



Unit : Ketosis

Lesson : 3

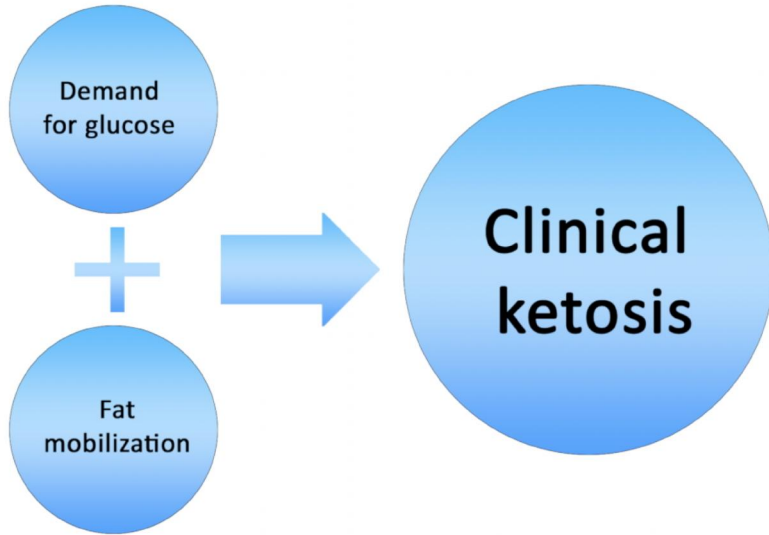
Pathogenesis of Bovine and Ovine Ketosis

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Pathophysiology



VFAs

Acetate (70%)

- Mainly used in fat synthesis
- Ketogenic

Butyrate (20%)

- Ketogenic condensed into acetocetyl CoA
- ↓
- Oxidized to ketone bodies
- ↓
- Transformed into acetyl CoA

Propionate (10%)

- Glycogenic
- Enters the TCA directly at the level of succinyl CoA

VFAs

Oxidation of acetyl CoA depends on an adequate supply of oxaloacetate

- Oxaloacetate is generated from gluconeogenic precursor propionate
- Oxaloacetate are reduced slowing down the TCA cycle
- Acetyl CoA is diverted to the formation of ketone bodies

VFAs

To increase gluconeogenesis and offset the negative energy balance, more triglycerides are mobilized from body fat stores



Triglycerides are hydrolyzed to release FFA/NEFAs



In liver TG are re-synthesized from the released FFAs/NEFAs and stored by the liver or exported as very low density lipoprotein

VFAs

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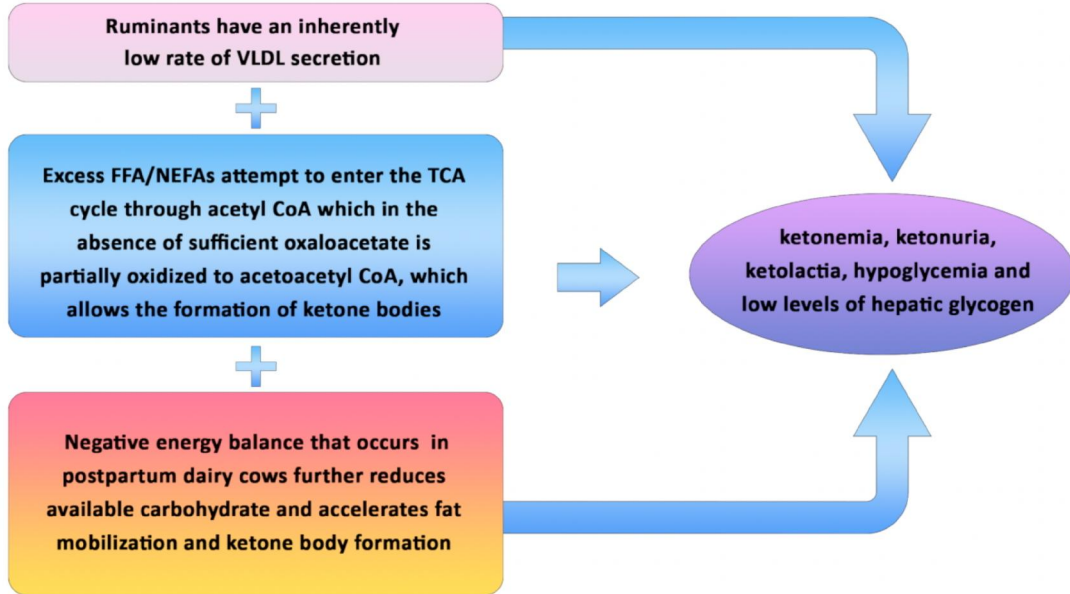


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VFAs





Ovine Ketosis

In adequate energy intake in late pregnancy



Ineffective gluconeogenesis



Hypoglycemia, lipid mobilization



Accumulation of ketone bodies and Cortisol



Bovine Ketosis

- **Principle metabolic disturbances are hypoglycemia and ketonemia**
- **The nervous signs occur in bovine ketosis is due to the production of isopropyl alcohol which is a breakdown product of acetoacetic acid in the rumen hypoglycemia**

Ovine Ketosis

- **Ovine ketosis Encephalopathy is due to hypoglycemia in the early stages of the disease**
- **Encephalopathy and the disease are frequently not reversible unless treated in the early stages**
- **High level of cortisol and adrenal steroid diabetes contributes to the pathogenesis**



Thank you