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WEBINAR: Setting the cow up for success

Tips for measurement & prevention of ketosis

Presented by SHAUN BALEMI

OVERVIEW

1. Ketosis overview

2. How to measure and interpret subclinical ketosis in transition and early lactation?

3. Tips for prevention of ketosis and improving peak milk/FCE

KETOSIS OVERVIEW

What is ketosis?

- A metabolic disorder measured by the level of ketone bodies in the blood
- Ketone bodies made up of beta-hydroxybutyrate, acetoacetate & acetone

What are these ketone bodies: BHB/BOHB, AcAc & Ac?

- These ketone bodies are produced as a secondary energy source alongside glucose as adipose tissue is mobilised
- However, high levels of adipose tissue mobilisation do not automatically mean high ketone bodies

How does ketosis relate to the liver?

- When the liver hepatocytes are “overloaded” by NEFA, they produce more ketone bodies as their capacity to convert NEFAs is limited
- This is where liver health and performance come into the equation, and why getting a complete picture of what’s happening in the cow is critical when dealing with ketosis

Melendez et al., 2024

NO KETOSIS

BALANCE IS CORRECT

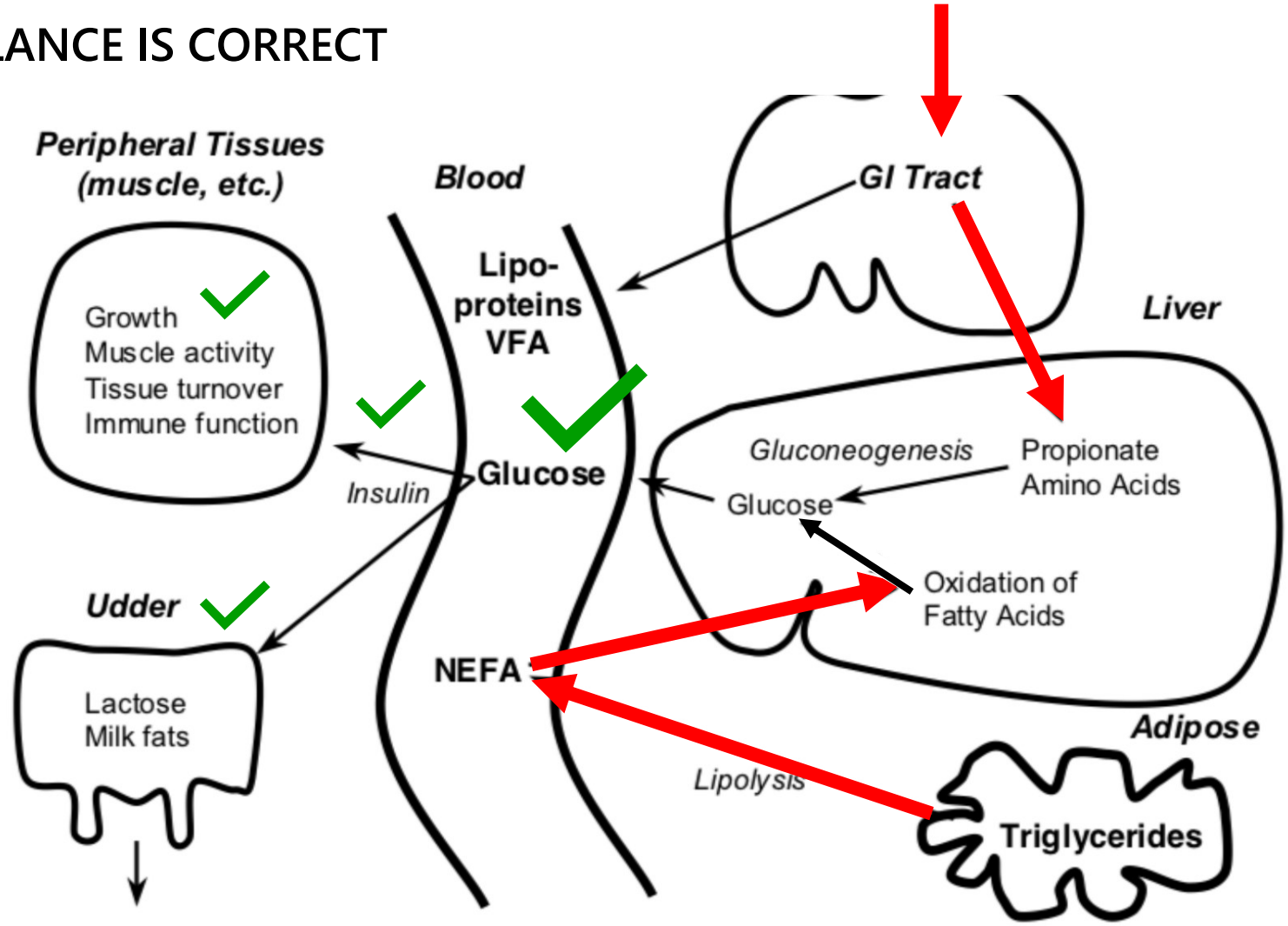


Figure 4. Schematic of glucose metabolism in a normal dairy cow.

1. Liver **IS** getting sufficient propionic acid + amino acids

2. Liver **IS** getting sufficient fatty acids + amino acids

OR

1. Fat mobilisation **IS** below the liver capacity limit

2. Liver **IS** oxidating NEFA efficiently

Cow function and milk production are **not** impaired

TYPE 1 KETOSIS: POOR NUTRITION

BALANCE IS OFF

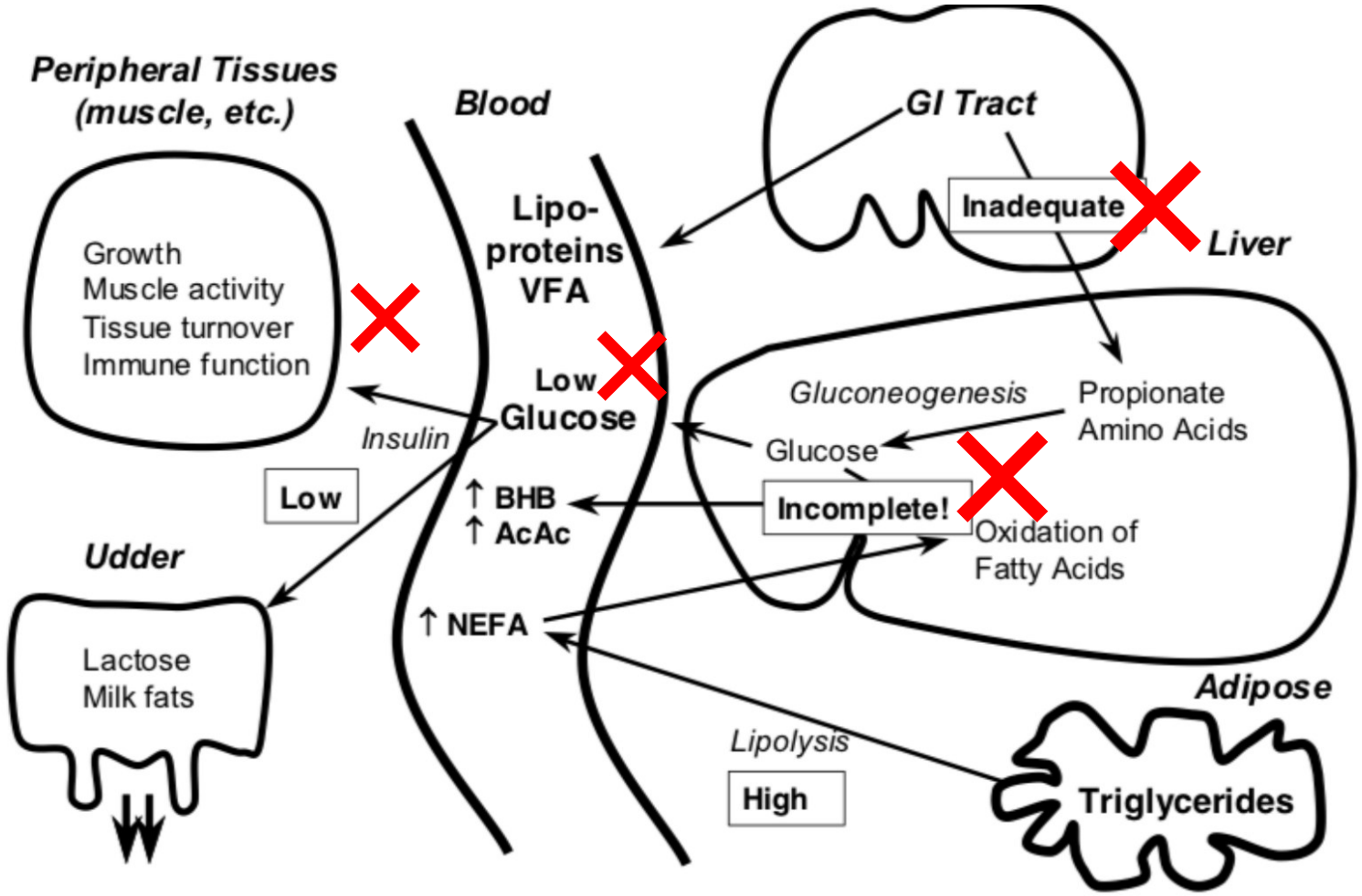


Figure 5. Schematic of glucose metabolism and ketone body formation with type I ketosis.

- Liver is NOT getting sufficient propionic acid and/or amino acids
- Liver CANNOT oxidate NEFA efficiently/completely
- NEFA, BHB & ACAC levels INCREASE
- NEFA entering the liver has reached liver capacity
- Cow function is impaired, milk production drop will follow

TYPE 2 KETOSIS: FAT COW/IMPAIRED LIVER

BALANCE IS *WAY* OFF

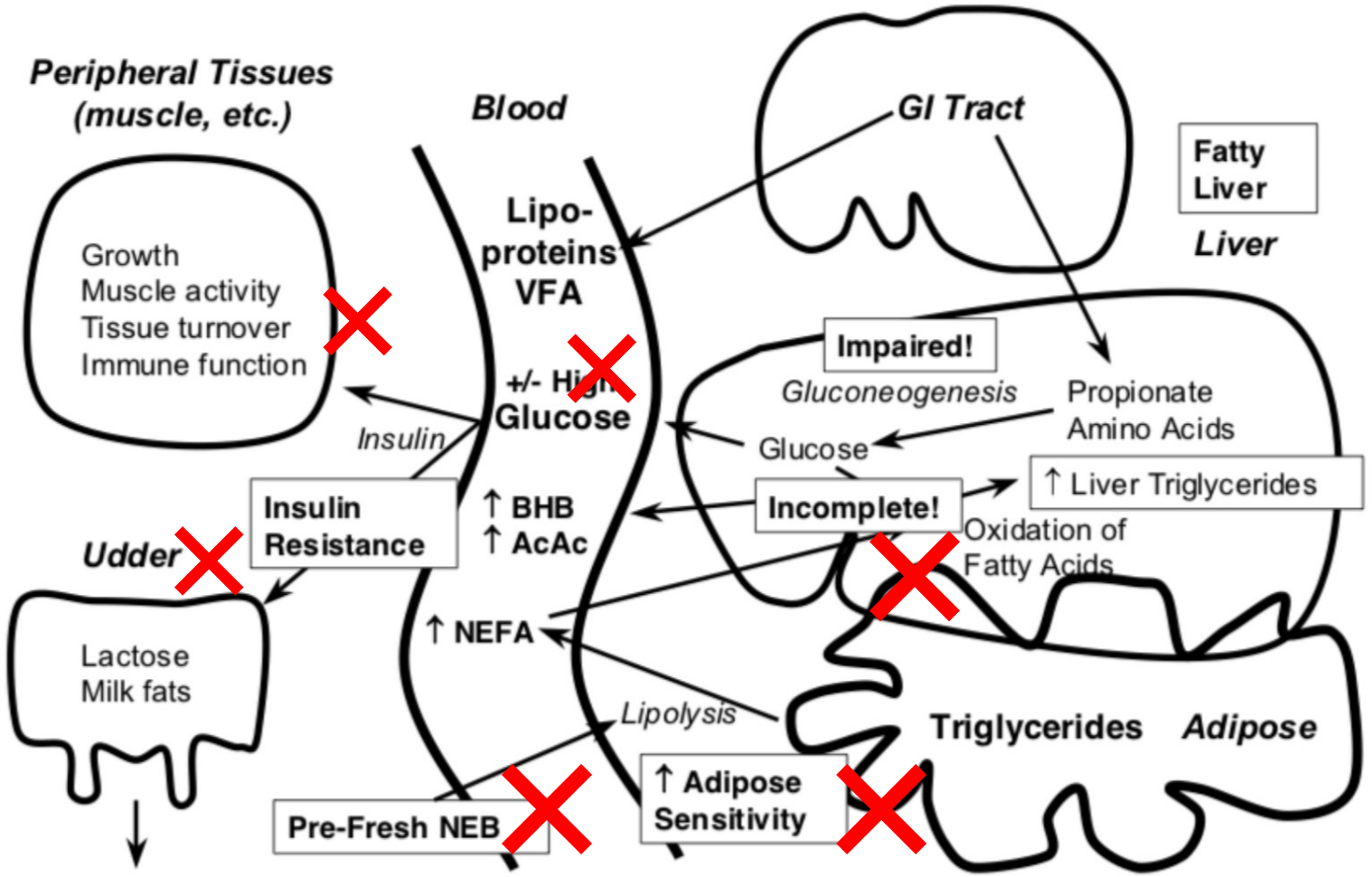


Figure 6. Schematic of glucose metabolism and ketone body formation with type II ketosis.

- Liver is getting sufficient propionic acid or amino acids
- Liver is overloaded with fat
- Gluconeogenesis IS restricted
- NEFA, BHB & ACAC levels continue to increase
- Fat mobilisation IS at a dangerous level
- Cow function and milk production are impaired
- Cow reproduction IS impaired from insulin resistance

TYPE 3 KETOSIS: BAD SILAGE

BALANCE IS OFF

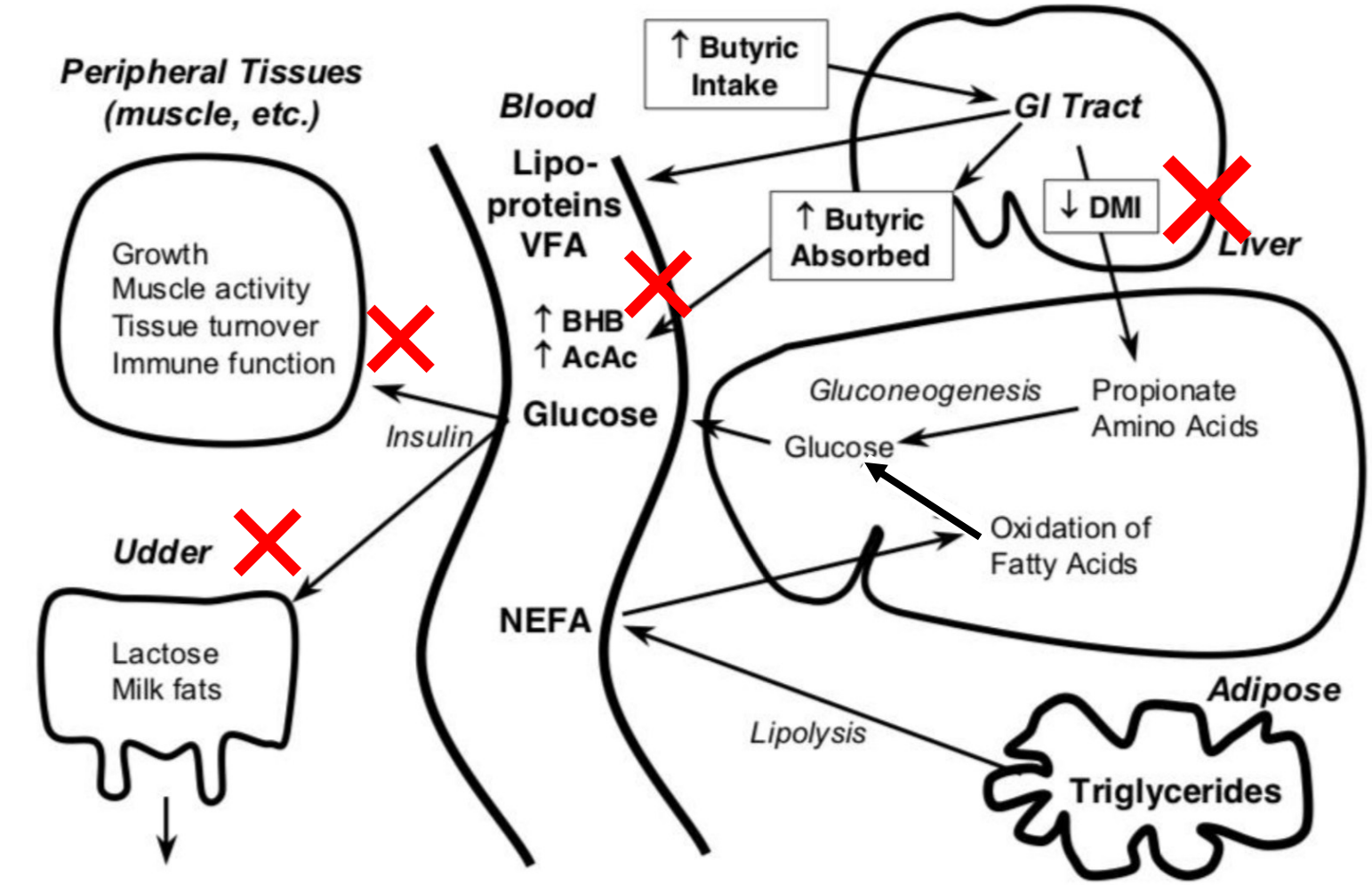
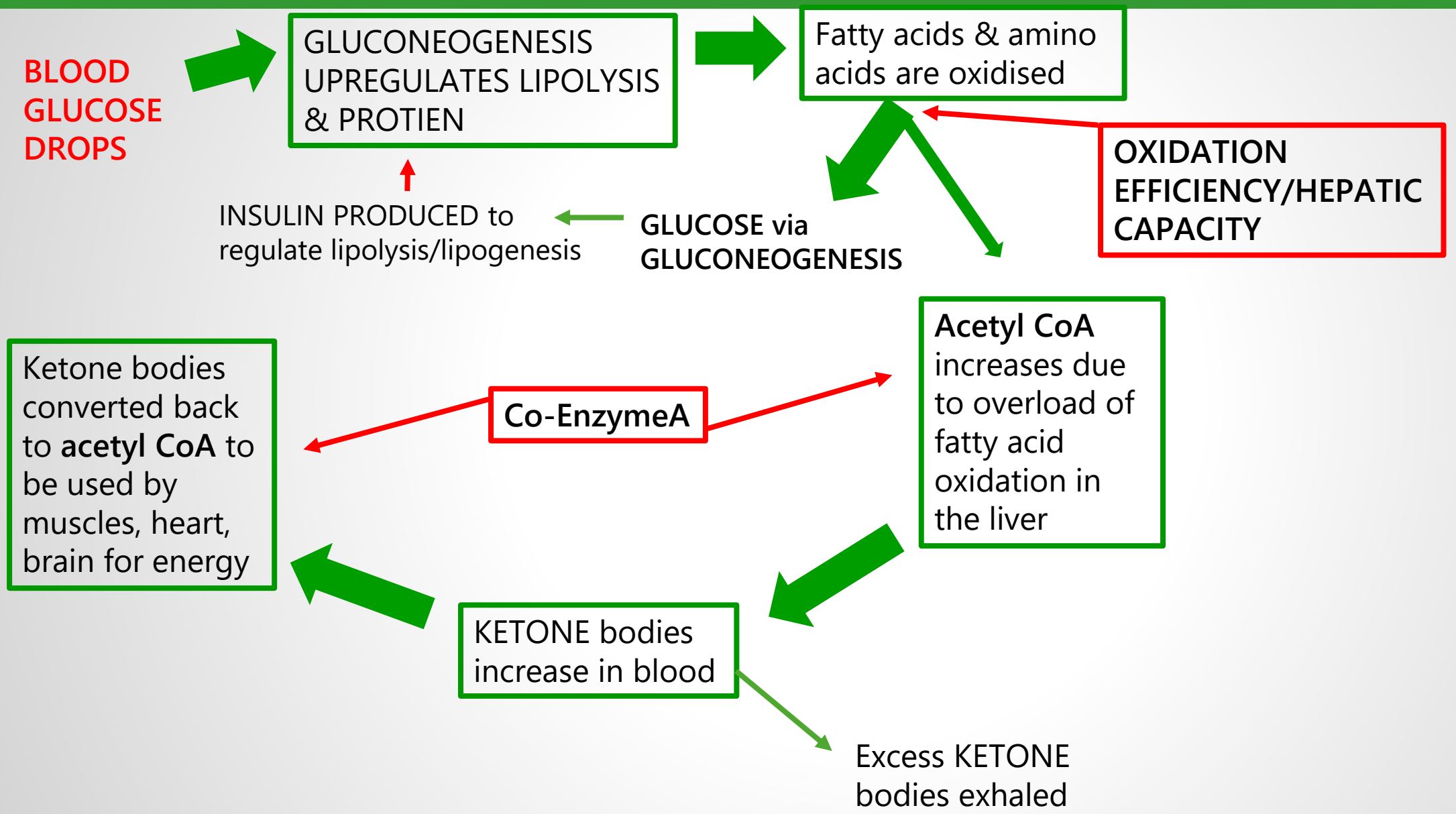


Figure 7. Schematic of glucose metabolism and ketone body formation in a dairy cow consuming excessive butyric acid from silage.

- High levels of butyric acid getting into the blood
- Liver is NOT getting sufficient propionic acid or amino acids due to depressed DMI
- Liver IS oxidating NEFA efficiently
- Only BHB & ACAC (liver damage) levels INCREASE
- Fat mobilisation is NOT above the dangerous level
- Cow function and milk production IS impaired

TIMELINE OF KETOSIS EVENTS



Adapted from Melendez et al., 2024

SIGNS & SYMPTOMS OF KETOSIS

Rapid condition loss

Downer cow/metabolic disease

Blood tests
NEFA >0.7 mEq/L
BHB > 0.8 mmol/L

Reduced appetite

Sweet smelling breath

Lacking energy

Unsteady on their feet

Cows are jumpy/ nervous

Excessive chewing, licking or salivating

BLOOD TESTING FOR KETOSIS

Adapted from McArt et al., 2013, Bertoni & Trevisi et al., 2013, Zhang et al., 2023, Veshkini etal., & Drackley et al., 2024

Non-esterified fatty acid = NEFA

Pre-calving…<0.3…0.3-0.5…>0.5 mEq/L

Post-calving…<0.7…0.7-1.0…>1.0 mEq/L

- Also known as free fatty acids
- Indicates the amount of fat being mobilised

Beta hydroxybutyrate = BHB/BHOB

Pre-calving…<0.4…0.4-0.7…>0.7 mEq/L

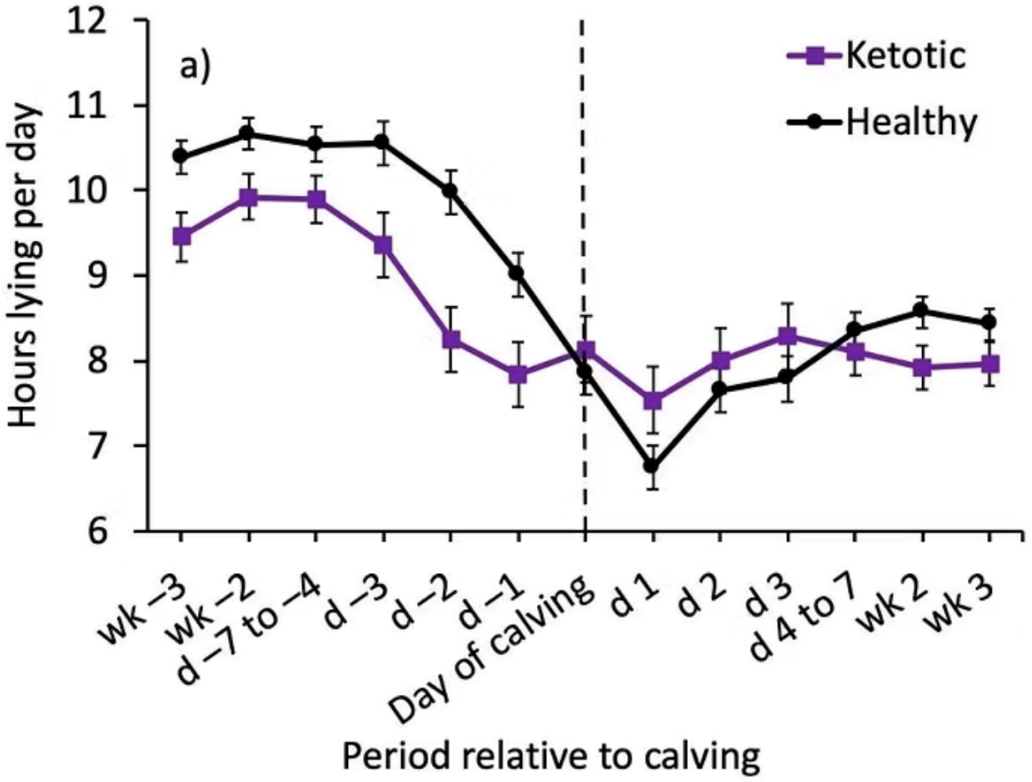
Post-calving…<0.8…0.8-1.2…>1.2 mEq/L

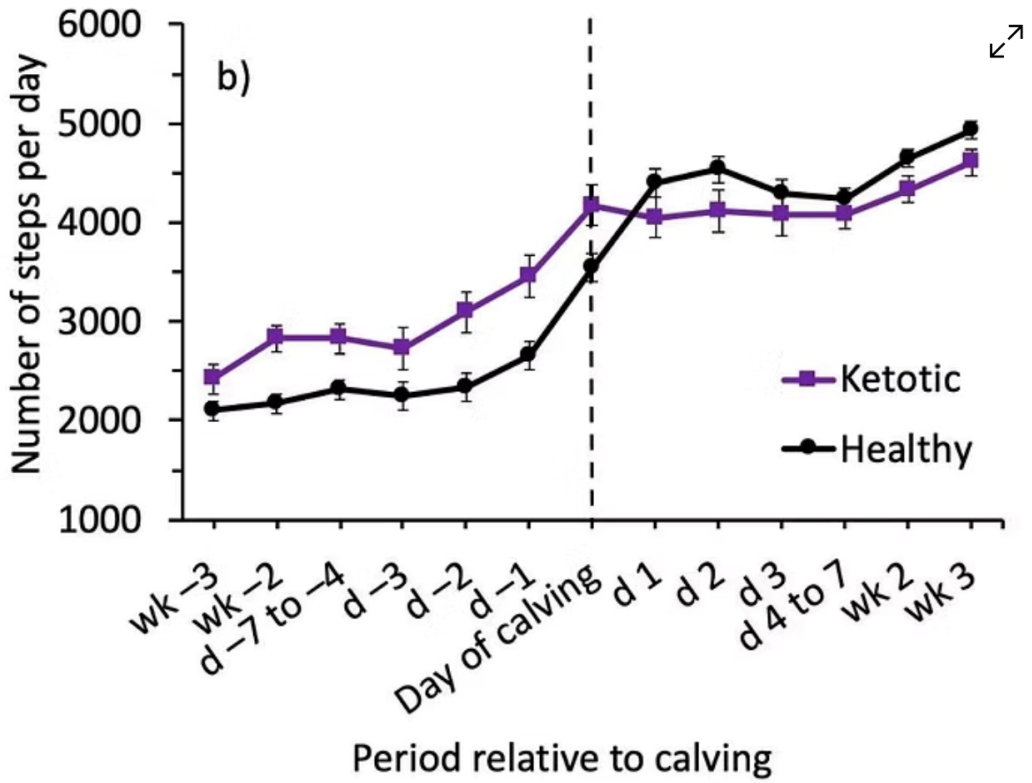
- Indication of how efficiently the cows are converting fatty acids into energy
- Can be skewed somewhat by high butyric acid feed

Liver function blood tests

- Glucose (Glu)……39-78 mg/dL
- Albumin (Alu)……3.2-3.8 g/dL
- Total cholesterol (TC)……3.0-6.0 mmol/L
- Total protein (TP)……6.0-8.3 g/dL
- Creatine kinase (CK)……<175 U/L
- Gama glutamyl transferase (GGT)……<45 U/L

COW ACTIVITY VIA WEARABLES & KETOSIS PREDICTION





Sub-clinincal ketotic = **>1.0mmol/L** NEFA & BHB
Healthy cows = **<1.0mmol/L** NEFA & BHB

- Research showed potential for ketosis prediction
- Lower lying mins pre-calving = Increased ketosis risk
- Higher activity mins pre-calving = Increased ketosis risk

Hendriks S., 2020

KETOSIS TREATMENTS

I am only going to cover nutritional/management solutions today

I want to focus on the triggers to help with solutions for prevention

<u>TREATMENTS</u>

- MPG
- Ketol
- Molasses/Soluble sugar
- Coated glucose?
- …..?

TRIGGERS FOR KETOSIS

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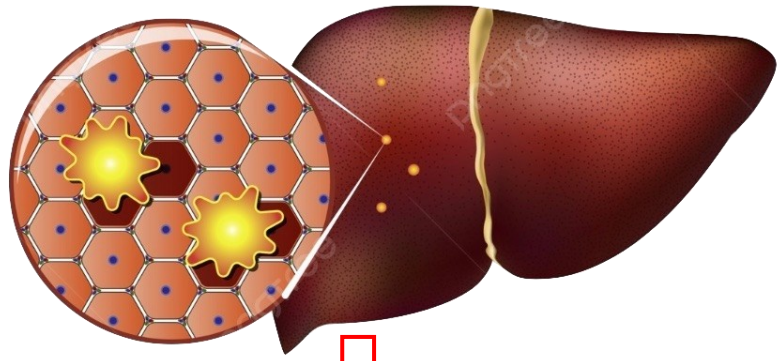
- Low protein diet
- Hypocalcemia
- Low energy density
- Poor transition
- Low protein stores
- Over-conditioned cows
- Poor intakes/appetite
- Rumen instability
- Poor liver health
- Rapid dry period condition gain



ELEVATED BLOOD KETONES

IMMUNOSUPRESSION

- ↓ Milk production
- ↓ 6-week in-calf rate
- ↓ Feed intakes
- ↑ Metritis & mastitis
- ↑ RFMs
- ↑ Condition loss

1. POOR TRANSITION FEEDING

- Increase transition length out - 21-28 days, ideal with history of ketosis.
- Calculate ME carefully: 90-130ME depending on LWT.
- Quality and quantity of protein: 14-18% CP, AA quality is important.
- Calculate and manage the DCAD: particularly if history of low blood calcium.
- Ensure trace minerals and key vitamins are supplemented.
- Betaine through transition and early lactation can help: 15g/cow/day.

2. POOR APPETITE LINKED TO HYPOCALCEMIA

- Confirm with full spectrum blood testing.
- Look at the transition diet, as per previous slide.
- Adjust calcium sources/availability in colostrums.
- Increase calcium and switch to higher bioavailable Ca sources short-term.
- Quality starch and quality protein will improve appetite.
- Calcium bolus for at-risk cows can be a short-term solution, but still needs to fix the underlying issue.

3. LOW FEED INTAKE: RUMEN INSTABILITY/STRESS

- This is shown by variable milk volumes and low fat% in milk.
- Can also show in poor immune system function.
- Stabilise the rumen with calsea/rumen buffer and live yeast/yeast metabolite.
- Make sure the balance of fibre with carbohydrate is correct.
- Increasing glucogenesis will help refire appetite, increase starch and quality protein.

4. LOW ENERGY DENSITY OF DIET

- Low grass availability/quality, wet weather, and low sunlight hours all culminate to make feed energy density a challenge.
- Quality feed supplements are the first critical element.
- Having highly palatable feed is also important.
- Starch and sugars are often an easy way to lift energy density quickly and easily – grain and molasses.
- Make sure you match energy density with quality protein, or you will trade one deficiency for another.
- Make sure phosphorus and calcium levels are properly balanced, as glucose and sugar utilisation require more phosphate.
- Transition onto starch and having rumen stability tools like calsea and yeast is important for good starch utilization.

5. RUN OUT OF STORED AMINO ACIDS

- Often seen 10-14 days into lactation when rumination, production drops, and it can trigger rapid condition loss. Protein stores have run dry.
- 30% of protein requirements must be pulled from the muscle in early lactation until intakes match production
- Feeding good quality protein through the dry period, transition, and early lactation period is the best way to prevent protein stores from running out
- Soya meal, quality canola, single-cell protein or straight amino acids. Methionine will help.

6. POOR LIVER FUNCTION

- Seeing poor appetite, condition loss, immune system issues or bloods showing high NEFA/BHB and low albumin/glucose/cholesterol.
- This requires a combined approach: feed + additives.
- Increasing the energy and protein density of the diet alongside rumen stabilisers, as the rumen will already be stressed. This will increase propionate going to the liver.
- Betaine to help clean fat out of the liver and increase efficiency, giving the liver a chance to repair and insulin sensitivity to come down.
- Chromium will help to increase insulin sensitivity.
- B-group vitamins and selenium might also be needed for liver repair.

7. OVER-CONDITIONED COWS

- Cows have wintered better than expected and are above 5.5BCS on average.
- High cow condition means more stored fat, which can mean a higher susceptibility to ketosis.
- We can manage this by carefully managing and monitoring the transition diet: Maintenance ME, but do not underfeed!
- Feeding the early lactation cows a high-energy energy-dense diet.
- High-energy starter drenches can help along with betaine through transition and early lactation.
- If ketosis issues start, use all the techniques from the previous slide "Poor Liver Function".

8. High Visceral/Abdominal Fat

- Slightly over-conditioned cows are not always an issue - it's the dynamics of fat mobilisation that are.
- There are three types of fat deposition in cows: Subcutaneous (under skin), visceral (around organs) and muscular.
- When cows gain condition quickly, they build more visceral fat. When cows gain condition slowly, they build more subcutaneous fat.
- There is an association between higher levels of visceral fat and a higher risk of ketosis.
- Building/maintaining good condition in our cows is critical, but building it slowly is just as critical.

Melendez et al., 2024

FEED ADDITIVE TOOLS FOR KETOSIS

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1. Copper/zinc/manganese
2. Quality protein
3. Quality starch
4. B-group vitamins
5. Betaine: 15g/cow/day
6. Selenium: RP 8mg/cow/day
7. Chromium: 9mg/cow/day
8. Boron:120mg/cow/day
9. Calsea/yeast: 50-70gm/3gm
10. Monensin: 300mg/cow/day

THANK YOU FOR ATTENDING

For more information and previous webinars:

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