Who Let the Dogma Out of Transition Cow Physiology

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1

2

"Todays problems stem from yesterday's solutions."

-Peter Senge 2010 in "The Fifth Discipline"

"It's what we think we know that keeps us from learning."

-Claude Bernard

3

Nutritionists are Often Incorrectly Blamed for:

- High NEFA
- Hyperketonemia
  - Clinical and subclinical ketosis
- Subclinical hypocalcemia

- These are due to 1 of 2 things:
  - High productivity in healthy cows (profitable dairy producer)
    - The nutritionist deserves a raise
  - Metabolic reflection of immune activation
    - Likely stemming from metritis, mastitis, pneumonia or GIT inflammation
      - These are mostly management issues and not caused by nutrition

4

Everything in today's talk is more thoroughly covered in our recent review

Horst et al., 2021, JDS 14:8380-8410

J. Dairy Sci. 104:8380–8410
https://doi.org/10.3168/jds.2021-20330

Invited review: The influence of immune activation on transition cow health and performance—A critical evaluation of traditional dogmas

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Department of Animal Science, Iowa State University, Ames 50011

ABSTRACT

The progression from gestation into lactation represents the transition period, and it is accompanied by marked physiological, metabolic, and inflammatory adjustments. The entire lactation and a cow's opportunity to have an additional lactation are heavily dependent on how successfully she adapts during the periparturient period. Additionally, a disproportionate amount of health care and culling occurs early following parturition. Thus, lactation maladaptation has been a heavily researched area of dairy science for more than 50 yr. It was traditionally thought that excessive adipose tissue mobilization in large part dictated transition period success. Further, the magnitude of hypocalcemia has also been assumed to partly control whether a cow effectively navigates the first few months of lactation. The canon became that adipose tissue released nonesterified fatty acids (NEFA) and the resulting hepatic-derived ketones coupled with hypocalcemia lead to immune suppression, which is responsible for transition disorders (e.g., mastitis, metritis, retained placenta, poor fertility). In other words, the dogma evolved that these metabolites and hypocalcemia were causal to transition cow problems and that large efforts should be enlisted to prevent increased NEFA, hyperketonemia, and subclinical hypocalcemia. However, despite intensive academic and industry focus, the periparturient period remains a large hurdle to animal welfare, farm profitability, and dairy sustainability. Thus, it stands to reason that there are alternative explanations to periparturient failures. Recently, it has become firmly established that immune activation and the ipso facto inflammatory response are a normal component of transition cow biology. The origin of immune activation likely stems from the mammary gland, tissue trauma during parturition, and the gastrointestinal tract. If inflammation becomes pathological, it reduces feed intake and causes hypocalcemia. Our tenet is that immune system utilization of glucose and its induction of hypophagia are responsible for the extensive increase in NEFA and ketones, and this explains why they (and the severity of hypocalcemia) are correlated with poor health, production, and reproduction outcomes. In this review, we argue that changes in circulating NEFA, ketones, and calcium are simply reflective of either (1) normal homeorhetic adjustments that healthy, high-producing cows use to prioritize milk synthesis or (2) the consequence of immune activation and its sequelae.
Key words: inflammation, hypocalcemia, ketosis, insulin, homeorhesis

THE PERIPARTURIENT PERIOD

Early lactation is a unique physiological state in which nutrient consumption often does not meet maintenance and milk production costs, creating a negative energy balance (**NEB**; Drackley, 1999). Milk energy output increases more rapidly than the increase in consumed energy. The magnitude of NEB varies, but nadir usually occurs within the first 10 DIM, and cows return to calculated positive energy balance between 30 and 100 DIM (Moallem et al., 2000; Coffey et al., 2002). To support milk synthesis during NEB, significant alterations in carbohydrate, lipid, protein, and mineral metabolism are implemented.

A thorough appreciation of how important glucose is to milk synthesis is required to understand why these changes (energetics in particular) occur. Glucose is the precursor for lactose synthesis, and lactose is the primary osmoregulator driving milk volume (Neville, 1990). For every 1 kg of milk produced, approximately 72 g of glucose is required (Kronfeld, 1982). During established lactation, hepatic glucose output is exquisitely orchestrated to precisely meet peripheral tissue (e.g., mammary, muscle, adipose, central nervous system) glucose requirements (Baumgard et al., 2017). However, inadequate feed intake during the periparturient period means that the contribution of diet-derived gluconeogenic precursors to hepatic glucose output is insufficient to meet the mammary gland's increasing

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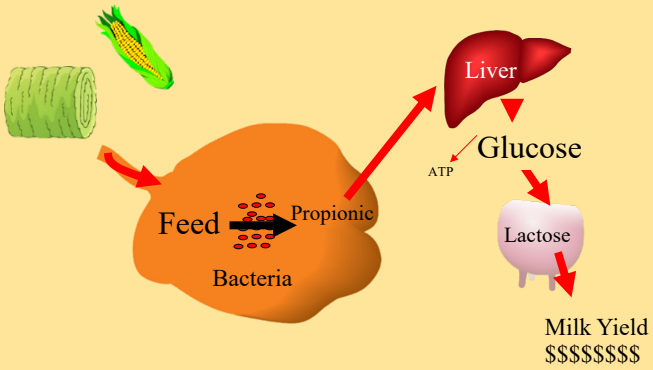
5

Glucose is made from propionate
Lactose is made from glucose
It requires 72 g of glucose to synthesize 1 kg of milk (Kronfeld et al., 1982)
Milk yield is determined by the amount of synthesized lactose

Friendly reminder of how important glucose is to milk synthesis



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Foundation of Our Thinking

- The best indicators of "health" are feed intake and milk yield.
- If everyone agrees that "stress" reduces productivity……..
  then high productivity CANNOT be stressful
  - The idea that high milk production is stressful is an oxymoron
- It's too easy to overthink the immune system.
  - Don't need a PhD or DVM to know when a cow is healthy
  - Cows that are eating and producing large quantities of milk ARE healthy
- Inconsistent data creates doubt
  - When scientific papers do not agree we should think twice

7

Transition Period Morbidity

Disorders affects 50%:

- D
- M
- R
- M
- K
- DA
- Fatty liver
- Lameness
- Death

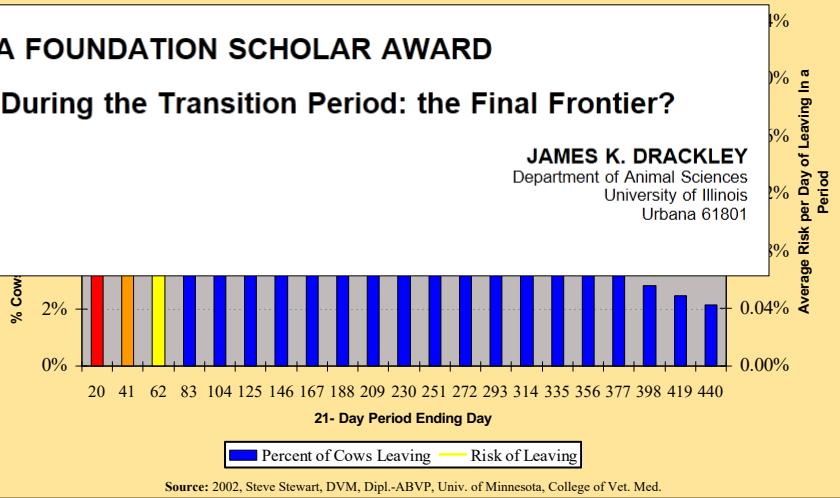
Drackley, 1999

When cows leave the herd

ADSA FOUNDATION SCHOLAR AWARD

Biology of Dairy Cows During the Transition Period: the Final Frontier?

JAMES K. DRACKLEY
Department of Animal Sciences
University of Illinois
Urbana 61801



Source: 2002, Steve Stewart, DVM, Dipl.-ABVP, Univ. of Minnesota, College of Vet. Med.

8

Incidence of Health Disorders
in the First 60 d in Milk in High-Producing Holstein Herds

5,719 postpartum dairy cows evaluated daily for health disorders from eight experiments in seven dairy farms in the US.

Healthy: 56%
Calving problem: 15%
Metritis: 16%
Clinical endometritis: 21%
Fever: 21%
Mastitis: 12%
Ketosis: 10%
Lameness: 7%
Pneumonia: 2%
Digestive: 3%

44% Unhealthy

Santos et al. (2010) Reprod. Dom. Rum. VII:387-404

9

Path Analysis of Dry Period Nutrition, Postpartum Metabolic and Reproductive Disorders, and Mastitis in Holstein Cows[1]

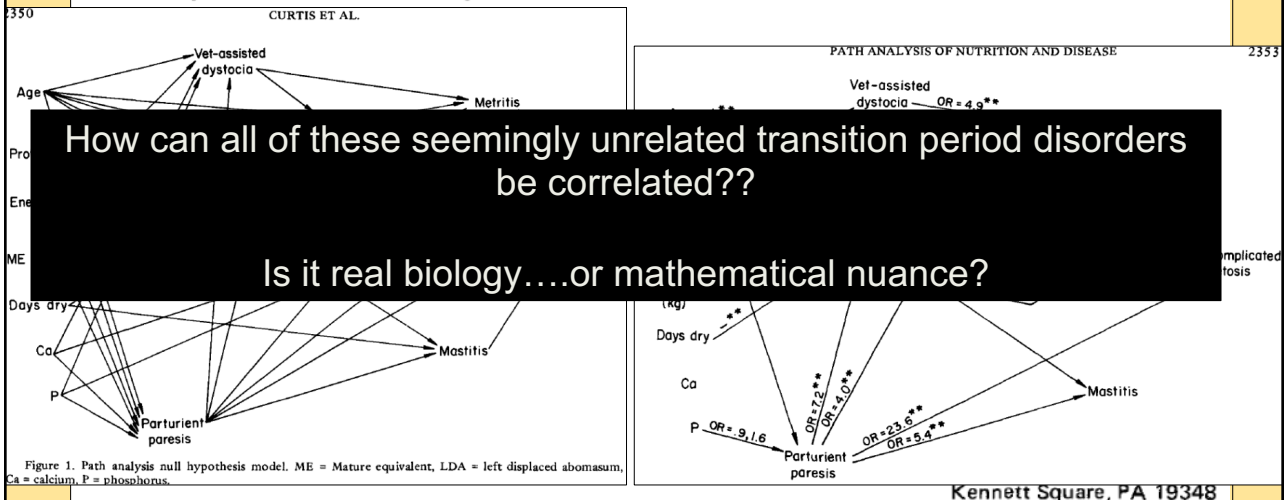


Figure 1. Path analysis null hypothesis model. ME = Mature equivalent, LDA = left displaced abomasum, Ca = calcium, P = phosphorus.

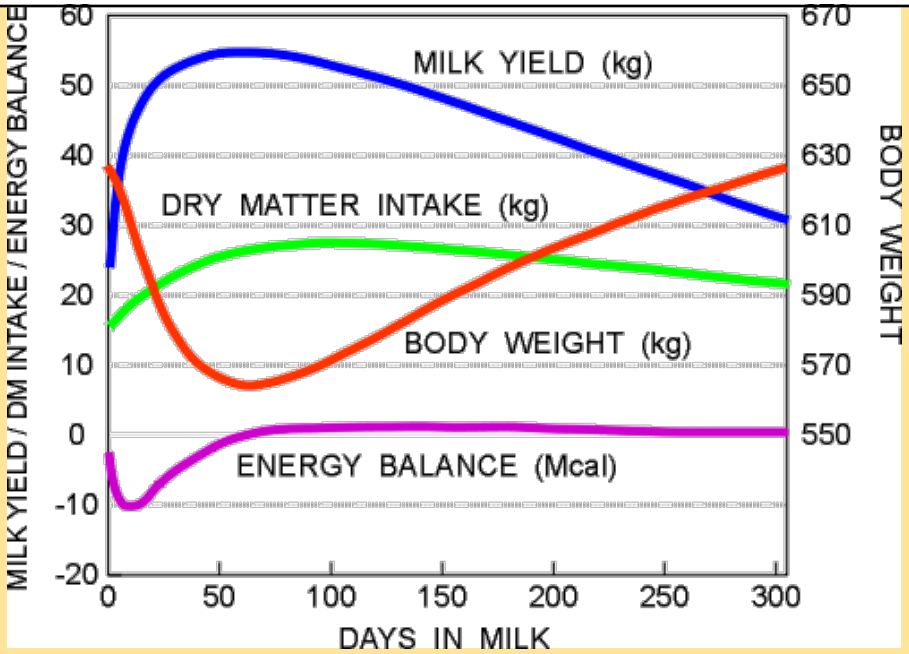
How can all of these seemingly unrelated transition period disorders be correlated??

Is it real biology….or mathematical nuance?

Kennett Square, PA 19348

1985 J Dairy Sci 68:2347–2360

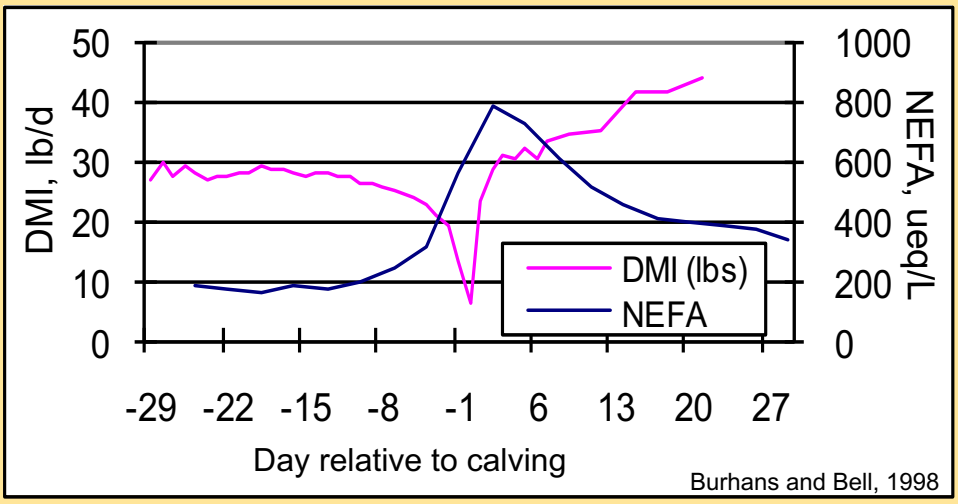
10



Alberta Dairy Management

11

Transition Period Energy Balance



Burhans and Bell, 1998

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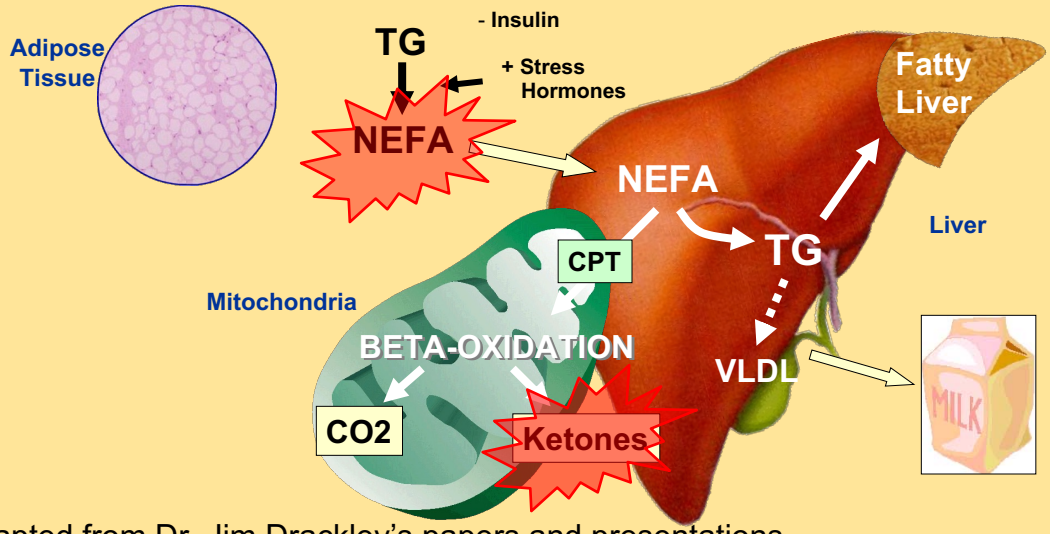
Liver Lipid Metabolism During the Transition Period



Adapted from Dr. Jim Drackley's papers and presentations

13

Retrospective and Observational Studies

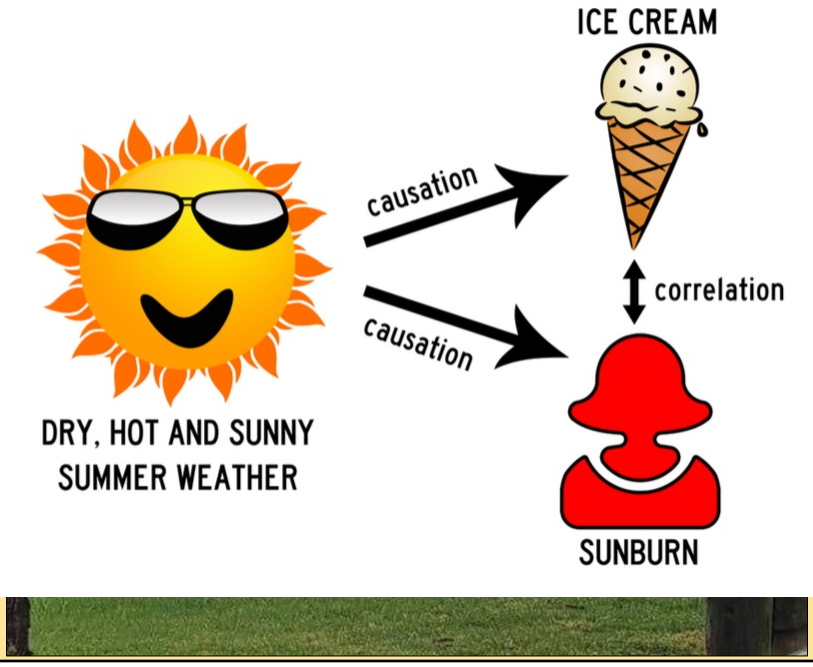
- Hundreds of studies *associate* and correlate NEFA, BHBA and Ca with:
  - Increased risk of ketosis, decreased milk yield, LDA, metritis, retained placenta, laminitis, or poor reproduction
    - Chapinal et al., 2011; Huzzey et al., 2011; Ospina et al., 2010a, 2010c; Duffield et al., 2009; LeBlanc et al., 2005
- Many papers do not agree…..inconsistent effects in the literature
- Plasma NEFA are markedly increased (>700 mEq/L) following calving in almost all cows
  - ~15-20% get clinical ketosis
  - What makes these cows more susceptible to ketosis?
    - Predisposition to developing fatty liver?
- Reductionist approach (one metabolite = one disease)

14

Cause and Effect??

- The incidence of health problems is highest in the first month of lactation
- The largest swings in energetic metabolites, hormones and minerals occurs in the first month of lactation
- Thus…a lot of moving parts and events occurring simultaneously
  - Consequently they will all be correlated
- Causality and correlation are incorrectly interchanged when an observational relationship between 2 events is claimed to be inevitable rather than coincidental.

15



16

This correlation interpretation then causes suspect decision making and unnecessary farm expenses

17

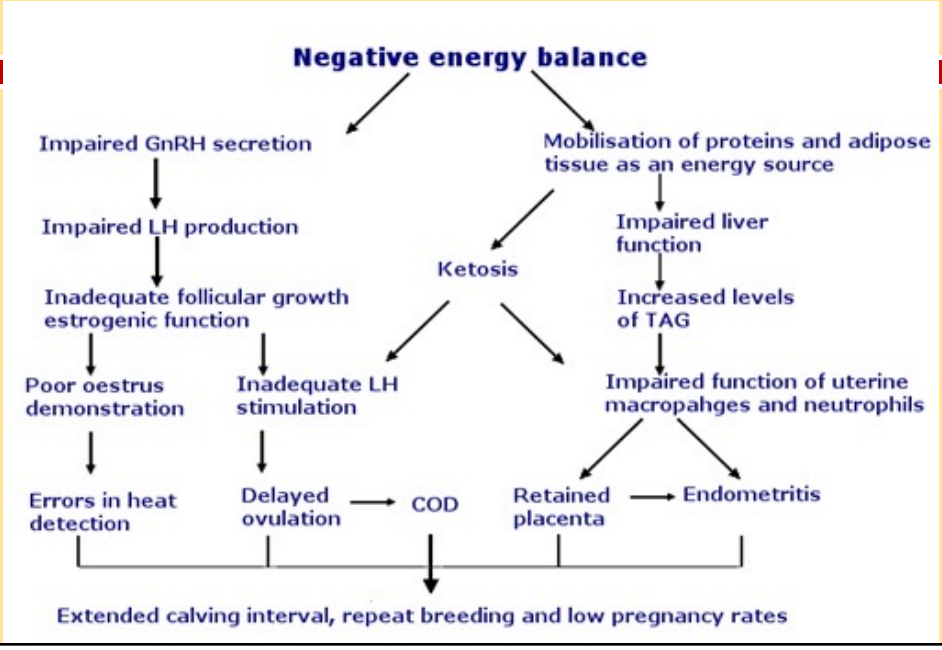
Traditional Belief

Assuming Correlation Equals Causation

Increased NEFA, Hyperketonemia, and Hypocalcemia…..**CAUSE** production and health problems

18

Dogma: Negative Energy Balance CAUSES problems



19

Dogma: Ketones cause problems

https://slideplayer.com/slide/13774593/

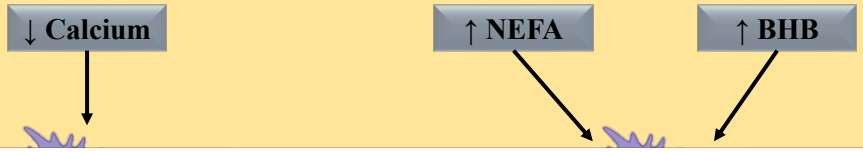
20

How (and why) do NEFA, Hyperketonemia and Hypocalcemia cause problems

- Biological plausibility?
  - Why would evolution favor a scenario where the mother endangers herself and compromises her ability to nourish her young?
- There remains little mechanistic evidence for how NEFA, ketones and Ca can directly have such a large influence on a variety of seemingly unconnected systems and diseases
- Best line of evidence is extrapolated from their purported role in immunosuppression

21

Impact on Leukocyte Function



But too many inconsistencies (as reviewed by LeBlanc, 2020)

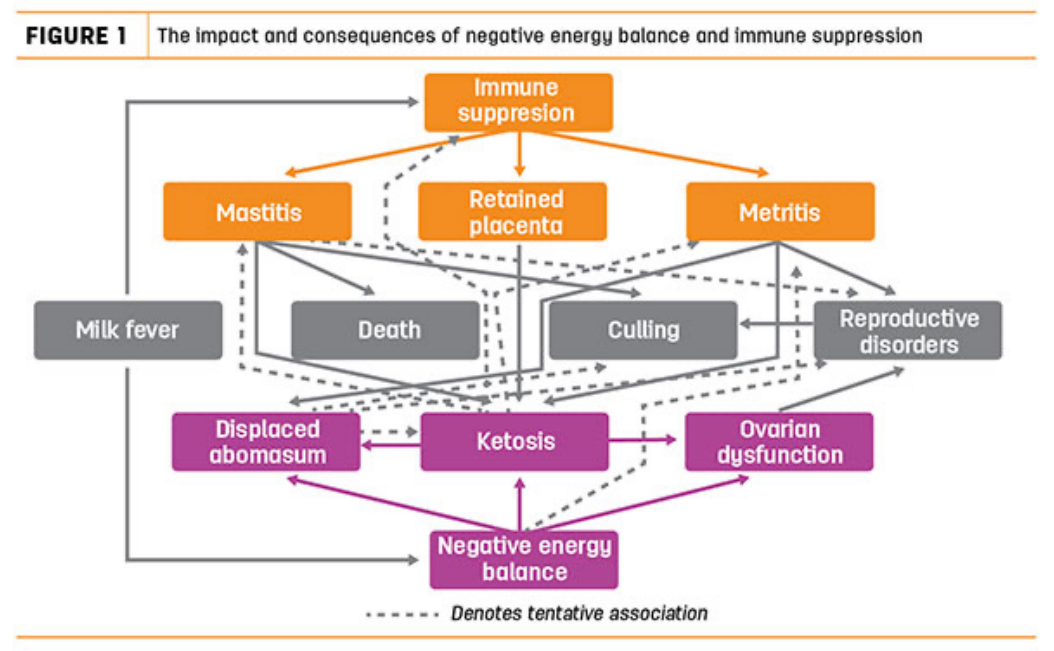
A solid foundation of any dogma should be a consistent pattern

↓ intracellular Ca stores
↓ neutrophil phagocytosis
(Ducusin et al., 2003; Kimura et al., 2006; Martinez et al., 2014))

No change in phagocytosis (Scalia et al., 2006)
No difference in mononuclear cell proliferation or cytokine production (Ster et al., 2012)
No impact on neutrophil killing ability (Hammon et al., 2006)

22

Dogma:



https://www.progressivedairy.com/topics/herd-health/four-ways-to-reduce-the-risk-of-transition-cow-problems

23

If hyperketonemia, high NEFA and subclinical hypocalcemia are pathological….it stands to reason that therapeutically treating these disorders would improve cow health

24

Culling Trends Over Time

Culling Reason	NAHMS (1996)	NAHMS (2002)	NAHMS (2014)
Voluntary Reasons	21.3	19.3	21.1
Re			
Injury	4.1	6.0	5.2
Death	3.8	4.8	4.2
Disposition			
Lameness	14.2	16.3	16.8
Other	3.9	4.1	

Despite emphasis, time and money spent on preventing high NEFA, hyperketonemia and subclinical hypocalcemia herd health is not improving

Maybe we're "medicating" the wrong things??

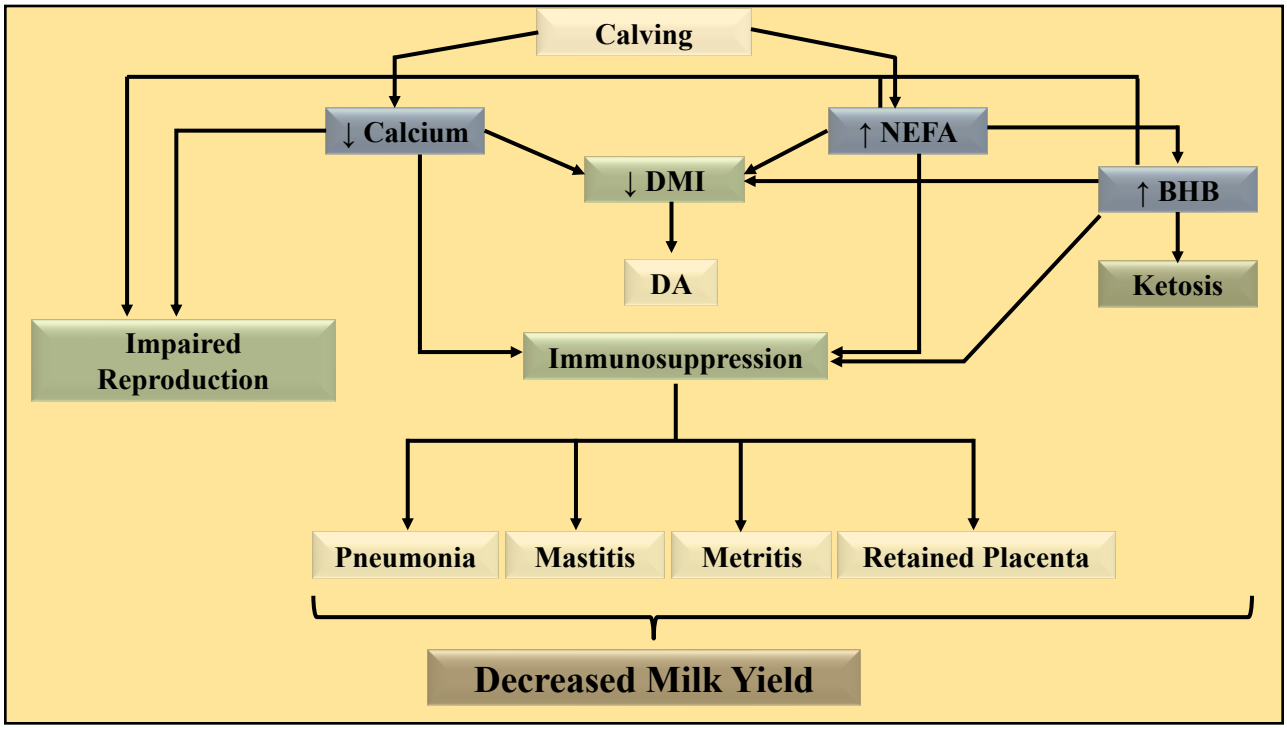
National Animal Health Monitoring Systems

25

Traditional Belief

Increased NEFA, Hyperketonemia, and Hypocalcemia…..**CAUSE** production and health problems

26



27

Historical Dissection the Dogmas

28

Ketosis Dogma

- Excess adipose tissue mobilization causes fatty liver and ketosis
- This is exacerbated in high producing cows
- Academic & Industry Goal: Reduce blood NEFA

29

Observations that should have been red flags:

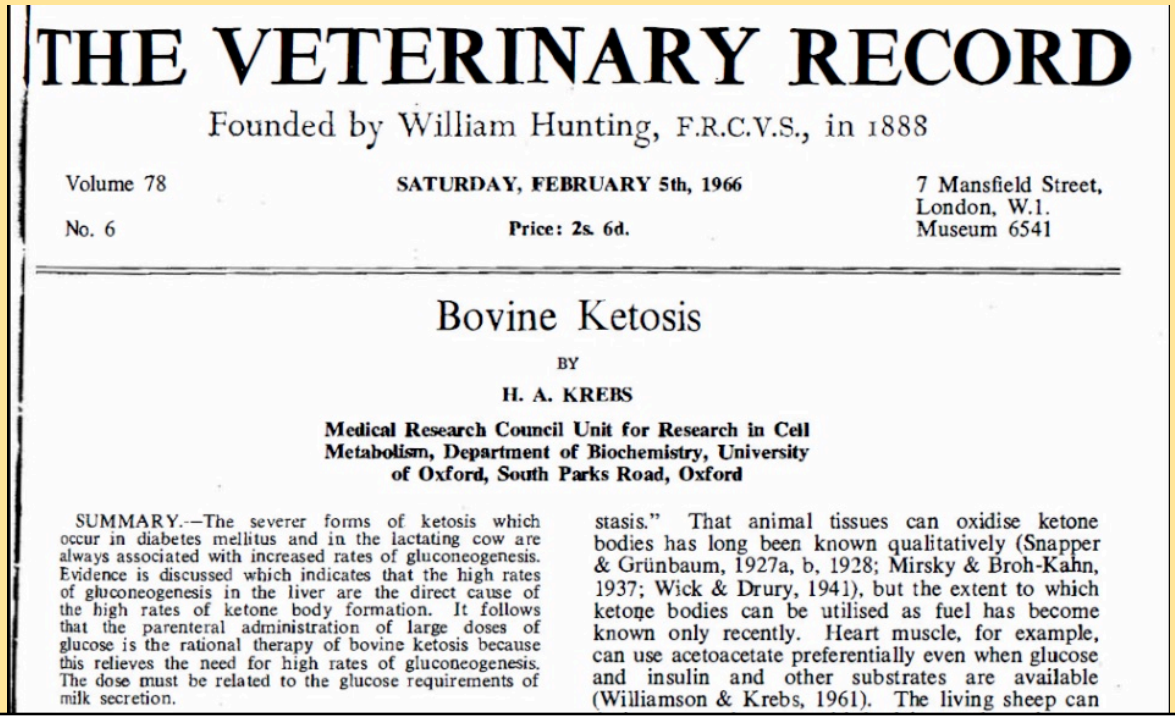
1) Associations and correlations
   - No cause and effect…..and the correlations are weak anyway…not probative
2) Infusing ketones or NEFA does not cause negative outcomes
   - In nature, animals ebb and flow out of ketosis ALL the time
3) Ketotic cows are not hypoinsulinemic
   - Often times they are hyperinsulinemic
4) Ketones do not decrease feed intake
   - Otherwise a starving animal would not have an appetite
5) Preventing adipose mobilization reduces milk yield
   - Transition period hyperinsulinemia is associated with immediate and long-term low milk yield
   - Insulin or TZD administration
6) Some females do not consume any food after parturition
   - Ocean mammals
7) Regional differences in the rate of clinical ketosis
   - Clinical ketosis rates in Arizona are less than 1%. Most dairy producers in AZ have never treated a ketotic cow.

30

Hyperketonemia

- Ketogenesis is the coordinated convergence of CHO and Lipid metabolism
- Highly conserved amongst almost all mammals
  - Some ocean mammals are an exception
- Ancient strategy that even the simplest of organisms utilize during energy insufficiency
- Millions of people are on low CHO/ketogenic diet
  - Dozens of meta-analysis papers in the scientific literature
- Claiming hyperketonemia is a disease is akin to assigning hyperglycemia as the pathological origin of diabetes

31



THE VETERINARY RECORD

Founded by William Hunting, F.R.C.V.S., in 1888

Volume 78 | SATURDAY, FEBRUARY 5th, 1966 | 7 Mansfield Street, London, W.1.

No. 6 | Price: 2s. 6d. | Museum 6541

Bovine Ketosis

BY

H. A. KREBS

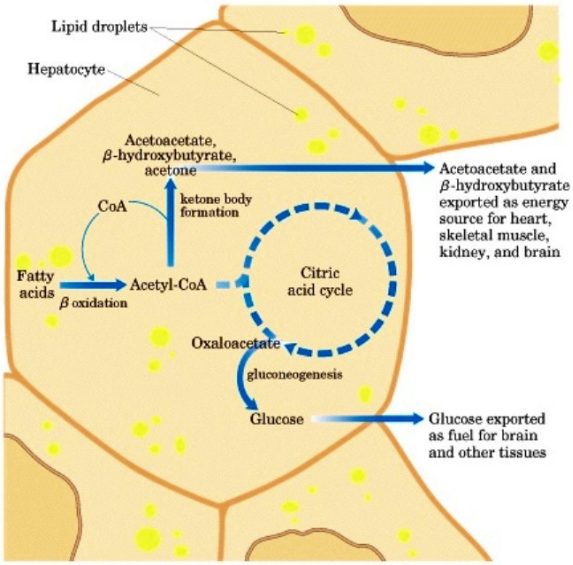
Medical Research Council Unit for Research in Cell Metabolism, Department of Biochemistry, University of Oxford, South Parks Road, Oxford

SUMMARY.—The severer forms of ketosis which occur in diabetes mellitus and in the lactating cow are always associated with increased rates of gluconeogenesis. Evidence is discussed which indicates that the high rates of gluconeogenesis in the liver are the direct cause of the high rates of ketone body formation. It follows that the parenteral administration of large doses of glucose is the rational therapy of bovine ketosis because this relieves the need for high rates of gluconeogenesis. The dose must be related to the glucose requirements of milk secretion.

stasis." That animal tissues can oxidise ketone bodies has long been known qualitatively (Snapper & Grünbaum, 1927a, b, 1928; Mirsky & Broh-Kahn, 1937; Wick & Drury, 1941), but the extent to which ketone bodies can be utilised as fuel has become known only recently. Heart muscle, for example, can use acetoacetate preferentially even when glucose and insulin and other substrates are available (Williamson & Krebs, 1961). The living sheep can

32

Ketone bodies in metabolism



33

Partitioning of Nutrients During Pregnancy and Lactation: A Review of Mechanisms Involving Homeostasis and Homeorhesis

DALE E. BAUMAN and W. BRUCE CURRIE
Department of Animal Science
Cornell University
Ithaca NY 14853

ABSTRACT

Control of metabolism during preg-

tions and physiological processes in which food is transformed into body tissues and activities

Received January 28, 1980.

1980 J Dairy Sci 63:1514–1529

1514

environment (Figure 2). There are many well established examples of homeostasis, such as regulation to maintain constancy of body

← Introduced the Homeorhesis concept

Mobilization of adipose tissue and partial conversion of NEFA into Ketones is ESSENTIAL for maximum milk yield in early lactation

34

Common Critique of My Message

- When PG is given, SHK cows produce more milk
  - McArt et al., 2011
    - 2 out of 3 farms had increased milk yield when SHK cows given PG
  - Lomander et al., 2013
  - [illegible]

The body of evidence does not support the practice of giving subclinical hyperketonemic cows PG

- But inconsistent patterns. No effect of PG on milk in SHK cows
  - Bors et al., 2013
  - Ostergaard et al., 2020
  - Capel et al., 2021

35

Transition Period and Insulin

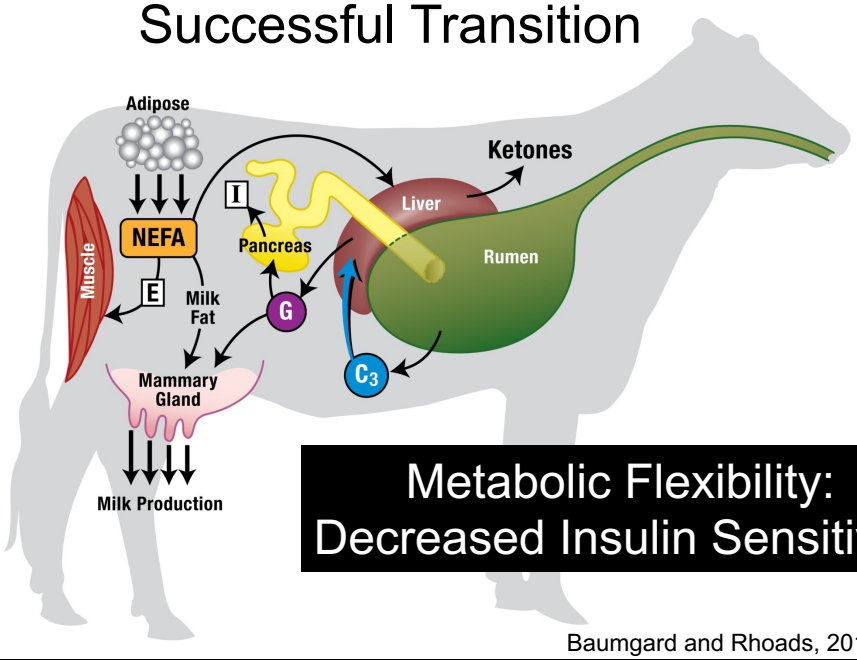
- Higher producing cows are more hypoinsulinemic
- Periparturient insulin is inversely related to whole lactation performance
- Insulin clearance is increased by genetic selection for milk yield
- Administering insulin or insulin sensitizing agents decrease milk yield

The primary endocrine profile required for high production is hypoinsulinemia

Horst et al., 2021

36

Successful Transition



Metabolic Flexibility: Decreased Insulin Sensitivity

Baumgard and Rhoads, 2013

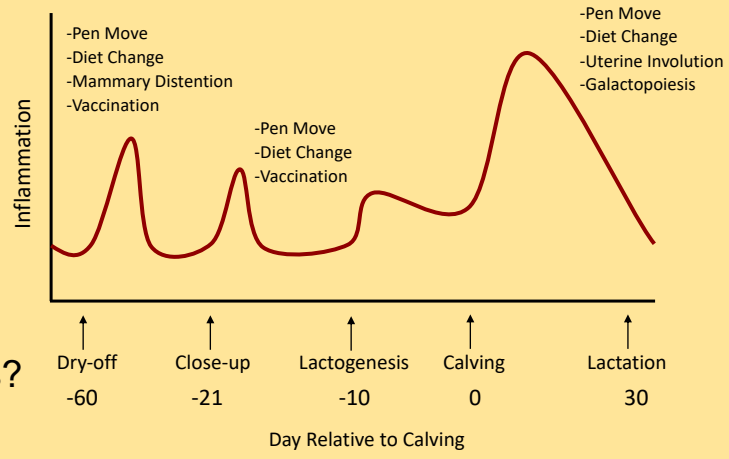
37

Inflammation in Transition Cows

- Observed in all cows

(Bertoni et al., 2008; Trevisi and Minuti, 2018)

- What is the source?
  - Mammary Gland
  - Uterus
  - Gastrointestinal tract
- What are the consequences?



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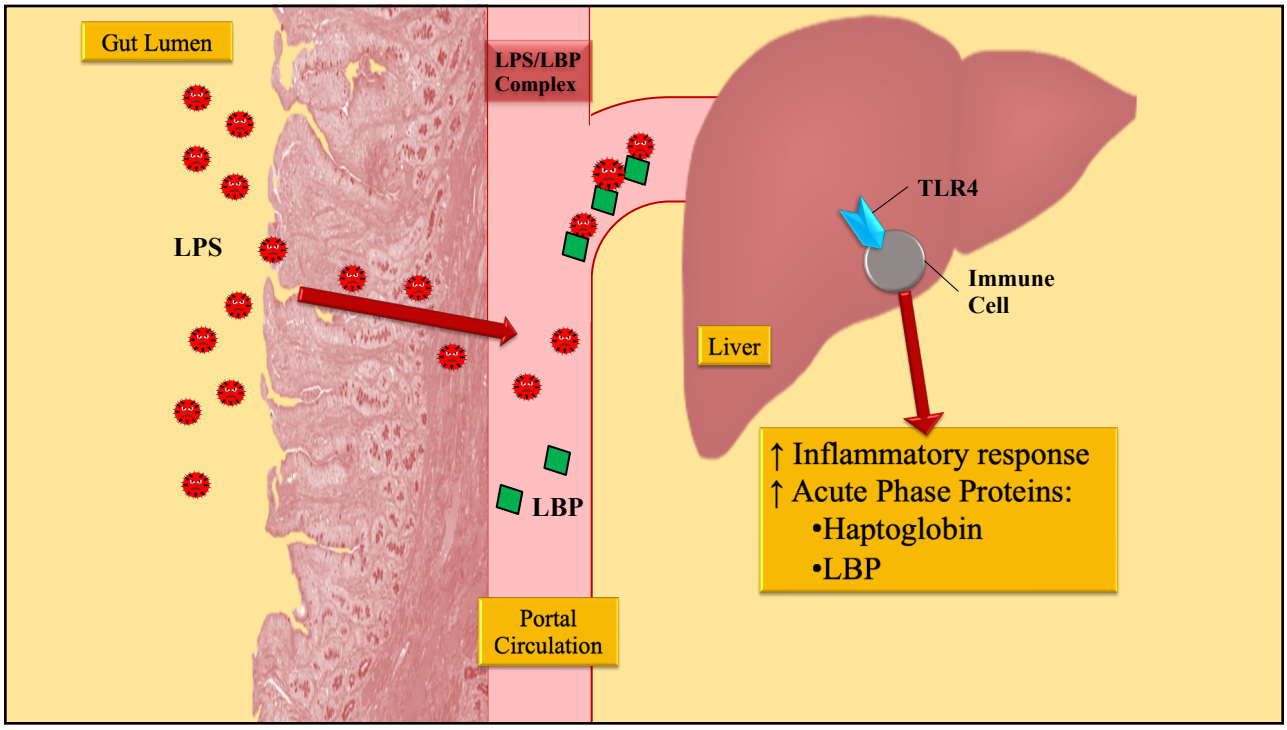

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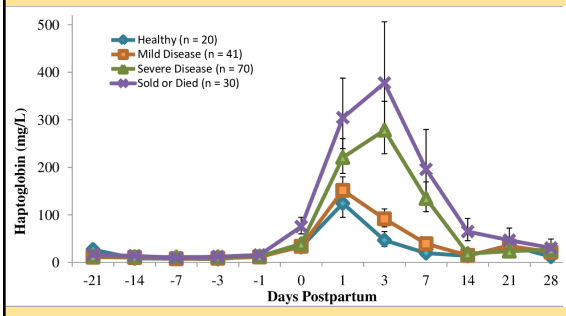
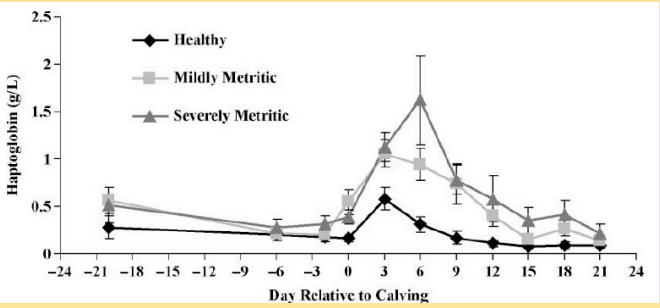
Immune Activation (Haptoglobin) Precedes Clinical Disease



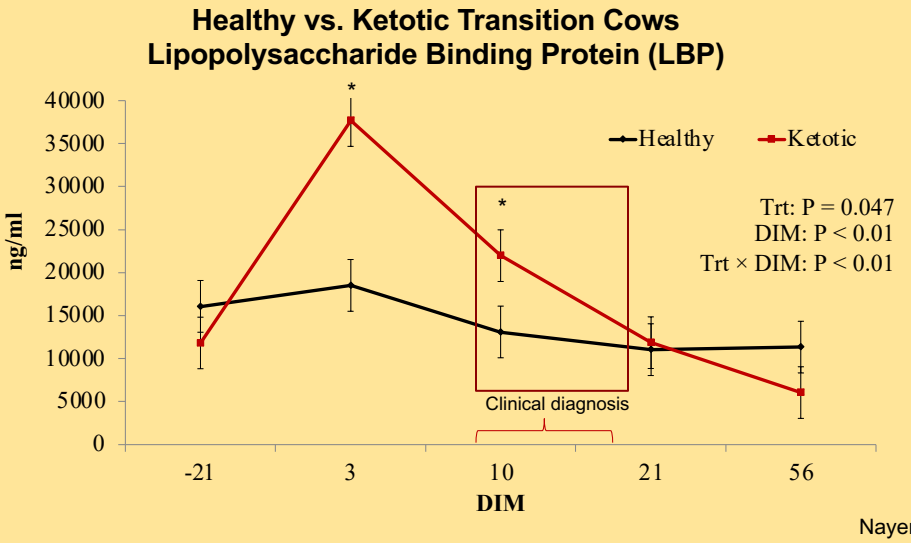
Sebedra 2012



Huzzey et al., 2012

40

Increased LBP in Ketotic Cows



Nayeri et al., 2013;

41

Transition Period Immune Activation

Inflammation's role in:

Suboptimal DMI

42

Immune Activation and Feed Intake

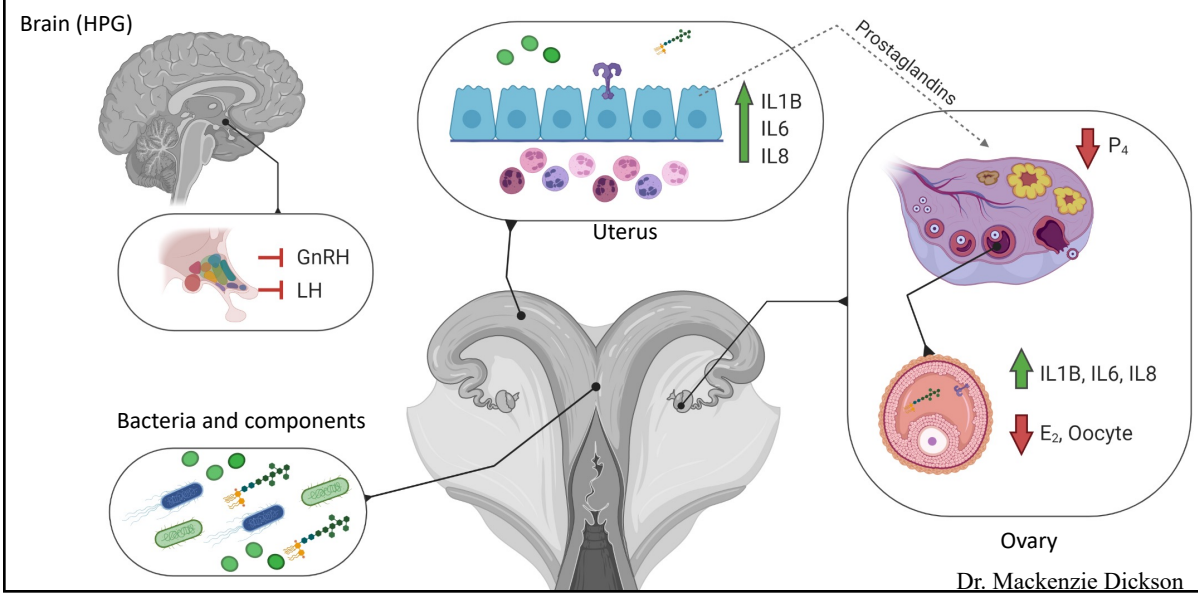
- Inflammatory mediators are potent anorexic compounds Kushibiki et al., 2003
- Reduced feed intake is a highly conserved species response to infection (Aubert et al., 1997; Wang et al., 2016)
- Infection decreases feed consumption, even in insects (Adamo, 2005)
- Cows with increased inflammation have decreased DMI (Trevisi et al., 2002)
  - ….and also increased NEFA and BHB (Trevisi et al., 2010, 2012; Zhou et al., 2016)
  - **Inflammation is the simplest and most logical explanation for why some cows don't eat well before and following calving**

43

Inflammation and Reproduction?

44

LPS Negative Effects Repro From Multiple Angles



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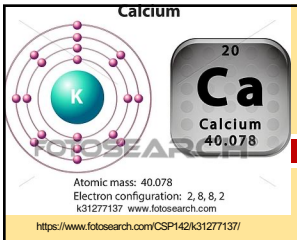


Hypocalcemia Dogma

https://www.farmersjournal.ie/milk-fever-the-problem-of-low-blood-calcium-in-cattle-319488

- Milk uptake of Ca is so quick and extensive that it exceeds the homeostatic capacity to replenish it.
- Academic & Industry Goal: Minimize postcalving hypocalcemia

46

Hypocalcemia

- Clinical hypocalcemia (milk fever) needs a pre-calving dietary strategy
  - The marked reduction in clinical milk fever is arguably the biggest advancement in dairy nutrition in the last 40 years
- Clinical hypocalcemia is pathological
  - It warrants immediate intervention
- Is subclinical hypocalcemia pathological?
  - Is it detrimental to health, productivity and profitability?

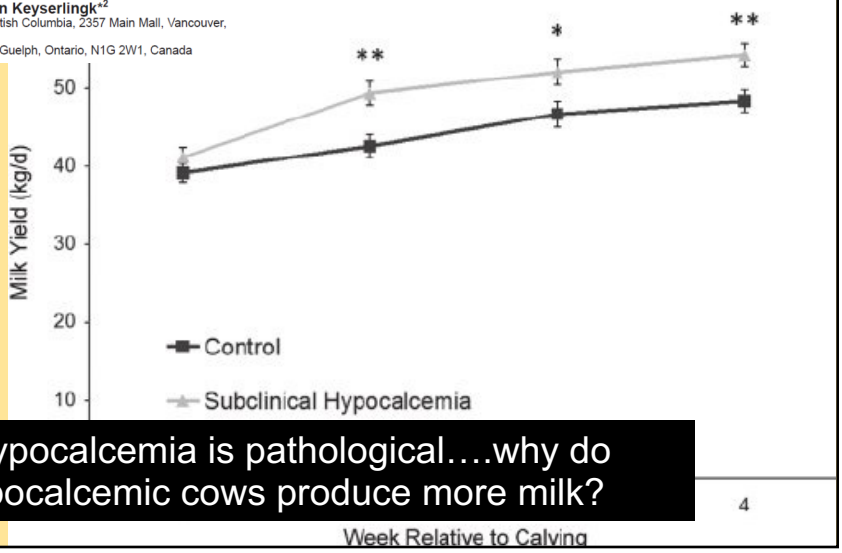
47

J. Dairy Sci. 95:1240–1248
http://dx.doi.org/10.3168/jds.2011-4586

Associations of subclinical hypocalcemia at calving with milk yield, and feeding, drinking, and standing behaviors around parturition in Holstein cows

P. E. Jawor,*[1] J. M. Huzzey,* S. J. LeBlanc,† and M. A. G. von Keyserlingk*[2]
*Animal Welfare Program, Faculty of Land and Food Systems, University of British Columbia, 2357 Main Mall, Vancouver, British Columbia, V6T 1Z4, Canada
†Department of Population Medicine, Ontario Veterinary College, University of Guelph, Ontario, N1G 2W1, Canada

Subclinical hypocalcemia is often associated with increased productivity



If subclinical hypocalcemia is pathological….why do subclinical hypocalcemic cows produce more milk?

48

Immunoactivation was identified as a cause of milk fever more than 130 years ago

MILK FEVER (PARTURIENT PARESIS) IN DAIRY COWS—A REVIEW

J. W. HIBBS

Ohio Agricultural Experiment Station, Wooster

Milk fever (parturient paresis) is an afebrile disease which typically is associated with parturition and beginning lactation. It is characterized by a sudden paralysis, gradual loss of consciousness and, if untreated, usually terminates in death. Few diseases of livestock have caused as much theoretical controversy and interest as has milk fever. Gradually, through the years, much has been learned about the nature of milk fever, and effective means of treatment have been devised, resulting in a reduction in mortality of from 60 to 70 per cent to less than 1 per cent. The basic physiological cause of milk fever has yet to be proven. The "parathyroid deficiency (hypocalcemia) theory" of Dryerre and Greig (54) seems to come the nearest of the many theories that have been advanced to accounting for the immediate cause, but many fundamental questions

THE VETERINARY JOURNAL

AND

Annals of Comparative Pathology.

JANUARY, 1889.

PARTURIENT APOPLEXY IN COWS—A FORM OF SEPTICÆMIA.

BY A. HARRISON THOMAS, M.B., C.M., B.SC., ETC., WHITTINGHAM, PRESTON.

49

J. Dairy Sci. 103:690–701
https://doi.org/10.3168/jds.2019-17191

Association of transient, persistent, or delayed subclinical hypocalcemia with early lactation disease, removal, and milk yield in Holstein cows

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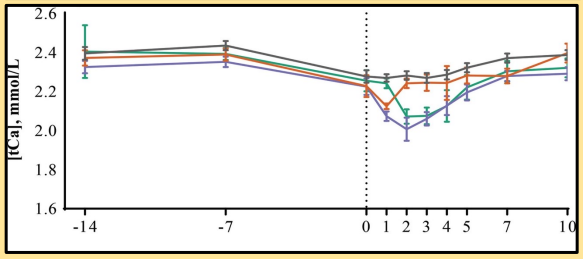

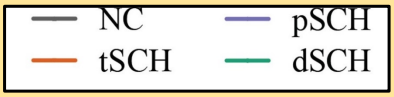

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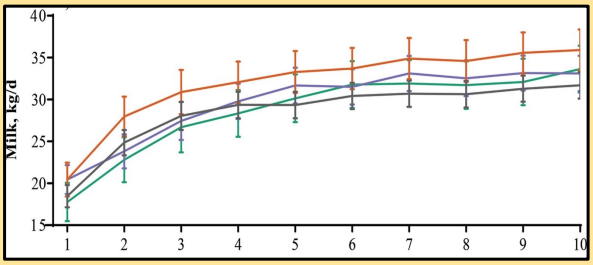
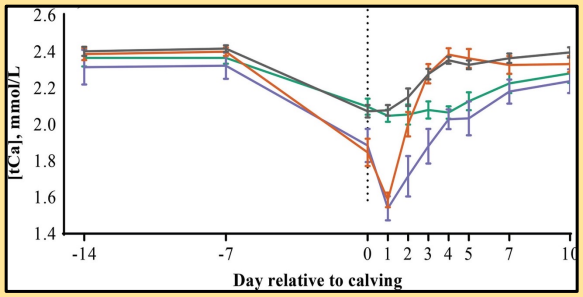
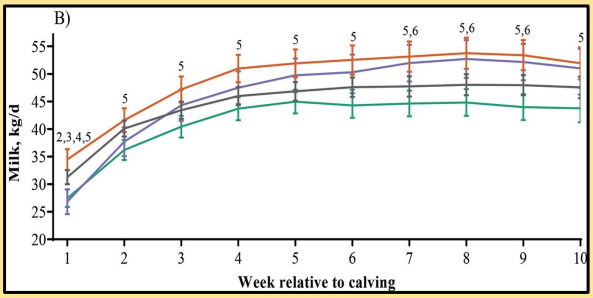




Cows



50

Patterns of Subclinical Hypocalcemia

- Clearly there are different causes for hypocalcemia…..and each cause has different implications.
- What could contribute to chronic and delayed subclinical hypocalcemia?

51

Immune Activation and Hypocalcemia

Research in Veterinary Science 129 (2020) 74–81

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Research in Veterinary Science

journal homepage: www.elsevier.com/locate/rvsc

Effects of an oral supplement containing calcium and live yeast on post-absorptive metabolism, inflammation and production following intravenous lipopolysaccharide infusion in dairy cows

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52

Evolution of the Immunometabolic Field

53

Strange Insulin Status in Multiple Scenarios and Pathologies

- Increased circulating insulin during catabolic conditions:
  - Heat stress
  - Ketosis (especially Type II)
  - Feed restriction ([insulin]/DMI)
  - Intentionally induced leaky gut
  - LPS administration:
    - Mammary
    - Muscle
    - I.V.


J. Dairy Sci. 104:8380–8410
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Invited review: **The influence of immune activation on transition cow health and performance—A critical evaluation of traditional dogmas**

E. A. Horst, S. K. Kvidera, and L. H. Baumgard*
Department of Animal Science, Iowa State University, Ames 50011

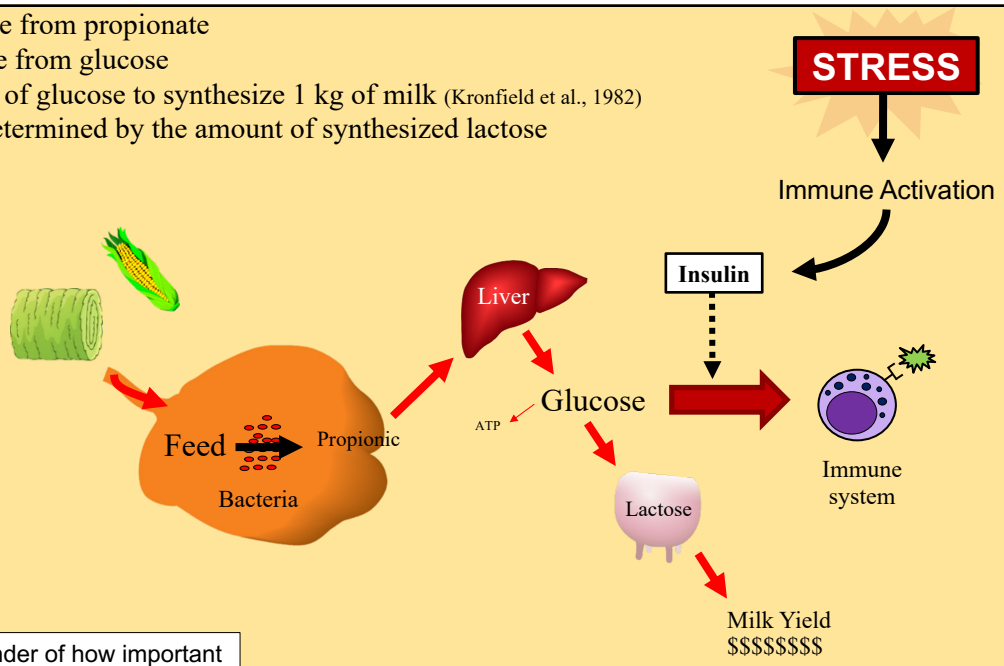
54

Why Increase Insulin??

- Why increase the most acutely anabolic hormone during hypercatabolic conditions?
- Most leukocytes (immune cells) are insulin responsive and need insulin to acquire large amounts of glucose

55

Glucose is made from propionate
Lactose is made from glucose
It requires 72 g of glucose to synthesize 1 kg of milk (Kronfield et al., 1982)
Milk yield is determined by the amount of synthesized lactose



Friendly reminder of how important glucose is to milk synthesis

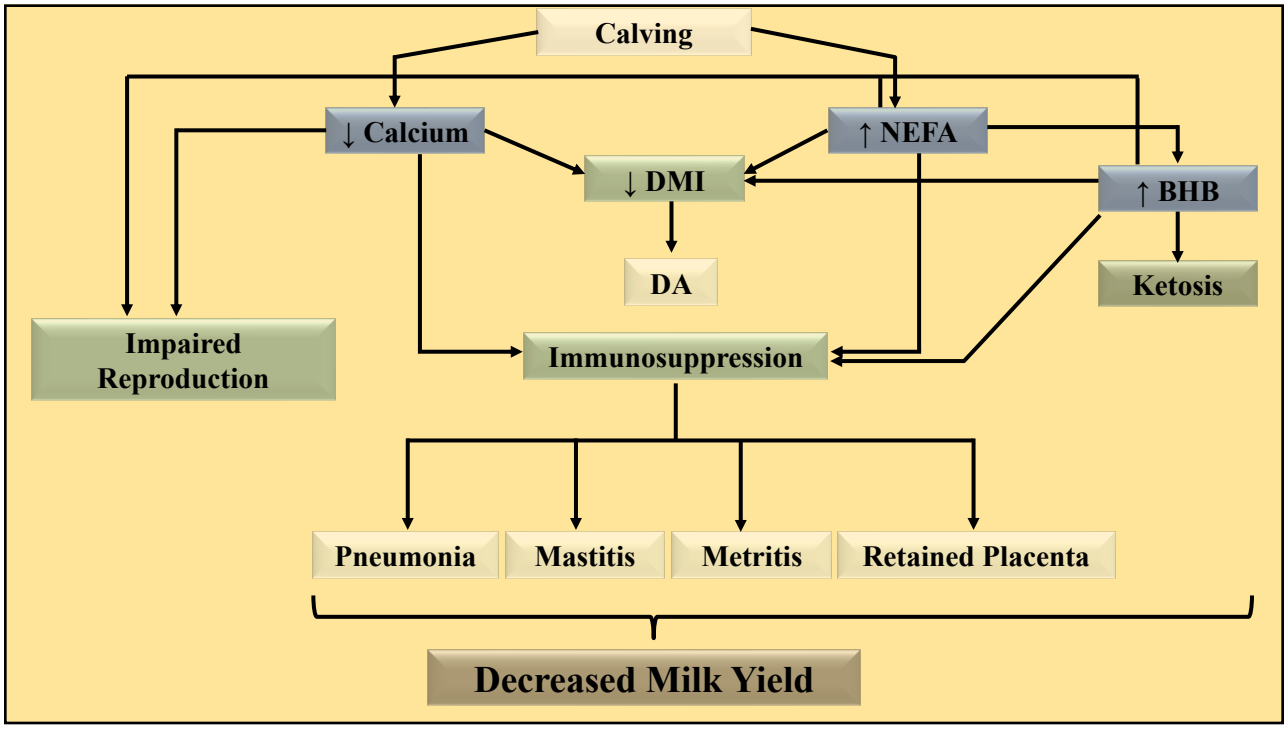
56

Traditional Belief

Increased NEFA, Hyperketonemia, and Hypocalcemia…..**CAUSE** production and health problems

This is not just an ivory tower debate, it has pragmatic and economic consequences

57



58

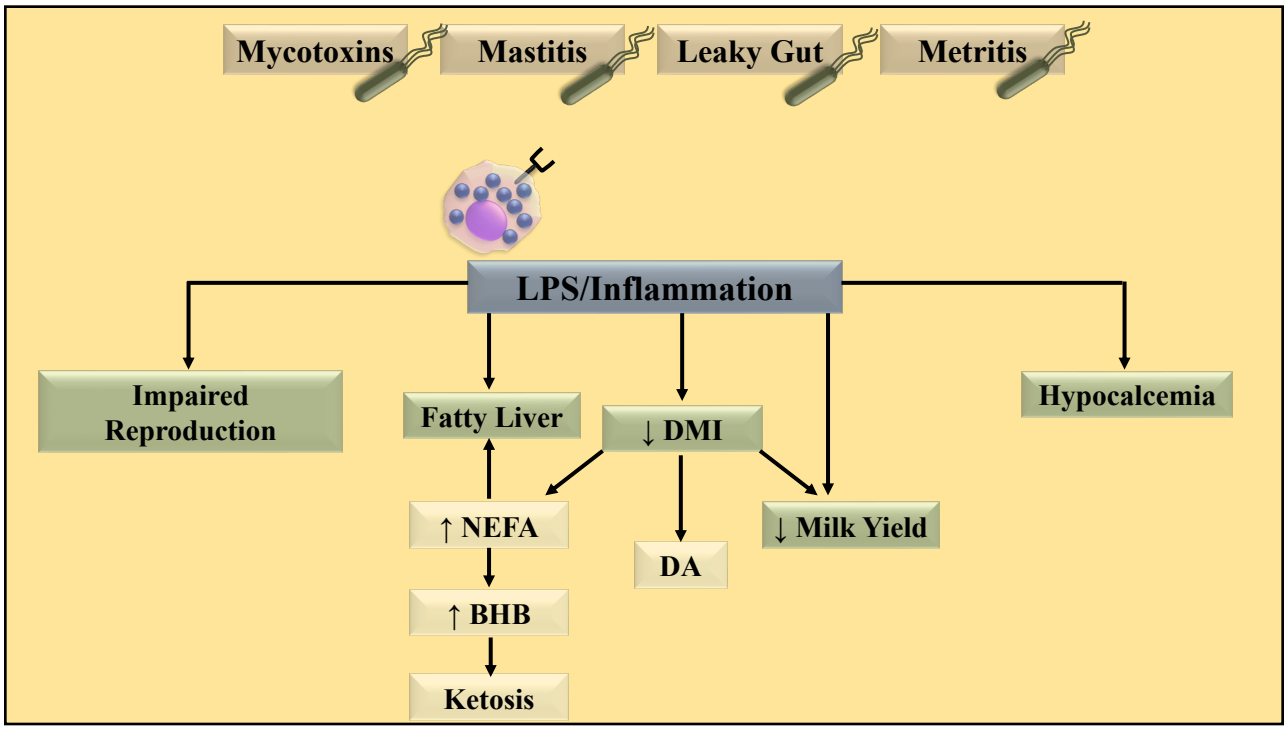
Paradigm Shifting Concept

Increased NEFA and Hyperketonemia are c[illegible]d

Low Feed Intake, high NEFA, Hyperketonemia and hypocalcemia are merely SYMPTOMS….a reflection of prior immune stimulation

Hypocalcemia is a consequence of immune activation

59



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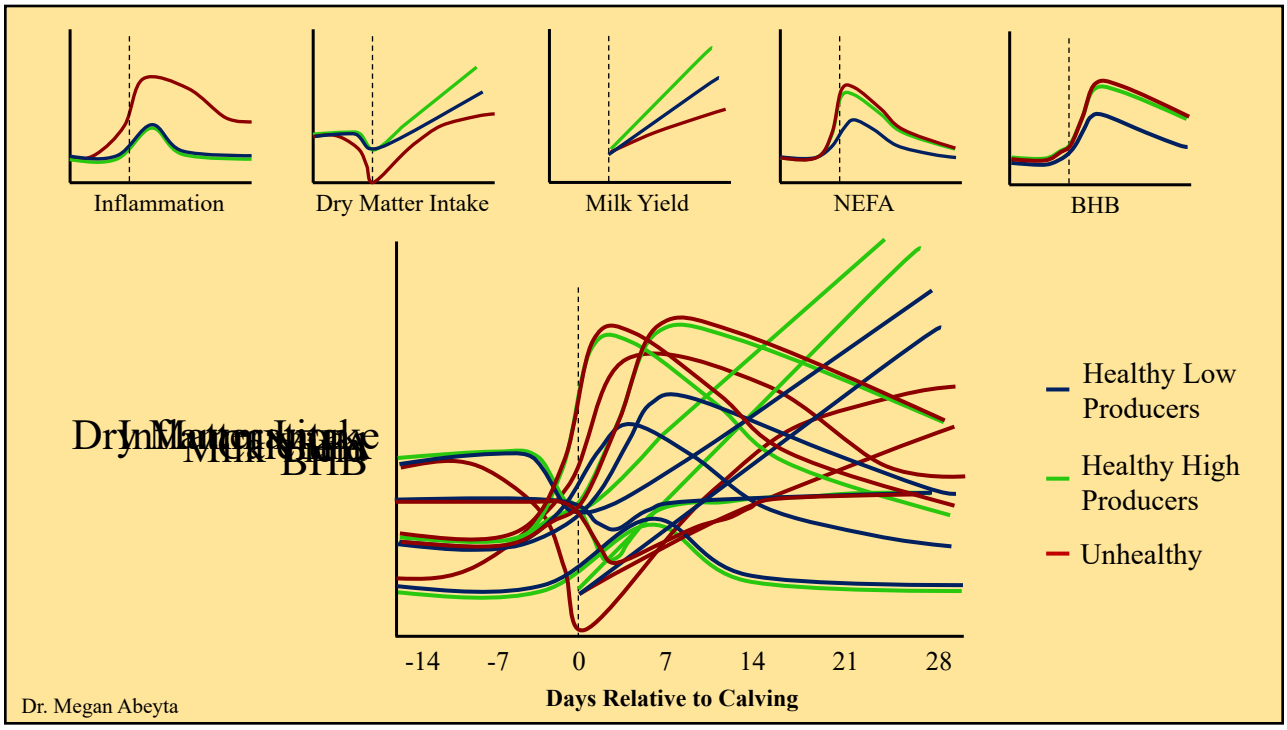
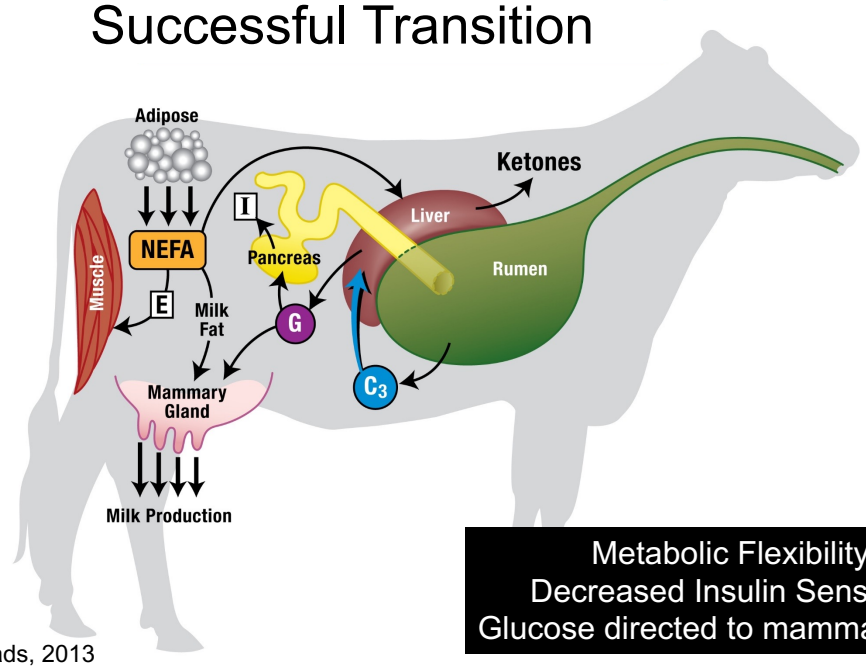


61

Successful Transition

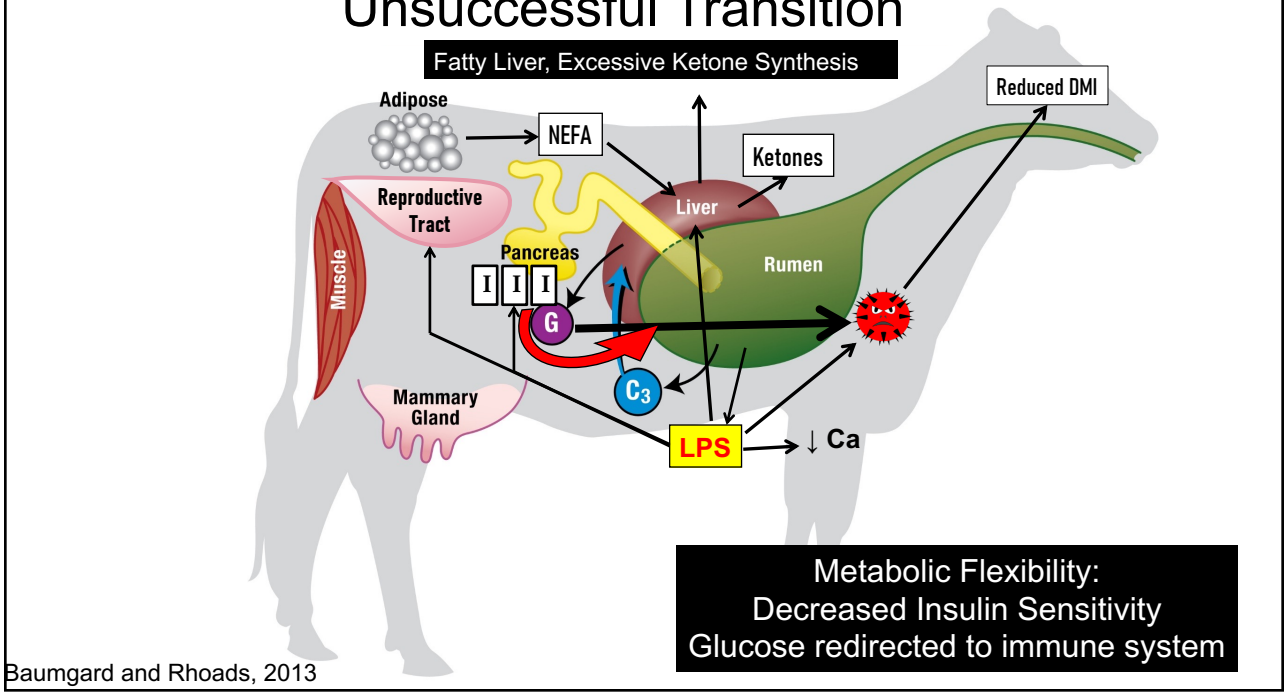


Metabolic Flexibility:
Decreased Insulin Sensitivity
Glucose directed to mammary gland

Baumgard and Rhoads, 2013

62

Unsuccessful Transition



Baumgard and Rhoads, 2013

63

Two Causes for "Ketosis"

- Healthy
- High feed intake
- High NEFA and Ketones
  - Muscle and organs use for energy
- "spares glucose" for milk synthesis

High milk production

- Immune-Activation
- Reduced feed intake
- High NEFA and Ketones
  - Muscle and organs use for energy
- "spares glucose" for immune system

Low milk production

  - Decreased milk synthesis is another strategy to conserve glucose for immune system

64

Ketosis Scenario

- Two cows in the fresh pen
  - 10 DIM
  - Multiparous
- Both are hyperketonemic (i.e. 1.5 mmol/l)

65

Ketosis: When (or if) to intervene?

- Treat:
  - High ketones
  - Not coming into milk
  - Not aggressively eating
  - Looks lethargic and melancholic
  - Has a mild fever

But treating with energy does nothing to address the real problem…..somewhere…..immune-activation is putting the clamp on appetite

- Don't mess with
  - High ketones….but she's eating like a champ
  - Milking like a world-record holder
  - Looks great
  - No fever

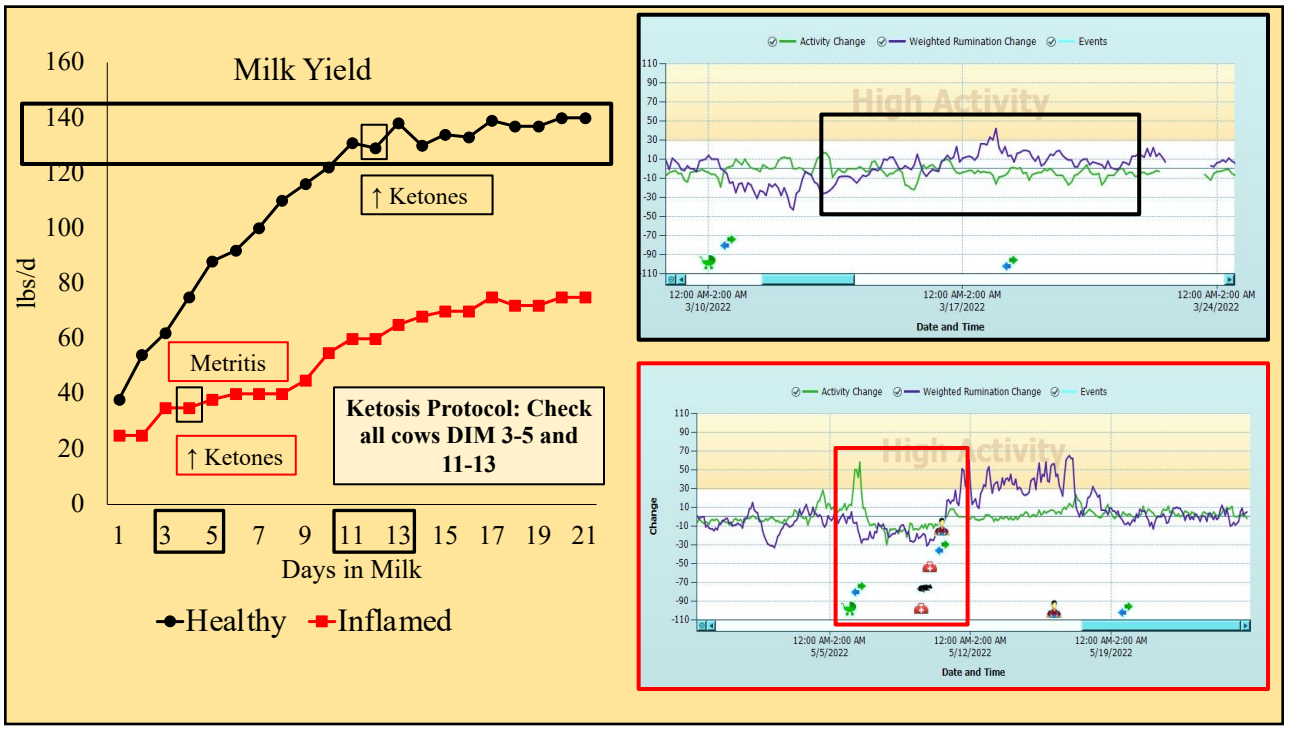
She's the healthiest cow in the herd

66

Real World Example

ISU Dairy Farm
Spring of 2022

67



68

Management Changes?

High production can only occur in the absence of stress and morbidity

- temperature during the transition period?
  - Costs money and time
  - Costs associated with the "treatment"
  - Costs associated with improper treatment administration
    - i.e. Propylene glycol inadvertently down the trachea
  - Opportunity costs for that dedicated labor
- Instead pay more attention to feed intake (rumination) and milk yield

69

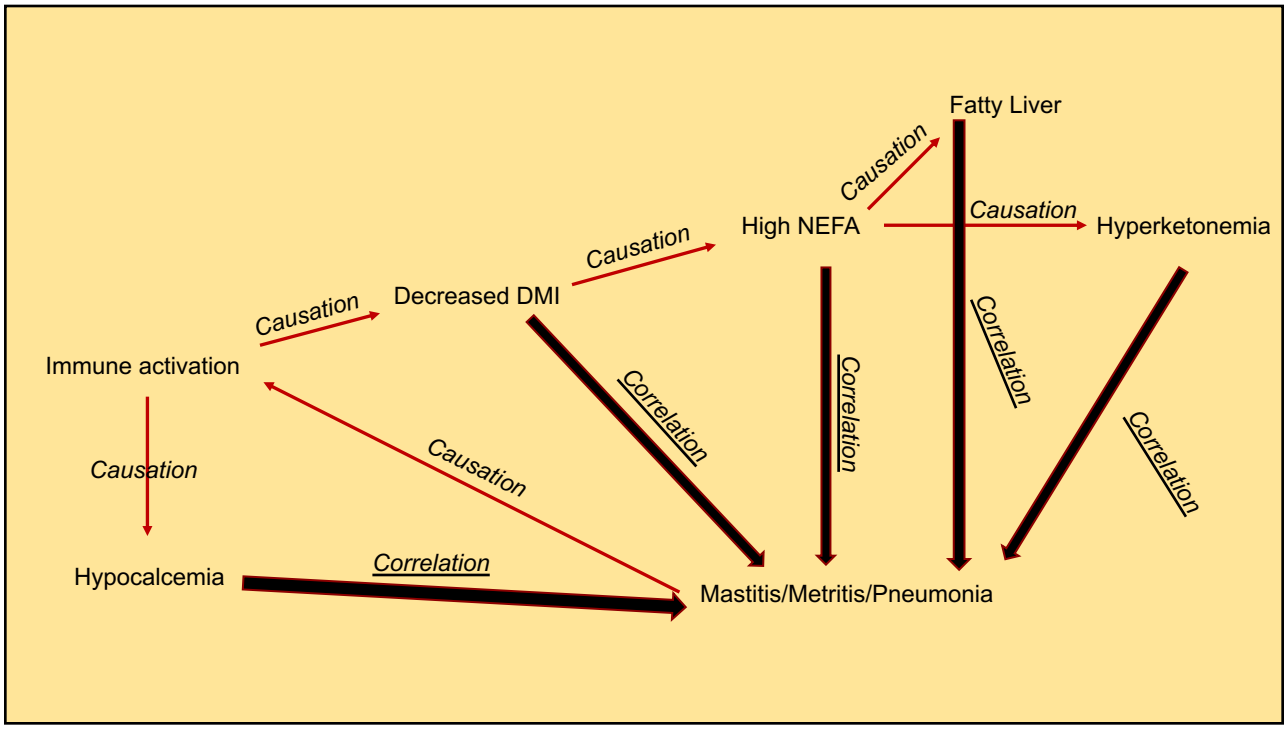
Immune Activation Causes Inflammation

- All transition cows are inflamed (just the magnitude differs)

The immune system "pumps the brake" on feed intake, prevent immune activation and put the "pedal to the metal"!!

- Immune activation causes hypocalcemia
  - It's clearly not the only reason for subclinical hypocalcemia
- LPS causes infertility
- Immune activation reduces feed intake
  - It's the simplest and most logical explanation for why some cows don't eat well following calving

70



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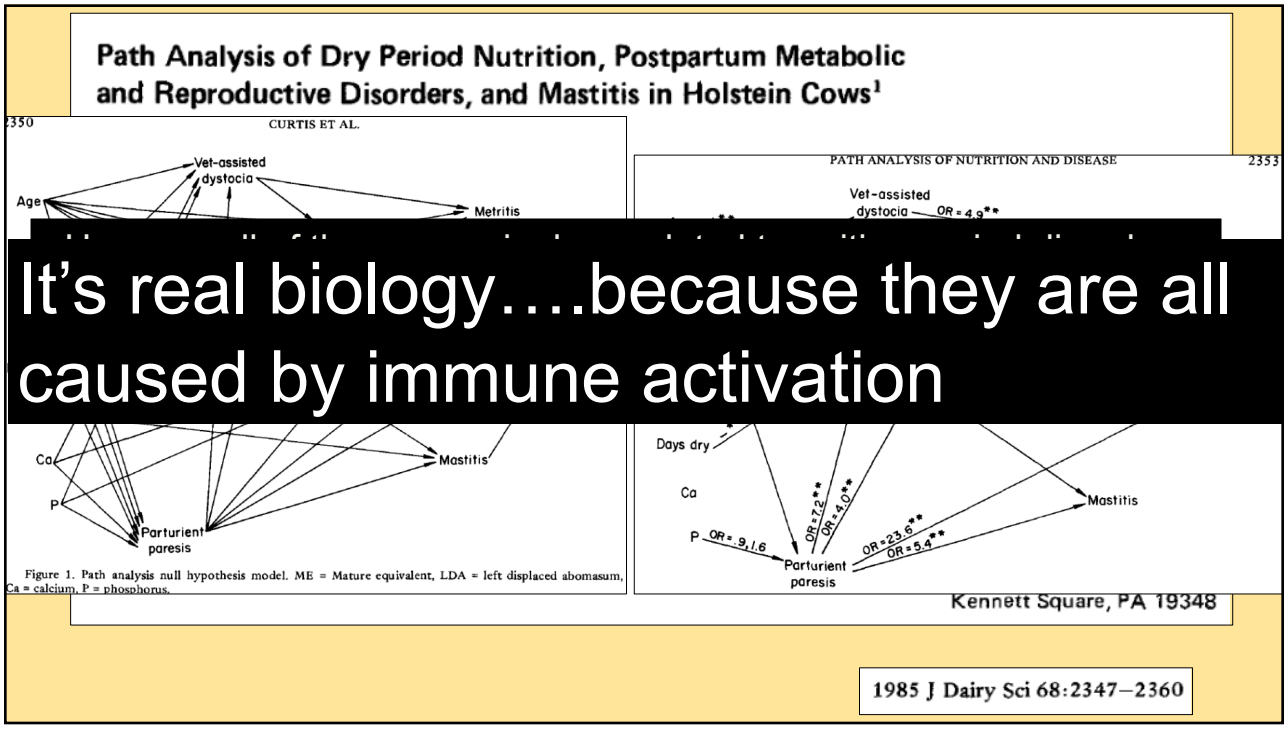


It's real biology….because they are all caused by immune activation

72

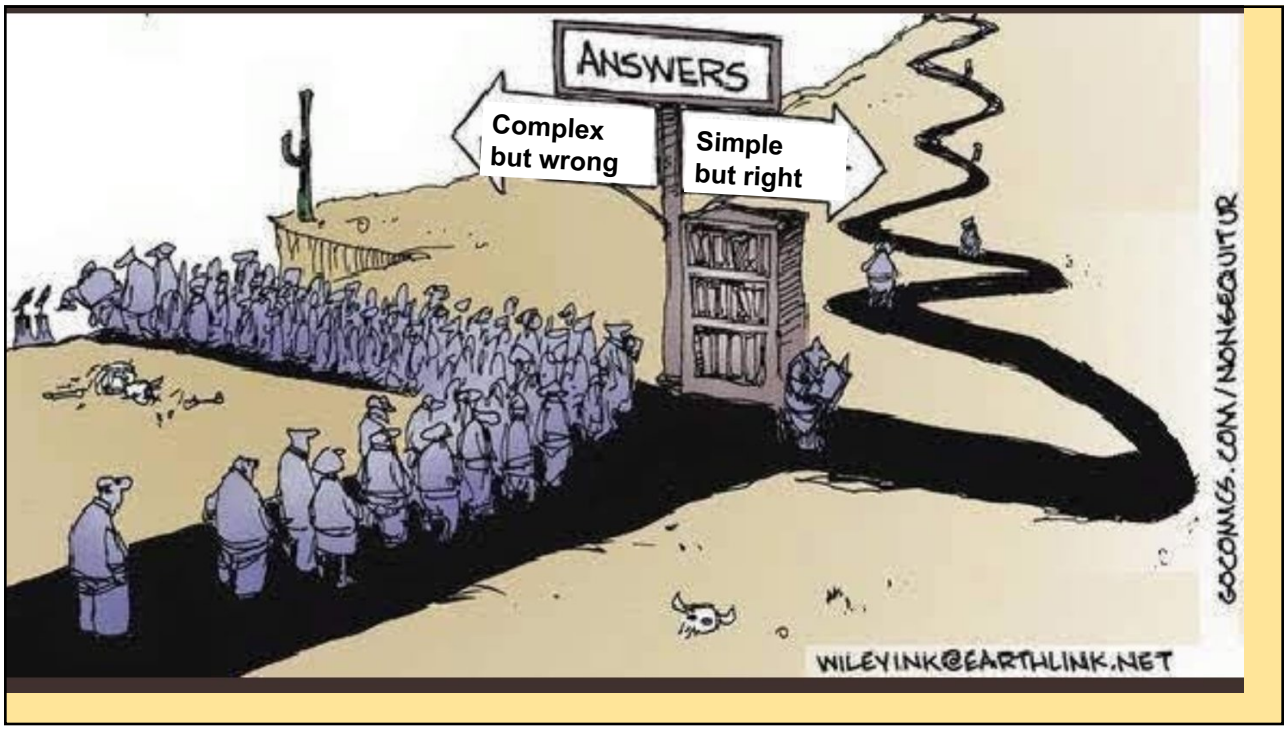
Summary: Inflammation and the Transition Period

- These metabolic and mineral changes are not "dysfunctional"
- They are required to prioritize survival or required for maximum productivity
  - They aren't to blame (they're not the cause) for poor productivity

Profitable Production is a Consequence of Wellness

  - Minimize stress (overcrowding of prefresh and fresh pens, on time feed delivery, etc.)
- Hygiene
  - Minimize environmental pathogens (dry off procedure, dry environment, etc.)
  - Pen cleanliness
- Dietary strategies
  - Feed hygiene
  - Prevent GIT disturbances
  - Target molecules aimed and minimizing leaky gut
  - Immune modulation

73



74

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USDA NIFA United States Department of Agriculture National Institute of Food and Agriculture

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75

76

What are Producers, Nutritionists and Veterinarians to do?

- Need to identify the source of infection/subclinical infection
  - Can't just show up and quickly treat subclinical hypocalcemia and hyperketonemia and hurry to next client
  - Need thorough evaluation…requires time
- Train farm personnel to utilize full array of information
  - Feed intake and production
  - Cow appearance

77