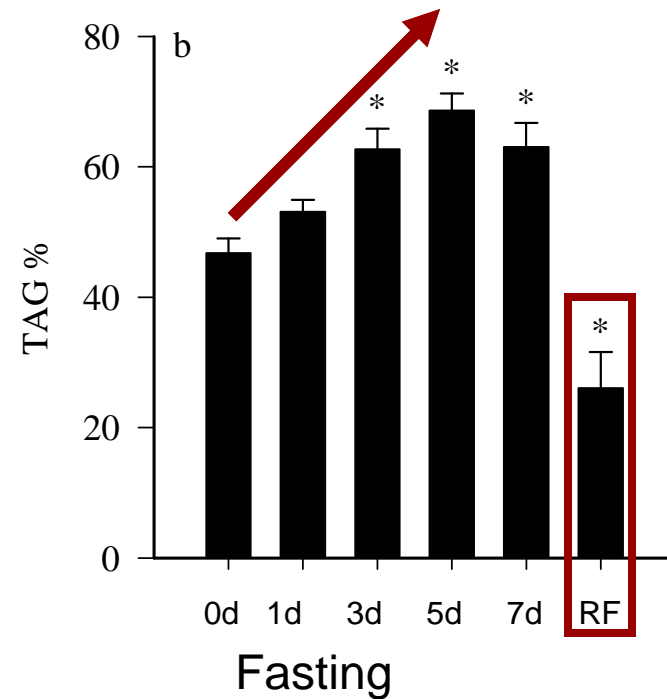


# Mesenteric Fat and Liver TAG

Mesenteric fat is mobilized



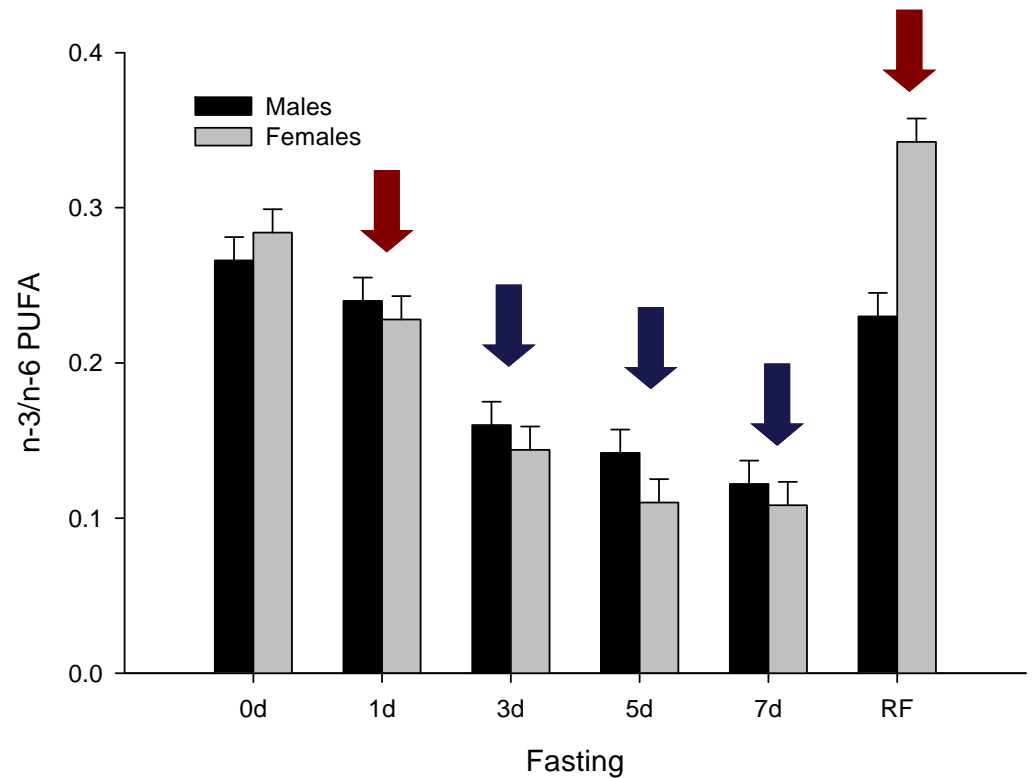
Liver fat is increased



# Liver Fat Accumulation and Loss of n-3 Fatty Acids

Body fat mobilization depletes n-3 PUFA

Loss/Recovery more rapid in females



0d:  
5.9±1.5%

1d:  
6.4±1.5%

3d:  
13.2±1.5%

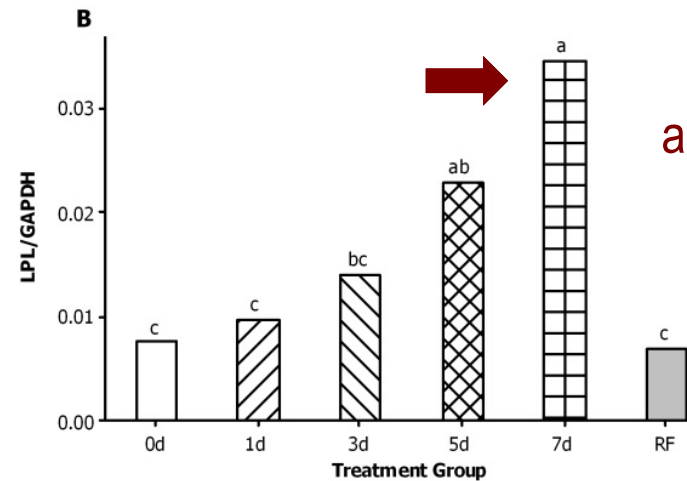
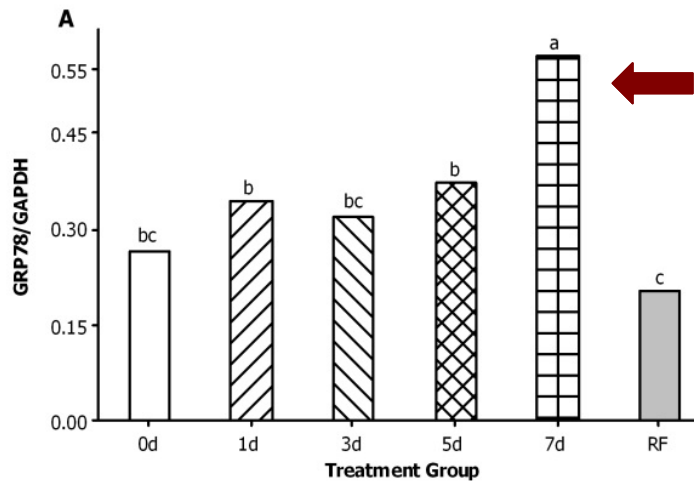
5d:  
19.2±1.5%

7d:  
19.7±1.4%

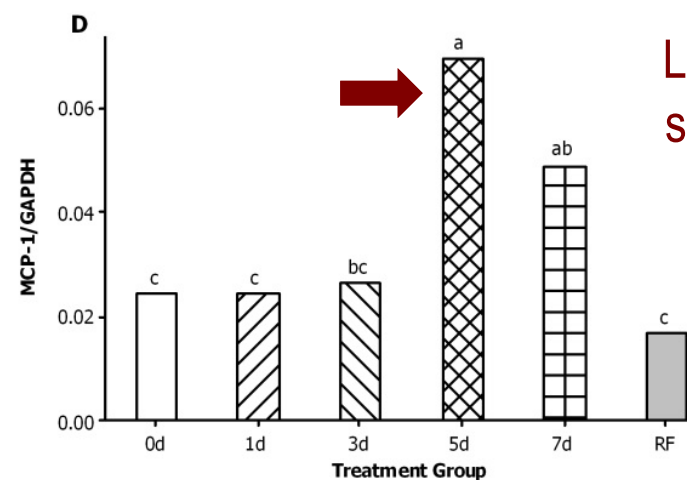
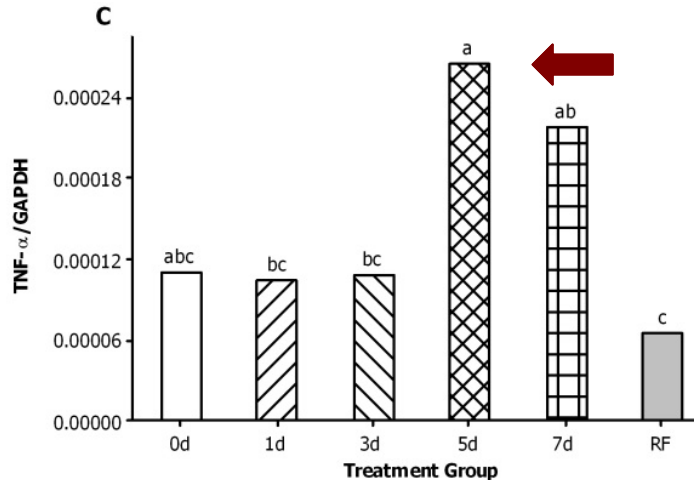
RF:  
5.3±1.6%



# Indicators of Liver Inflammation



Excessive fat accumulation induces liver inflammation



Liver inflammation is a sign of a more serious liver disease



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# Recommendations

- Prevent over-conditioning of mink during the fall (Sept-Dec)
- Maintain normal feed intake during stressful periods
- Increase or maintain intake of omega-3 PUFAs during periods of body weight loss



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# Urinary Tract Infections and Bladder Stones

- Urolithiasis, cystitis, plum bladder disease
- Bladder and kidney infections
- Stones (urinary calculi, uroliths) in bladder, kidneys, urethra
- Can be a serious problem in mink
  - ▶ Mortality 0.02-0.41%, 5–35 % of all dead kits
- Sudden death of apparently healthy animals
  - ▶ Blocked urethra
- Symptoms in earlier stages
  - ▶ Straining and vocalizing in discomfort, pain when urinating
  - ▶ Blood in the urine, oral ulcerations





# Urinary Tract Infections and Bladder Stones

- Bladder infections late June – July
  - ▶ Large turnover of feed, high excretion of waste products
  - ▶ Predisposes to formation of crystals
  - ▶ 3/4 of mortality male kits
  - ▶ Male urethra long and narrow; voiding of calculi more difficult
- Uroliths from August onwards in the growing-furring period
- Breeding females during winter and lactation
  - ▶ Water balance is critical!



# Urinary Tract Infections and Bladder Stones

- Bladder infections may be caused by e.g. *Staphylococci*, *Pseudomonas spp.*, *Proteus spp.*, *E. coli*
- Urease producing bacteria raise pH of the urine
  - ▶ Urine becomes oversaturated
  - ▶ Crystals are formed
  - ▶ About 60 – 80 % of the bladder stones are formed after bladder infection



# Bladder Stones and Urine pH

- Most bladder stones found in mink are struvite uroliths
  - ▶ Magnesium ammonium phosphate
- **Struvite stones precipitate in alkaline urine > pH 7.0**
  - ▶ Dissolve in acidic urine < pH 6.6
  - ▶ Fasting urine pH in mink ranges from 6.05 to 6.50
  - ▶ Urine samples with crystals > pH 6.6



# Important Factors in Prevention of Urinary Tract Infections and Bladder Stones

## ● Animal's water balance

- ▶ Encourage consumption; *ad libitum* access
- ▶ Winter period, lactation

## ● Environment and management

- ▶ Encourage frequency of urination
- ▶ Size of enclosure (territory marking)
- ▶ Cage, nestbox, litter box hygiene
- ▶ Antimicrobial treatment of animals

## ● Genetic background



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# Important Factors in Prevention of Urinary Tract Infections and Bladder Stones

- Feeding of frequent small meals helps manage urine pH
- High diet digestibility, high caloric content
  - ▶ Mineral intake ↓, faeces volume ↓, faecal water output ↓
  - ▶ Volume of urine voided ↑
- Diet high in animal protein (SAA) decreases urine pH
- Lowering of feed pH will reduce/prevent stone formation
- Low magnesium (Mg) content in the feed is beneficial
- Diets high in cereal grain (K) produce more alkaline urine
- Dry diet increases risk





# Summary

- Mink nursing sickness and fatty liver disease common causes of mortality in mink
  - ▶ Metabolic diseases linked to non-ideal body condition
  - ▶ May result from loss of appetite or obesity
  - ▶ Focus on prevention; no effective treatment
- Bladder infections in June-July
- Stones increased from August onwards
  - ▶ Good hygiene, diet and water balance
  - ▶ Management of urine pH



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## *Code of Practice for the Care and Handling of Farmed Mink – Section 3: Feed and Water*

- Welfare and productivity are highest in breeders when they are managed to maintain an ideal body condition
- Proper nutritional management will help prevent or manage metabolic diseases
- Feeding programs should focus on minimizing fluctuations in body condition



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Thank you!



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