* How all the pathways are integrated
* Major sources of energy – proteins, glycogen, and lipids
* Energy is counted as ATP
* Gluconeogenesis – under extreme starvation – when need glucose for brains
* Ketone bodies can go to brains and provide energy
* Glycolysis - G6P is the key metabolite – glucose after phosphorylation
* Glycogen – excess glucose stored as glycogen
* Acoa and mcoa make fatty acids
* Amino acid degradation – aa becomes urea and get rid off – carbon goes to pyruvate
* Adipose tissue is storage of fat
* Brain produces nothing – consumer
* Liver makes some stuff and store some stuff
* Heart – cac, etc, oxidation phosphorylation to generate ATPs – oxygen only appears in complex III
* Glycerol – when hydrolyse tga get fatty acid and alcohol that is glycerol – go directly into glycolysis
* Malonyl-coa is the choke point in liver
* Blood is a transporter – glycolysis is primary energy source
* Epinephrine and norepinephrine – override insulin control – insulin says store – epinephrine says burn
* GPCRs – important for response to glucagon and epinephrine – get glucose immobilised
* Muscles – first thing they burn is ATP – creatine is a protein that is phosphorylated and give P to ADP to make ATP – oxidative phosphorylation is the last one that gives the most ATPs
* NADPH is needed in lipid synthesis – stronger reducing equivalent than NADH
* Glycerol fed to glycolysis
* Fatty acids fed to CAC
* Insulin and glucagon are long-lived
* Epinephrine and norepinephrine are short-lived
* Lots of transporters that facilitate glucose transport into cells – eg. GLUT 4
* Insulin likes making thing – prevent break down of glycogen
* Lipogenesis is making lipids
* Lysis means breaking down
* Genesis mean making
* Excess energy can be stored using AMPK – activated when we have high AMP – very low energy – inhibit ATP utilising pathway (all the synthesise pathways)
* mTOR – axillary pathway – store lipids
* AMPK and mTOR – triggered by nutrient availability (usually aa)
* If break down too much lipids – too much energy – oxidative stress
* Obese mice – mice that lack Leptin – become fat – same food supplies were available – adiponectin controls how many fats we can mobilise – Ghrelin is the opposite of the 2 by making us hungry
* Insulin and leptin prevent taking food in – stop getting hungry
* We have to eat some times – stop eat and cope with what already stored – if adjust metabolism, can fast for months
* In ancient time, people were always hungry – when metabolic flexibility evolves