## Carbohydrates digestive in non-ruminant

-begins with **salivary amylase** in the mouth  (specific to non-ruminants and monogastrics)
-continues in small intestine with **pancreatic amylase**
-finished by intestinal **maltase, sucrase and lactase**

## Carbohydrates digestive in ruminants

-saliva does not contain **amylase**
-bacterial cellulase and hemicellulase are capable of breaking **beta 1,4 bonds** between CHO of cellulose and hemicellulose
-Limited starch digestion in the **small intestine**

Diet higher in fiber increases **acetate** production

Which VFA is attributed to marveling : **Propionate**

**Increase** propionate to Increase **carbs** to increase **starch** = leads to **marveling**

To increase energy you increase **acetate** → increases **fiber** → make you feel **full** and increases **energy**

## END PRODUCTS OF CARBOHYDRATE DIGESTION

Non-ruminants : **monosaccharides**, mainly glucose
Ruminants: VFAs: **acetate, propionate, butyrate** + MONOSACCHARIDES

## Carbohydrate Gelatinization

Add **water** and **heat** makes **CHO** swell and take up water
Structure changes from **crystalline** to **Amorphous** to become more **available** and **digestible** and can help increase **feed efficiency**

Improves starch digestibility for rumen bacteria by **5% to 8%**

Steam flaking: adding **water** and **heat** in corn

## CARBOHYDRATE ABSORPTION

- Glucose absorbed via **active transport**
- Once absorbed enters the **portal vein** and sent to the **liver**
- Stored as **glycogen** or used as **fuel**
- Insulin regulates uptake **beta** cells of **pancreas**

- Glucagon regulates breakdown **alpha** cells of **pancreas**

## METABOLIC DISORDERS OF CARBOHYDRATE METABOLISM

Diabetes - **high blood sugar concentrations**

- Insulin produced by **pancreas**
- Type 1 - **low insulin production** (juvenile diabetes )
- Type 2 - **non insulin dependent diabetes** (adult onset)
    - **Normal** insulin production
    - Decreased **receptors** and reduced insulin **sensitivity**

Gestational diabetes : **increased tissue resistance to insulin action**

## NUTRITION MANAGEMENT OF DIABETIC DOGS

- Consistent feeding plan and food that minimizes post prandial changes in **blood glucose levels**
- Single most effective dietary changes is to include either insoluble or soluble **fiber** at 8 to 18% DM basis
- **Weight** loss program
- **Insulin** injection when > 75% of **beta** cells are destroyed

## Metabolic disorders- KETOSIS

- In **lactating** dairy cattle and sheep in late **pregnancy**
- In early lactation milk production **increases** rapidly
- Cow is in **negative** energy balance - can't eat enough to meet energy demands for milk production ( heavy glucose drain for lactose synthesis
- Begins to mobilize body tissues for **energy** (protein and fat)

KETOSIS:

- Because of high milk production, gluconeogenesis is vital for **lactose** synthesis
- Acetyl Co A from body fat mobilization cannot enter **krebs** cycle because of inadequate **oxaloacetate** concentrations
- Acetyl co a converted into **ketones**

Result of ketosis

- Excess of **ketones** (acetone, acetoacetone, beta-hydroxybutyrate) accumulate in **blood** and **tissues**
- Cow goes off feed and milk production **decreases** dramatically
- Cows with advanced ketosis or those that die from ketosis have **fatty livers**
- **Fatty liver syndrome** (7-30% fat in liver on wet basis)

Fat replaces functioning **tissue** of liver

KETOSIS PREVENTION/TREATMENT

Maximize **lactation** feed intake

- Don't **overfeed** during gestation
- Keep cows in **good** condition not excessive
- Supplement **niacin**
- **Propylene glycol** common treatment compound