Fats

- Functions of fats in foods
    - Energy dense foods
    - Taste/flavor
    - Tenderness of meats
    - Flakiness of baked goods
    - Satiety (Feeling of satisfaction after meals)
        - Decreased gastric emptying
    - Transportation of fat-soluble vitamins
        - A, D, E, and K
    - Provides Essential fatty acids
        - Linoleic and alpha-linolenic acid
- Functions of fats in the body
    - Major energy storage
    - Shock absorbers/Insulator
    - Major Component of Cell Membrane
    - Transport of proteins in the blood
    - Precursor/Regulator for bodily processes
        - Fat is used to make to make substances (hormones) that help regulate many body processes, such as blood pressure and blood clotting
        - Essential fats can be made into eicosanoids
- Essential Fats
    - Linoleic acid (omega-6)
        - Sources: Veggie oils and nuts
        - Used to make eicosanoids for blood clotting and vasoconstriction
    - Alpha-linolenic acid (omega-3)
        - Sources: Walnuts, Soy, Canola oils, and fish oils
        - Used to make eicosanoids to reduce blood clotting and vasoconstriction
- Types of lipids
    - Fatty acids
    - Triacylglycerols/Triglycerides

- Diglycerides
- Monoglycerides
- Phospholipids
- Sphingolipids
- Sterols

- Fatty acids – basic structure
  - Simple Lipid
  - Straight hydrocarbon chain with a methyl group at one end and a carboxylic acid group at the other
  - Most common fatty acids have 18 carbons
  - Saturated, Monounsaturated or Polyunsaturated
  - Most naturally occurring fatty acids have cis configuration at double bonds
    - # of carbons in a chain
      - Short chain: <6 carbons = fermentation in the colon
      - Medium chain 6-12 carbons
      - Long chain > 12 carbons
    - If double bonds are present
    - Where double bond exists
      - Fatty acid nomenclature
        - 18: 2 Delta 9,12
        - Or 18: 2 n-6
- Triglycerides – basic structure, the major form of fat in food and in the body
  - The major form of fat in foods
  - The storage form of fat in the body
  - Structure: Trihydroxy alcohol (glycerol) is attached by ester bonds to 3 fatty acids
- Phospholipids – basic structure, major functions
  - Glycerol backbone
  - 2 fatty acids
  - Phosphatidic acid
  - In the cell membrane for phospholipid bilayer
- Sphingolipids – basic structure, function of sphingomyelin

- Looks similar to phospholipids
- Instead of glycerol, they have a sphingosine backbone
- Ex.) sphingomyelin and fxn component in cell membrane structure and abundant in myelin sheath
- Myelin Sheath surrounding neurons

- Sterols/steroids – basic structure of cholesterol, cholesterol is precursor for?
  - Four-ring steroid nucleus and at least one hydroxyl group
  - Benzene ring
  - Cholesterol
    - Most common sterol in humans
    - Component of cell membrane
    - Precursor for steroids; sex hormones, bile acids, adrenocortical hormones. Vitamin D
    - Bile acids act as detergents in small intestine to emulsify dietary lipids for digestion and absorption
    - Adrenocortical hormones aka cortisol aldosterone
- Recommended intakes
  - 20-35% of calories should come from fat
  - Minimize trans fatty acid intake
  - Unsaturated fatty acids should be primary source of dietary fat
- Digestion of fats
  - Where in the GI tract? Mouth = lingual lipase, some digestion in stomach, most in small intestine
  - Triglycerides in Small Intestine
    - Bile helps emulsify (mix) fat and water
  - Purpose of pancreatic lipase and bile
    - Pancreatic lipase results in the partial hydrolysis of triglycerides
  - final products: small enough to be absorbed
    - Free fatty acids
    - Glycerol
    - Monoglycerides

        - Some Diglycerides
    - Digestion of cholesterol esterase
        - Cholesterol esterase = enzyme for cholesterol
        - Cholesterol only comes from animals or our body makes it
        - Cholesterol esters -> free CHOL + fatty acid
    - Phospholipids
        - 1 glycerol backbone, 2 fatty acids, Phosphatidic acid
        - Phospholipase
        - Removes fatty acid from phospholipid
        - Phospholipid -> lysophospholipid
- What are micelles?
    - Needed to emulsify fat and formed into chylomicrons
    - After digestion, fat products associate with bile salts to form micelles
    - Micelles interact at brush border & lipid contents diffuse into intestinal cells (enterocytes)
- Fat absorption into intestinal cells
    - Occurs mostly in the duodenum and jejunum of small intestine
    - Absorbs up to 98% efficiency
    - Excessive fat in the stool is: steatorrhea
    - Absorbs
        - Glycerol
        - Monoglycerides
        - Fatty acids
    - Monoglycerides & long chain fatty acids are resynthesized into triglycerides
    - Phospholipids are resynthesized
    - 70-80% of cholesterol is esterified (Chol bound to fatty acid)
- What happens to bile in the small intestine after it completes its job as an emulsifier?
    - 95% is reabsorbed in the ileum & reused through the enterohepatic pathway
    - Only 5% is excreted in the feces
- Absorption of lipids into lymph vessels and blood
    - Glycerol & short/medium-chain fatty acids are absorbed into the portal vein

- Where they bind with albumin (protein) and are transported directly to the liver
- Nutrients head off to the liver first

- Lipid Transportation in the Blood
  - Know parts of a lipoprotein: protein, chol, triglycerides, phospholipids
  - Lipoproteins are transport vehicles for lipids and they act as emulsifiers (fat droplets)
    - Protein
    - Cholesterol
    - Triglycerides
    - Phospholipids
  - What are apolipoproteins?
    - Specific proteins on ALL lipoproteins
    - Serve as enzyme activators for cell receptors
    - apoA
    - apoB
    - apoC
    - apoD
    - apoE
    - Know the function of apoB100 apolipoprotein
      - LDL Cholesterol
  - **4 lipoproteins**
    - Chylomicrons
      - Carry products (mostly TG) from digestion/absorption of lipids
      - Chylomicron remnants are sent to the liver
      - Most TGs in chylomicrons go to muscle & adipose tissue, where they are used for energy or stored
    - VLDL
      - Synthesized in liver
      - Contains TGs, Cholesterol, and Phospholipids
      - TGs are removed from VLDL in muscle and adipose (like chylomicrons)

- LDL
    - Synthesized in liver
    - Made from VLDL (after removing TGs)
    - Contain less TG and more CHOL vs VLDL
    - Major transporter of CHOl in the blood
    - Predictive of heart disease risk -- atherogenic
- HDL
    - Synthesized in liver & intestinal cells
    - Transports cholesterol from peripheral tissues to liver (CHOL can be used in bile acid synthesis)
    - Compared to LDL, has way more protein
    - Bigger than LDL
- Where are they made?
- A major transporter of which fat?
- where are they going?

- Moving lipids from the blood to cells (LPL – where do we find it and its function)
    - Lipoprotein lipase (LPL)
        - *Enzyme found in endothelial cells lining capillaries*
        - Widely distributed in adipose, heart, and skeletal muscle tissue
        - Functions to hydrolyze (break down) triglycerides in chylomicrons and VLDL (thus removing fats from blood).
        - Estrogen increases the activity of LPL in gluteofemoral adipocytes
- Role of liver and adipose tissue in lipid metabolism
    - Liver Synthesizes
        - Bile acids
        - Lipoproteins
        - New lipids from non-lipid precursors
        - During fasting, the liver produces ketone bodies and continues to synthesize VLDL & HDL
    - Adipose Tissue

        - Takes triglycerides & cholesterol from chylomicrons and VLDL (lipoprotein lipase is required)
        - Stores TG
        - During fasting, lipolysis occurs, releasing fatty acids into blood (hormone-sensitive lipase required)
- Process of atherosclerosis and how is endothelium injured p.21
    - Atherosclerosis is the hardening of arteries caused by lipid-rich plaques
        - Injury to endothelium
        - Macrophages (immune cells) react
        - Smooth muscle cells of arteries replicate
        - Forms plaques/blood clots
    - Inflammation increases the adherence of macrophages and platelets
        - Engulf LDL Chol
    - Injury to the endothelium can be caused by:
        - Oxidized LDL, saturated fats and trans fats
        - Inflammation
        - Triglycerides and high blood pressure
        - Smoking
        - Glycated proteins
- **Cholesterol** (sources, major component of atherogenic fatty plaques, and how is total blood CHOL measured)
    - CHOL is a major component of atherogenic fatty plaques
    - 2 sources of body cholesterol
        - Diet (animal cholesterol)
        - Liver
    - The latest evidence: dietary chol has little or no impact on serum cholesterol
    - Total Blood Cholesterol = LDL + HDL + VLDL (trace amounts)
    - Want to:
        - Raise HDL (good cholesterol)
        - Lower LDL (bad cholesterol)
    - Ratio of Total CHOL: HDL-CHOL is best predictor of CVD deaths

- Lipoproteins and CVD risk (including how to decrease LDL and increase HDL)
    - To increase HDL
        - Exercise
        - Weight Loss
        - Moderation of Alcohol to 1 drink or less
        - Soy Protein
        - MUFA
        - Estrogen
    - To decrease LDL
        - Estrogen
        - MUFA + PUFA
        - Soluble fiber
        - Soy Protein
        - Weight loss/exercise
        - Dietary sterols/stanols
- Recommended lipid levels in the blood
    - TCHOL = less than 170
    - LDL = <100 mg/dl
    - CHOL = <200 mg/dl
    - HDL = >60 mg/dl
    - Blood TG less than 150 mg/dl
- Saturated fats
    - Solid at room temperature
    - Animal fats contain 40-60% saturated fats
    - Plant oils contain 10-20% saturated fats
    - Increases risk of CVD
- Unsaturated fats
    - Liquid at room temperature
    - Animal fats contain 30-50% monounsaturated
    - Plant oils contain 80-90% unsaturated
    - Decreases risk of CVD

- Benefits of omega 3 fats (n-3 fatty acids)
    - Interfere with platelet aggregation (which is a good thing!)
    - Reduce the release of cytokines
    - Reduce triglyceride concentration
- Trans fatty acids
    - Sources
        - Hydrogenation of unsaturated fats
        - Small amounts found naturally in beef and dairy
    - Characteristics
        - Raises LDL and lowers HDL
        - Behaves like saturated fat
        - Associated with increased inflammation
        - Correlates more strongly with CVD mortality than saturated fatty acids
- Unique lipoproteins which may increase the risk of CVD/atherosclerosis
    - Lipoprotein A [Lp(a)]
        - Genetic variant of LDL
            - Attached to a unique marker protein referred to as apo(a)
        - Associated with atherosclerosis
        - May deliver CHOL to regions of recent injury and for wound healing
    - Apolipoprotein E
        - apoE is a structural component of HDL, VLDL, and chylomicrons
        - ApoE may be involved in atherogenesis
        - 3 isoforms: apoE2, E3, and E4
        - E4 phenotype associated with increased CVD risk
- Lipid pathways
    - Eicosanoids
    - Ketones
    - Lipogenesis of fatty acids
    - Cholesterol synthesis
    - Beta Oxidation of fatty acids
- Eicosanoids

    - Compounds made from the essential fats (linoleic and alpha-linolenic acids)
    - They have hormone-like properties
    - Include:
        - Prostaglandins (PG)
        - Leukotrienes (LT)
        - Thromboxanes (TX)
- Overall effects of making more n-6 derived eicosanoids, overall effects of making more n-3 derived eicosanoids
    - More n-6 and n-3 go on to make eicosanoids
    - Fatty acids make specific eicosanoids
        - Linoleic acid -> arachidonic acid -> eicosanoids
            - Proinflammatory
            - Pro-arrhythmia
            - Platelet Activator
            - Vasoconstrictor
        - Linolenic acid -> eicosapentaenoic acid -> eicosanoids
            - Anti-inflammatory
            - Anti-arrhythmia
            - Platelet inhibitor
            - Vasodilation
- What are the essential fatty acids and food sources of omega 3 fats and omega 6 fats?
    - Omega-6 sources: corn oil, soybean oil, sunflower oil, nuts and seeds
    - Omega-3 sources: fatty fish, walnuts and flaxseed
- Essential fatty acid deficiency
    - N-6 Deficiency
        - Poor growth
        - Scaly Lesions
    - N-3 Deficiency
        - Neurological Abnormalities
        - Visual Abnormalities
- Ketones

- Substances that are made when the body breaks down fat for energy
    - What are they made of: Acetyl CoA
    - When are ketones made: When the body breaks down fat for energy when it lacks carbs
    - What are the three ketone bodies
        - Ketogenesis: Liver converts excess acetyl CoA to ketone body
        - Ketosis: Mild increase in ketone bodies
        - Ketoacidosis: Dangerously high levels of ketone bodies

- Lipogenesis
    - All fatty acids (except for the essential fats) are made from acetyl CoA
    - Except for n-3 and n-6 PUFA's all fats can be synthesized from acetyl CoA
- Sources of Acetyl CoA
    - Pyruvate
    - Carbohydrates
    - Amino Acids
    - Fatty acids
- Facts to know about fatty acid synthesis
    - Remember the Pentose phosphate pathway (hexose monophosphate shunt)
    - Main purposes:
        - Produce ribose-5-P, which is necessary for synthesis of nucleic acids found in DNA and RNA and other nucleotides
        - Produce NADPH (nicotinamide adenine dinucleotide phosphate), which is required for the synthesis of fatty acids
    - Starts with acetyl CoA
    - fatty acids are elongated by adding more acetyl CoA
    - NADPH is required to make fatty acids
- How do we use fatty acids once they are eaten or made?
    - Stored as energy (triglycerides and adipose tissue)
    - Used to make phospholipids (bilayer in every cell)
    - Used to make cholesterol esters (that bond with fatty acids)
    - Used to make eicosanoids

- Cholesterol synthesis
    - Made from acetyl CoA
    - HMG-CoA reductase is the rate-limiting step of cholesterol synthesis and step is irreversible
    - The statin drugs inhibit HMG-CoA reductase = enzyme, thus decreasing CHOL synthesis
        - Statins
            - Atorvastatin (Lipitor)
            - Simvastatin (Zocor)
            - Lovastatin (Mevacor)
            - Pravastatin (Pravachol)
            - Rosuvastatin (Crestor)
- Beta oxidation
        - What is it: Breakdown for energy of fatty acids
        - Where does it occur: within the mitochondria of cells
        - One cycle produces:
            - 1 Acetyl CoA
            - 1 FADH2 electron carrier
            - 1 NADH electron carrier
    - How many ATPs can we produce from one fat?
        - A 16 Carbon Fatty Acids can produce 129 ATPs
        - 1 glucose molecule produces 36-38 ATPs

Proteins

Introduction to proteins

- They are polymers of amino acids, bound by peptide bonds
- 100 - 10,000 amino acids
- There are 20 amino acids - all with different side chains

- Functions of proteins
    - Structural and Mechanical fxns
        - Contractile Protein in Muscles

        - Collagen (skin, cartilage, blood vessels)
        - Keratin (hair and nails)
    - Hormones
        - Messenger molecules
            - Insulin
            - Glucagon
    - Immune functions
        - Antibodies attack bacteria and viruses
    - Enzymes: catalyze reactions
        - Ex.)
            - Hydrolase - split compounds
            - Dehydrogenase: remove H+
            - Kinase: add phosphate group
            - Ligase (synthase): joins 2 compounds together
    - Transporters (in the blood)
        - Albumin: vitamins and minerals (B)
        - Transthyretin (prealbumin): retinonal, thyroid hormone
        - Transferrin: Fe transport
        - Ceruloplasmin: Copper mineral
        - Lipoproteins: carry fat
    - Fluid Balance
        - Proteins attract fluid
    - Acid-base balance
        - Buffers
    - Intracellular signaling
        - Plasma membrane receptor
    - Energy
        - Accounts for about 5-10% of daily calories)
- Basic structure of amino acid
    - Each amino acid has:
        - Central Carbon
        - Amino Group (NH2)
        - Carboxy (acid) group (COOH)
        - Side Chain (R group)
            - Makes amino acid unique
- Basic structure of proteins (primary, secondary, tertiary, quaternary)
    - Primary
    - Secondary
        - alpha-helix
        - Beta conformation or Beta pleated sheet
        - Random coil
    - Tertiary
        - Hydrophobic and Hydrophilic interactions
    - Quaternary

        - Interactions between 2 or more polypeptide chains determine quaternary structure
- 3 ways of classifying amino acids
    - Net Electrical Charge
        - 2 amino acids = negative charge
        - 3 amino acids = positive charge
        - 15 amino acids = no net charge (neutral)
    - Polarity (tendency to interact with H2O)
        - Determined by side chain ®
        - 10 are polar and 10 are nonpolar
        - Polar amino acids are generally found on the surfaces of proteins
    - Essentiality
        - There are 9 essential (indispensable) amino acids
        - Some AA can become essential under certain physiological conditions
            - ex.) cyestine in premature babies
- Digestion and absorption of proteins
    - Digestion
        - Mouth/Esophagus = none
        - Stomach = HCl denatures proteins
            - HCl activates pepsin from pepsinogen
            - Enzyme (pepsin) breaks peptide bonds to form polypeptides
            - End Product = mostly large polypeptides
        - Small Intestine
            - Pancreatic Enzymes
                - Trypsinogen = trypsin
                - Chymotrypsinogen = chymotrypsin
                - Procarboxypeptidases A/B = carboxypeptidases
                - Several other peptides
            - Brush Border Peptidases
                - Aminopeptidases (digest: oligopeptides)
                - Dipeptidylaminopeptidasaes (digest: dipeptides)
                - Tripeptidases (digest tripeptides)
            - Final products of protein digestion:
                - Dipeptides
                - Tripeptides
                - Free Amino Acids
    - Absorption:
        - Most amino acids are absorbed in the duodenum and upper jejunum and some in the ileum
        - Amino acids can compete for the same carrier system
        - Some amino acids are used by intestinal cells
            - Ex.) glutamine is used by intestinal cells for energy and to stimulate cell proliferation
        - Amino acids enter portal vein to the liver

- Protein needs/recommendations
    - Adult RDA = 0.8 grams protein/kg body weight
    - Common recommendations for athletes: 1.2-1.8 g/kg
    - Acceptable Macronutrient Distribution Range = 10-35% of total cal
    - Protein requirements determined by nitrogen balance studies
        - Amount of N in = Amount of N out
- Nitrogen balance (what is it? What would cause positive and negative nitrogen balance?
    - Sources:
        - Dietary protein (proteins contain 16% nitrogen)
        - Nitrogen losses: urine, feces, and skin (skin it's hard to measure)
        - Nitrogen status = (protein intake g/6.25) - (urinary nitrogen + 2)
    - Positive Nitrogen Balance: More N in
        - Ex.) infants children, pregnant women, athletes
    - Negative Nitrogen Balance: More N out
        - Ex.) Starvation, Serious Injury, Illness, Physical Stressor
- Sources of protein
    - Exogenous sources (food)
        - Animal products
        - Plant products: grains/grain products, legumes, vegetables, nuts
    - Endogenous proteins (turnover of proteins in the body)
- Protein quality (based on digestibility and amino acid profile)
    - The measure of a protein's digestibility and how its amino acid pattern compares with your body needs
    - Influenced by
        - Digestibility
        - Proteins amino acid profile
    - Animal proteins are more easily digested and absorbed
        - Animal proteins: 90-99% digested
        - Plant proteins: 70-90% digested
- Sources of high-quality and low-quality proteins
    - High-Quality Proteins/Complete proteins
        - Supply all essential amino acids in the approximate amounts needed by humans
        - Sources: Milk, Yogurt, Cheese, Eggs, Meat, Fish, Poultry, and Soy Protein and Quinoa
    - Low-Quality Proteins (incomplete proteins)
        - Low in at least one essential amino acid
        - Most plant proteins
- What is mutual supplementation?
    - It's the incomplete protein foods can be ingested together so that amino acids become complementary
    - 2 incomplete proteins together can complement one another in digestion
- Introduction to amino acid metabolism
    - Sources of Amino Acids

        - Diet
        - Turnover of proteins in the body (Nitrogen balance)
    - Liver monitors absorbed amino acids and adjusts the rate of metabolism
        - For the catabolism of proteins/amino acids
        - For the anabolism of proteins
- The potential fate of an amino acid
    - Amino acids can be used
        - To build proteins
        - Convert to other AA or small nitrogen-containing molecules
    - Stripped of their nitrogen, amino acids can:
        - Can be used as energy
        - Converted to glucose, fat, CHOL, ketone, bodies
- How is the amine group (nitrogen) removed from amino acids? (transamination & deamination)
    - Transamination or Deamination of amino acids
        - Deamination = removal of amino group
            - Take amino acid → remove N group → makes ammonia = keto acid
        - Transamination = transfer of amino group from one amino acid to an amino acid skeleton (aka alpha-keto acid)
            - Keto acid A + Amino acid B → Amino Acid A + Keto Acid B
              transfer the N from the keto acid to amino acid
            - Catalyzed by aminotransferases
- Amino acid metabolism (How the body uses amino acids? To make proteins, other amino acids, small nitrogen-containing molecules, produce energy, make glucose and fat)
    - **Amino acids used to make proteins**
        - The liver makes hundreds of different plasma proteins
            - Albumin
                - Major protein transporter
                - Protein status = malnourished/deficient
                    - Check albumin levels
                - Used as an indicator of visceral (organ) protein status
        - Acute-phase proteins (inflammation/infection)
            - C-reactive protein = evaluates inflammation in patients
    - **Amino Acids are used to make other amino acids**
        - Essential amino acids can be converted to nonessential amino acids
        - Ex.) Phenylalanine to tyrosine
    - Amino Acids are used to make small nitrogen-containing molecules
        - Glutathione = major antioxidant
        - Carnitine = transporter of long-chain fatty acids into mitochondria to use for energy
        - Creatine = part of phosphocreatine found in muscle use for quick energy
        - Carnosine = antioxidant (muscle/brain)

        - Choline = is a part of acetyl choline (neurotransmitter) and some phospholipids (lysothine)
        - Purine and Pyrimdine Bases, which are needed to make DNA (deoxyribonucleic acid) and RNA (ribonucleic acid)
            - Synthesized in the liver
            - Nucleic acids are made of
                - A 5-Carbon Sugar
                - A Phosphoric Acid
                - Nitrogenous Bases (e.g. purines and pyrimidines)
    - Amino Acids used for energy
        - Glucodenic amino acids are converted to pyruvate → glucose
        - Ketogenic amino acids are converted to acetyl CoA → ketones
    - Some amino acids can be converted to glucose
        - Glucogenic amino acids are converted to pyruvate
    - Amino Acids can be converted to fat
        - Both glucogenic and ketogenic amino acids can be converted to fatty acids
            - Excess anything can be converted to fat
        - Con contribute to weight gain
- What is an alpha-keto acid?
    - It is what's left when the amino group is removed
- Summary of non-nitrogen uses of amino acids
    - Once the amino group is removed, the C skeleton (alpha-keto acid) can be used to produce:
        - Calories/energy
        - Most can be converted to glucose (gluconeogenesis)
        - Ketone bodies
        - CHOL
        - Fatty acids
- Disposal of nitrogen waste
    - The use of amino acids for energy also produces carbon dioxide and ammonia
    - Ammonia is highly is highly toxic and is converted to urea in the liver
    - The kidneys filter urea from the blood
    - Urine nitrogen
        - Mostly urea
        - Some Uric Acid (from breakdown of nucleic acid) (DNA/RNA)
        - Creatinine
        - Ammonia
- Metabolism of amino acids in the muscles (major site of BCAA metabolism, how do muscles get rid of nitrogen waste, what do creatinine and 3-methylhistidine measure?)
    - 40% of body's protein is found in muscles
    - Main site for metabolism (degradation) of branched chain amino acids
    - Since muscles do not make urea, nitrogen (waste) is used to make glutamine and alanine (nonessential amino acids) and they are transported to the liver

        - Liver makes urea
    - Muscles do give off N waste = ammonia
    - Muscles can't make urea so they need to get transported to the liver
    - Indicator of muscle mass
        - Urinary creatinine (degradation product of creatine)
        - Not always accurate
        - Creatinine helps regenerate ATP found in muscles
    - Indicator of muscle catabolism (degradation)
        - Urinary 3-methylhistidine (by-product of normal turnover of muscle proteins)
            - Malnourished people make more
- Metabolism of amino acids in kidneys (perform gluconeogenesis and rid the body of nitrogen waste, what are the major nitrogenous waste molecules?)
    - The kidneys preferentially take up a number of amino acids from the blood and metabolize them
        - Ex.) the kidneys are the major site for the production of arginine, histidine, serine, and possibly tyrosine
        - The kidneys perform gluconeogenesis
        - The kidneys are responsible for ridding the body of nitrogenous waste
- Kwashiorkor vs Marasmus (know their differences)
    - Protein-energy Malnutrition
        - Inadequate calories and/or protein
        - Most widespread form of malnutrition in the world
        - More common in children
        - 33,000 children die each day from malnourishment
        - 2 Types
            - Marasmus
                - Severe deficiency of calories
                - Insufficient protein
                - Signs
                    - Extremely thin
                    - Lack of Growth (stunt, waste, etc)
                    - Loss of fat stores
                - Mostly kids 6-18 months
                - Diarrhea can lead to death
                    - Severe dehydration
                    - Too weak to recover
                - Severe energy deficiency
                    - Lowers insulin
                    - Raises glucagon
                - Chronic protein deficiency
                - Net effect:
                    - Raises fat metabolization from adipose cells; break down

- Raises Ketone bodies (brain can decrease)
- Decreases Protein Synthesis
- Raises AA mobilization from muscles (breakdown)

- Kwashiorkor
  - Severe defiency of dietary proteins
  - Signs
    - Edema (swelling in abdomin)
    - Muscle loss
    - Skin rash/hair changes
    - Water and Electrolyte Imbalance
  - Typically occurs in older infants (1-3 y/o)
  - Eating carbs, so raises insulin
  - Muscles: decrease protein breakdown
  - Liver: Decreases release of plasma proteins
  - Liver: Decreases osmotic pressure
  - Water leaves blood vessel → edema
    - Bloated belly

- Why/how does abdominal edema occur with Kwashiorkor?
  - Liver decreased release of plasma protins (e.g. albumin) → osmotic pressure → outside the blood vessels → edema bloated belly.

Carbohydrates

- What are glycosidic bonds? What are the most common types of a bond (Alpha 1,4) and what does the Alpha 1, 4 mean?
  - Glycosidic bonds: the bond between two sugars/carbs at the 1st Carbon of 1 sugar and 4th carbon on another.
  - OH group of one monosaccharide and the OH group of another monosaccharide
- What are the monosaccharides and disaccharides? What sugars are in disaccharides? Common Name? Food Source?
  - Simple Sugars
    - Monosaccharides and Disaccharides
      - Glucose
      - Fructose
      - Galactose: milk sugar

        Glucose + Fructose = Sucrose: table sugar

        Glucose + Galactose = Lactose: milk sugar

        Glucose + Glucose = Maltose: malt in beer
- Sweetness Levels of Sugars:

- Sucrose the Sweetest at 100
- Maltose 46
- Lactose at 35
- Galactose at 32

- Trends of sugar availability in the U.S.
  - In 2017, 126.6 lbs per person of caloric sweeteners were consumed which is down from 151.5 in 1999.
- What are Oligosaccharides? Common Food Sources?
  We lack the enzymes to digest these.
  Oligosaccharides (3-10 sugars) Found in beans, peas, grains.
  - Raffinose
  - Stachyose
  - Verbascose

  Humans can't digest so bacteria throw a fermentation party = gas
- Polysaccharides: More than 10 monosaccharides (starch, glycogen, cellulose)
  - Starch and 2 forms: Stored Carb/sugar in plants. Amylose (unbranched) + Amylopectin (branched)
  - Glycogen and Where is it Found?: Glycogen is the stored form of carb/sugar in animals. Found in the liver and muscles.
  - Fiber: Supporting sugar of plants, can't be digested by humans, insoluble and soluble. ex.) cellulose
- Sugar Alcohol (Calorie, Ex. Details about Glycerol, Sorbitol, and Mannitol):
  - Mono- and Disaccharides
  - Less sweet than table sugar (sucrose)
  - Similar Caloric value as sucrose but absorbed slower
  - Sorbitol and Mannitol are incompletely absorbed which leads to a laxative effect.

SECTION 2: Digestion

- Digestion of Carbs (Where does it occur, What are the enzymes involved; where did the enzymes come from? What are the enzymes digesting?):
  - Starts in the mouth with salivary amylase, and hydrolysis
  - Digested in the small intestine with pancreatic enzymes **[pancreatic A(1-4)]** and **Dextrinase**. Digests starch into oligosaccharides (dextrins) and maltose.
  - Brush border disaccharidases digest disaccs-
- The process of breaking Glycosidic bonds is a hydrolysis reaction.
  - Disaccharides are hydrolyzed and broken into monosaccharides
- Mechanisms of Carbohydrate absorption:

- Absorptive capacity in humans is 5,400 g/day of glucose and 4,800 g of fructose.
- Monosaccharides are absorbed into enterocytes
    - Active transport: requires energy and an SGLT1 carrier (glucose + galactose)
    - Facilitated diffusion: concentration gradient (fructose, some glucose, galactose)
    - Simple diffusion: sugar alcohols

- Transportation of Carbs (Where do they go after absorption, what happens to galactose and fructose?):
    - All sugars are destined to enter the blood and then to the liver (via the portal vein)
    - Galactose and Fructose are converted to glucose in the liver
    - Glucose enters systemic blood circulation
- Glucose transporters: what are they and know the importance of GLUT4: Glucose cannot pass through the cell membrane just by simple diffusion. 14 different transporters used, but GLUT4 is the only one that requires insulin. HEART, MUSCLE, ADIPOCYTES
- **Fate of Glucose (4 Endpoints):**
    - Converted to Energy: **GLYCOLYSIS**
        - Anaerobic: 2-3 ATPs per glucose molecule
        - Aerobic: 36 - 38 ATPs per glucose molecule
    - Stored as Glycogen: Liver and Muscles. **GLYCOGENESIS**
    - Made into Fat: in Liver: Glucose to Fat **LIPOGENESIS**
    - Goes into the Pentose Phosphate Pathway:
        - **produces NADPH and ribose used to make DNA and RNA**
- Carb Metabolism Pathways (Beginning and Ending Substrate and know if there are any significant products made, like ATP or NADH/FADH2, # of carbons in glucose, pyruvate, lactate, acetyl CoA.)
    - **Glycogenesis**: Glycogen from glucose: muscle and liver. Create Glycogen
    - **Glycogenolysis**: Glucose from Glycogen, breaking down of glycogen. Liver shares it's glycogen and muscles do not
    - **Glycolysis**: Oxidation of glucose to produce: **2 pyruvates**, **2 ATP** (*3 if it was from glycogen*), and **2 NADH** (1 NADH = 3 ATPS) (1 FADH = 2 ATPS)
    - **Pyruvate to lactate**: Lactate (no O2) Acetyl CoA (with O2)
    - **Pyruvate to acetyl-CoA**: This must happen before the Krebs Cycle can; produces **1 NADH.**
    - What happens to pyruvate? (Depends if Aerobic or Anaerobic Environment):
        - Anaerobic = lactate/lactic acid
        - Aerobic = Acetyl CoA

- Krebs Cycle: Starts w/ Acetyl CoA ends with oxaloacetate. 3 NADH, 1 FADH, and 1 GTP. **(REMEMBER KREBS CYCLE HAPPENS 2x)**
- Pentose Phosphate Pathway: aka Hexose Monophosphate Shunt; produce **NADPH** *which is required for the synthesis of fatty acids* and **ribose-5-P.**
- **Gluconeogenesis**: Formation of glucose from non-carbohydrate sources (molecules with at least 3 Carbons) so:
  - (glucogenic amino acids, lactate, glycerol or triglycerides, and pyruvate)
  - Occurs mostly in the liver
- **Lipogenesis**: storing fat from excess carbs. Acetyl CoA used to make fatty acids

- ATPs produced from glucose in the aerobic and anaerobic environment: 2 or 3 ATP in anaerobic. 36-38 in an aerobic environment
- **Insulin:** Hormone secreted from where? When is it elevated? Insulin Actions?
  - Insulin secreted from the pancreas beta cells, elevated with an increase of plasma glucose
  - Increases the expression of enzymes to increase the synthesis of glycogen, lipids, and proteins.

    \+ **Glucose uptake by most cells**
    \+ **Glycogenesis**
    \+ **Glycolysis**
    -- Glycogenolysis
    --Glycogenogenesis

- Insulin Antagonists: When are they elevated? What are their actions?
  - Glucagon: released by the pancreas
  - Epinephrine: released from the adrenal medulla
  - Cortisol: released from the adrenal cortex

    \+ **Glycogenolysis**
    \+ **Gluconeogenesis**
    -- Glucose uptake by cells
- High Fructose Corn Syrup: Background/History, Advantages, Research regarding health effects
  - Really got a bad rapport for no reason
  - No direct link to obesity and diabetes, not inherently worse than other sugars.
- Sugar and Risk of Dental Caries:
  - Mouth bacteria love sugar, sticky sugar sticks around.
- Diabetes: 2 types, warning signs, and blood glucose interpretations, potential complications
  - Type 1: don't produce insulin
  - Type 2: Cells do not respond to insulin well (decreased effect)

- Warning Signs:
    - **Hyperglycemia**:
        - Eye: Retinopathy, Catarat, Glacoma: Blind
        - Kidney: Nephropathy, Microalbuminuria, Gross Album: Kidney Failure
        - Nerves: Neuropathy: Peripheral, Autonomic: Amputation
    - **Thirst** (polydipsia)
    - **Frequent Urination** (polyuria)
    - **Excessive eating** (polyphagia)
    - **Blurred Vision**
    - **Infections**
    - **Weight Loss** (type 1)
    - **Fatigue**
    - **Poor wound healing**

- Lactose Intolerance (lactase issue, symptoms, treatment)
    - Deficiency of lactase
        - Effect many as they age
        - Children of nonwhite races
        - Most often inherited
    - Symptoms:
        - Nausea
        - Cramps
        - Bloating
        - Diarrhea
        - Gas
    - Tips/Treatment
        - Small amounts through the day instead of all at once
        - Yogurt or Cheeses
        - Oral Lactose Digesting Enzyme