Glycolysis

- Cytosol of almost all cells, Pyruvate transported to mitochondrial matrix via pyruvate-H+ symport
- G-6-P[-] (Hexokinase) , ATP [-](PFK, Pyruvate Kinase), Citrate [-](PFK), AMP[+](PFK), F16P2 [+])Pyruvate Kinase)
- Glucagon[-](Pyruvate Kinase and PFK1) and Insulin [+](Activating synthesis of hexokinase, PFK and Pyruvate Kinase)

Pentose PP

- Cytosol of almost all cells
- High NADPH [-] (inhibits G6P dehydrogenase, glycolysis favored) High NADP+ [+](G6P dehydrogenase ,PPP favored)

Gluconeogenesis:

- Pyruvate → OAA → Malate in mitochondria, Malate shuttled out, OAA → PEP in cytosol of Liver and Renal Cortex cells
- G6P[+](Hexokinase), F26P2 and AMP [-] (F16-Bisphosphatase), Acetyl-CoA [+](Pyruvate Carboxylase)
- Glucagon[+](Activates PEP synthase), Insulin[-](Inhibits PEP synthase)

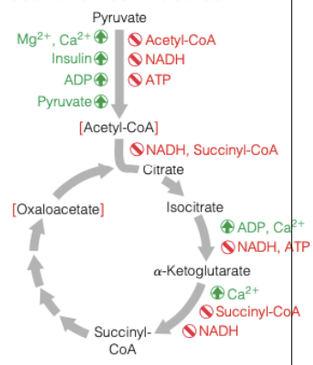
Alanine Cycle:

- Muscles: Glucose → Pyruvate → Alanine, alanine transported to Liver, Glucose reformed glucose transported to muscles

Cori Cycle

- Muscles: Glucose → Pyruvate → Lactate, Lactate transported to Liver, Glucose reformed, transported back to muscles

Citric Acid



- Pre TCA and TCA happen in Mitochondrial Matrix
- Feedback inhibition with Pyruvate[+]/Acetyl CoA[-]
- Covalent Modification of PDH: by Mg, Ca, Insulin [+] Inactivation by ATP, NADH, Acytl-CoA[-]
- Allosteric regulation by NADH[-] Succinyl-CoA[-], ATP [-], ADP[+]

Malate-Aspartate Shuttle

- Cardiac and liver cells use Malate-Aspartate shuttle because they lack NADH transporter

Glycerol-3-P Shuttle

- Brain and skeletal muscle use the irreversible Glycerol-3-Phosphate shuttle to charge FADH2

Glycogenesis and Glycogenolysis

- Glycogen stored in liver and muscle cells, glucose delivered to brain or muscles
- Reciprocal control of glycogen synthase and glycogen phosphorylase, cAMP[+](GP), ATP and G6P [-](GP), G6P[+](GS)
- Insulin(+GS and -GP), Glucagon(-GS and +GP, +Gluconeogenesis), Epinephrin(-GS and +GP, fight or flight)

Electron Transport Chain

- Happens in the inner membrane of mitochondria
- Regulated solely by ATP vs ADP concentration

Lipid Degradation

- Lingual, Gastral and Pancreatic Lipases, uptake in small intestine in micelles made of bile salts
- Regulated by available fat, lipase activity

Beta Oxidation

- In the mitochondria of all cells except brain and blood, fatty acids from the cytosol enter though carnitine shuttle (CPT)
- Substrate availability: Lipases activated by glucagon and epinephrine (Starved) and inhibited by insulin (Fed)
- Malonyl-CoA inhibits CPT1 (FED) and Acetyl-CoA carboxylase inhibited by FFA concentration (Starved) so CPT1 Active

Ketogenesis

- Ketone bodies are synthesized in the liver, but cant be used there, mainly used in muscles and brain
- Activated when OAA is scares, which is in the starved state, by the concentration of FFA from adipose, and Acetyl-CoA concentration

Fatty Acid Biosynthesis

- In Liver, Acetyl-CoA is transported from mitochondria to cytosol Via citrate transport system, rest happens in cytosol
- Regulation targets Acetyl-CoA carboxylase (Rate limiting), Citrate activates it, and Palmityl-CoA inhibits it
- Insulin activates Acetyl-CoA-Carboxylase, glucagon inactivates it through phosphorylation

Urea Cycle

- Glutamine is formed in most tissues, reforms glutamate in liver, depositing NH4 for Urea cycle: only in liver
- Urea cycle starts in mitochondria, ornithine and carbamoyl phosphate combined here, citrulline transported to cytosol
- Short term regulation by increased arginine, which increased N-Acetylglutame synthesis, which allosterically activates Carbamoyl Phosphate synthase, forming more Carbamoyl Phosphate
- Long term regulation: High protein diet increases Urea Cycle enzymes