* Lipids for energy – also build membranes – signalling
* Degradation of fatty acids in mitochondria – got acetyl-CoA for CAC
* Synthesise of lipids – in cytosol – different in compartment – need acetyl coa, malonyl-CoA and fatty acid
* Lipids or TGA – have to go up from acoa to fatty acids – linked with glycerol to make TGA
* Need system to transport acoa outside of the mitochondria
* Pyruvate dehydrogenase – pyruvate to acoa
* Carboxylation of acoa to generate malonyl-coa – adding co2 – acoa has 2Cs – malonyl has 3Cs
* Fatty acid synthase – enzyme – build 2Cs at a time – a loop that goes on until it finishes the cycle
* Desaturases – make double bond in fatty acid – unsaturated fatty acid
* Long chain can have double or single bonds – fatty acids and glycerol form triacylglycerol
* Intermediates for degradation and synthesis are the same
* Oxidation degradation starts with lipids that are linked to carriers – synthesisw starts with a or m coa linked to different carriers
* Fatty acid synthase can make up to 16Cs but elongases and desaturases can increase more Cs
* Get acoa outside of mitochondria – citrate goes out of mitochondira – citrate lyase that produces acoa and can be used to make fatty acids
* Oxaloacetate becomes malate then pyruvate and produce NADPH – NADPH used in fatty acids synthesis
* Bicarbonate is the source of CO2
* Fatty acid synthasis (Different enzymes doing different things but in nonplant eukaryotes and some bacteria, all done by 1 enzyme – type I fatty acid synthase
* All the enzymes in 1 protein with different subunits – very efficient – all substrates very close so no loss to diffusion
* Degradation is a catabolic process
* Beta oxidation uses 2Cs in every cycle – biosynthesis adds 2Cs every cycle – difference oxidation loses as acoa – biosynthesis need mCoA and need NADPH as electron donor
* NADPH only seen in PPP
* In b oxidation, produce reducing equivalents (used in etc) – biosynthesis needs reducing equivalents
* Essential fatty acid – we cannot produce
* Fatty acids – carboxylic acid + long chain of Cs
* TGA – glycerol and 3 fatty acids – glycerol can come from pyruvate
* Glycerophospholipid – 2 fatty acids linked to glycerol and have phosphate group – hydrophilic and hydrophobic parts
* Steroids – isoprene links with … - made from cholesterol
* Cholesterol linked with LDL – cell will be engulfed in LDL and attacked and cause inflammation
* So it is not cholesterol that is bad or good – it is what it is linked with
* Vitamins synthesise cofactors of many enzymes – need to be ingested
* Lower cholesterol in blood – inhibitors (approved drugs)
* Summary
  + Degradation from fatty acids to acoa is beta oxidation
  + Fatty acids can form other lipids or form triacylglycerol that store energy in adipose tissues
* Insulin is important when blood glucose high – bind to receptors allowing transport of glucose into cells – produce acoa – make energy or store as fat
* Glucagon is opposite – degrade fatty acid to produce energy – or from glycogen
* Citrate activates acc – stimulate production of fatty acid
* ACC is inhibited by fatty acid – cuz no need to produce more
* Glucagon wants degradation – activates lipases – adipose tissues cut the lipid and produce energy – inhibit ACC