Body Mass Index \( (\text{kgm}^{-2}) \) = \( \frac{\text{weight} (\text{kg})}{\text{height}^2 (\text{m})} \)

**Catabolism** – Oxidative (electron removal) breakdown from large to small
**Anabolism** – Reductive (electron acceptance) build-up of large from small

**Creatinine** – Produced in proportion to muscle mass from spontaneous breakdown of creatine & creatine phosphate
**Creatine Phosphate** – Short term energy store created from creatine, catalysed by creatine kinase
\[ \text{ATP} + \text{Creatine} \rightleftharpoons \text{Creatine Phosphate} + \text{ADP} \]

**Metabolic Disorders**
- **Lactose Intolerance** – Low lactase activity meaning gut bacteria ferment lactose to produce acid which leads to cramps & diarrhoea via irritation of the GI tract
- **Galactosaemia** – Can be a deficiency is galactokinase or a transferase deficiency, the latter is more common & hepatotoxic as galactose is reduced to galactitol, which uses up NADPH
- **Fructose Intolerance** – Aldolase deficiency causing an accumulation of fructose-1-phosphate that results in liver damage

**ATP Synthesis** (coupling) – Electrons from reduced carriers are transferred along protein complexes creating energy to pump proteins across impermeable membrane and build up a gradient; terminal electron acceptor is oxygen. Energy from dissipation of the proton motive force drives ATP synthesis

**Uncoupling** – Certain chemicals can increase membrane permeability to protons meaning proton motive force is dissipated as heat energy and not used to drive ATP synthesis

**Harmful Byproducts** – During electron transport electrons can leak and react with oxygen to form superoxide \( (\text{O}_2^-) \) radicals which are reactive oxygen species (ROS)
- **Glutathione** – Reducing agent that can protect from oxidative damage, recycled via action of NADPH

**Consequences of NADPH Depletion:**
- Cataracts
- Heinz Bodies
- Reduced anti-oxidant capacity
  All work via increased disulphide bond formation

**Lipids**

**β-Oxidation** – Activated by linking CoA (ATP dependant), then fatty acids are transported into mitochondrial matrix via carnitine shuttle then two carbons (acetyl CoA) are cleaved off at a time

**Lipogenesis** – Acetyl CoA converted to malonyl CoA (C3) which uses ATP and then fatty acid chain synthesis by adding 2 carbon units at a time which requires NADPH

**Ketone Bodies** – Synthesised in the absence of glucose for cells containing mitochondria, including CNS as the can cross the blood brain barrier

**Lipoproteins:**
- **Chylomicrons** – Transport dietary triacylglycerols from intestines to tissues; normally disappear from blood after 4-6 hours (present in fasting plasma in type 1 hyperlipoproteinaemia)
- **VLDL** – Transport liver synthesised triacylglycerols from liver to adipose for storage
- **LDL** – Transport synthesised cholesterol from liver to tissues
- **HDL** – Transport excess tissue cholesterol from tissues to liver for disposal in bile salts

**Bile Salt Sequestrants** – Increase bile disposal & thus cholesterol from body by preventing reabsorption & loss in faeces