* Beta-oxidation – fat burning – breaking down fat – catabolic process – break down fat for energy (lipid)
* Left part of the graph of slide 3 – lipid
* Triacylglyerols – main class of molecules to get energy and compose diet
* Catabolic pathway – red arrows going down
* Break down TAGs get fatty acids and glycerol
* Glycerol will feedback into glycolysis and gluconeogenesis
* When break down fatty acid – will get acetyl-CoA which goes to down to CAC and etc and oxidativeP to get ATPs
* TGAs – glycerol and fatty acids – fatty acids….- ATPs
* Lipids major source of our energy – 5 to 25% of body weight – 90% is TGAs
* Structure of TGAs represent glycerol – triacyl is the green (slide 4) – when break down they are fatty acids
* Can get from diet – stored in adipose tissues – or synthesise them
* Mainly though diet
* Major role is to produce ATPs
* Another important role is plasma membrane – several other membranes – inner and outer membranes of mitochondria
* Glucose has CHOH but TGA has … more opportunities to donate electron
* Lipids are lipophilic so they try to stay together and pack together more than glucose in water
* Stearate ion – form by 18 Cs – fatty acids – long chain of carbons – polar group is the carboxylic group that likes water
* Oleate ion – has one unsaturated bond – double bond – 18:1n-9 – carbon 9 has the unsaturated – mainly in cis position – bending
* Can get different fluidity – double bond in more oily face – saturated more solid
* Capric acid - Decanoic acid – 10:0 – these fatty acids have 10 Cs and non-double bond
* Palmitoleic acid – 16:1c(tri)9 – one unsaturated on carbon 9
* Will focus on palmitic acid and palmitoleic acid
* In order to make TGA break down, need bile salt – lipid insoluble in water – bile salt solubilised the lipid and transform them into the small intestine – enzymes cleave the lipids and store in the intestine
* Another way – synthesise in the liver – de novo synthesis – body able to synthesise it – store in adipose tissues
* Body can mobilise the lipids if they are stored
* TGAs ingested – bile salts will cover the lipids – act as a platform for lipase to bind and start digesting – lipase makes these lipids able to store in the intestine – work for vitamins too
* Transport the lipids through lipoproteins – mixture of lipid and protein
  + To transfer sth, lymphatic system is composed of water – lipids will precipitate – need protein to transfer
  + 5 classes of lipoproteins
  + The density is proportional to the amount of triacylglycerol – TGAs go down – density becomes higher
* Lipoproteins have a core – made by TGAs and/or cholesterol esters, phospholipid with tails going inside, cholesterol, hydrophilic facing out
  + Function to transfer lipids
* Chylomicrons – intestine mucosa converts the fatty acids to TGAs and packed into chylomicorns and can go to other parts of the tissues
* Chylomicron formed in the interstine – transferred to the capillary – chylomiconr and VLDL bind to the capillaries – lipases are waiting for them and cleave the lipids – release free lipids and glycerols that are taken up by the cells
* Chylomicron is unpacked by hydrolysis of TGAs – generate glycerol and fatty acids – resynthesised and stored in adipose tissues
* Liver can biosynthesis of fats and cholesterol – VLDL transfers them to capillaries and continue that pathway
* LDL – cells have receptors to capture LDL – LDL contains proteins and TGAs and phosphates and cholesterol – when cells take up too much LDL – will be deposit of cholesterol – will generate atherosclerotic plaque – heart damage
* HDL – transport cholesterol back to the liver
* Good cholesterol and bad cholesterol – cholesterol is part of membrane – what matters is what the cholesterol is – if LDL can accumulate – HDL is good cuz can send back to liver and recycle
* LDL receptors recognise LDL particles – ball of LDL coming towards cell – yellow is inside blue is out – LDL comes from blood stream – receptor mediated endocytosis – receptors recognise ligands and make endocytosis – invagination and then pH goes down – detached from receptors – fused with lysosome that has many enzymes that degrade proteins into aa – cholesterols can be reused by cells to make membranes or accumulate – bad if accumulate lots of cholesterol – LDL forms plaque – immune cells attack plaque
* LDL can be oxidised – once oxidised – macrophage can eat a lot of the LDL – accumulate a lot of cholesterol – convert to foam cell – precursor to atherosclerotic plaque
* Omega-3 – desaturation on the 3rd carbon starting from the back – double bond can be oxidised – omega 3 will be oxidised instead of LDL
* Adipose tissues can store these lipids – during stress or fasting or starvation – hormones will bind to the pathway that is cAMP mediated – activated by P – the TGA will become fatty acid
* Starvation – eg. low blood glucose – epineprhne and glucagon bind to receptors of cell – activate cAMP – phosphorylate – activate lipase – lipase degrades yellow ball of TGAs – make lipids available – can bind to albumin in blood and bring lipids to other tissues
* Oxidation – burn the lipids – degrade them through catabolic pathway – generate energy
* In 1904 – first experiment where metabolic traces were used – molecules resembling fatty acids – have phenyl ring at the end – took the 2 molecules and fed dogs and measure the urine – when have even number of C chain – got phenalytic acid (2C and phenyl group) – when fed odd got benzoic acid (1 C) – conclude cleaveage always alpha-beta – beta oxidation
* When chain very long – cannot simply diffuse – need transport system – CoA – converted and substitute by carnitine
* Fat Mobiliser and carnitine – carnitine transports fat – but it is not that easy to control burning of fat by drugs – heavily regulated by the body
* C16 – acyl-CoA – no double bond – dehydrogenation is the first reaction – generate 7 acetyl-CoA at the end