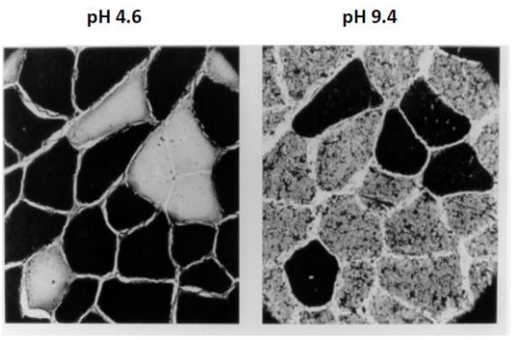
Type I and II fibres

Staining for Myosin ATPase which splits ATP during cross-bridge cycle; the faster this process is, the faster ATP can be cycled and muscle contracted



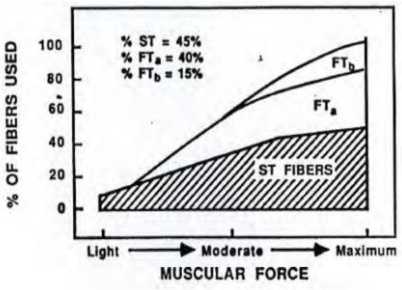
- The **fast twitch (II) Myosin ATPase is acid soluble** so it dissolves out on left; thus, when stained, the dark fibres are the slow twitch fibres and the light are the fast twitch fibres
- The **type I Myosin ATPase is dissolved out in alkaline pre-incubation**; the type II stain dark after alkaline
- A different isoform of the Troponin C can also be used to differentiate the fibres using immunoblotting (or by MHC I vs II)

Properties*	I	IIa	IIx
Alternative names	SO, ST	FOG, FTa	FG, FTb
Myosin heavy chain isoform	MHC1	MHC2A	MHC2X
Time to peak tension (msec)	80	30	
Force/power output	+	++	+++
Endurance capacity	+++	++	+
Distribution in whole muscle (%)	50-55	30-35	10-20
Mitochondrial density	+++	++	+
Capillary density (cap/fibre)	4.2	4.0	3.2
Fibre area (μm²)	5310	6000	5600

Human muscle tends to be **heterogeneous** (except in extreme circumstances)

- More mitochondria in Type I fibres
- GLUT4 expression is 20% higher in Type I than Type II
- **Capillary density tends to be higher in the slow twitch (I) fibres which is consistent with oxidative phenotype**
- Type II fibres generally bigger than Type I fibres

Henneman size principle: Slow twitch fibres will be recruited first



The fast twitch fibres will be recruited together with the slow twitch fibres at high muscular forces but you cannot maintain the fast twitch fibres as long (and force cannot be maintained)

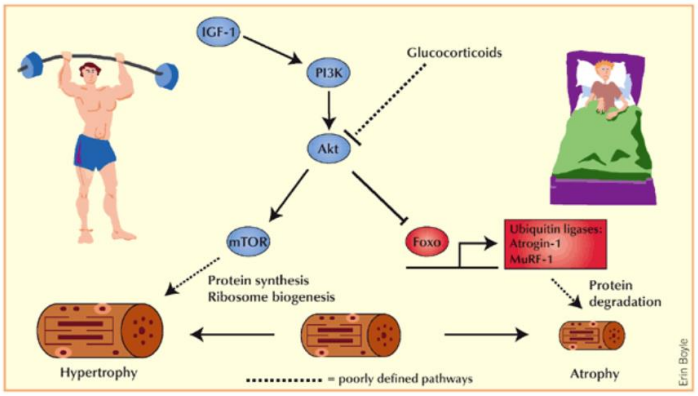
- As you increase exercise duration, you start to recruit fast twitch fibres as the slow twitch fibres drop out

In **sprinters, they have higher fast twitch fibres** compared to endurance athletes

- It is very hard/impossible to train fibres to be fast, so **sprinters are born i.e. largely genetic**
- However, **slow twitch fibres can be transformed from fast twitch fibres and the fast twitch fibres can also take on greater oxidative characteristics**; so, marathon runners are "made"
- There can also be **adaptation of fibre type** (especially of slow twitch fibres) at certain intensity can cause changes in metabolic characteristics (rather than contractile properties) in areas that are used more e.g. kayakers and their arms

An **increase in muscle cross-sectional area occurs with use** (especially high-intensity resistance exercise); there is increase in maximal voluntary contraction and EMG increase (indicating neuromuscular component)

If you do no use the muscle, there will be **atrophy (reduced CSA) with reduced myofibril synthesis** (so people are often mobilised after surgery and this can also occur in the diaphragm in people being ventilated)



- **GH works through IGF pathway** to increase muscle mass (through **mTOR**)
  - **GCs can suppress this pathway to induce atrophy**
- Disuse pathway acts through **Foxo**
- Anabolic steroids work through testosterone

Mitochondrial adaptations to exercise training: Increased mitochondrial density and oxidative enzymes which

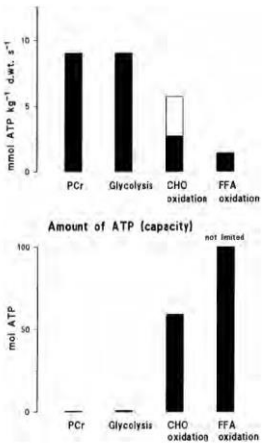
- **Reduces CHO reliance** and lactate production
- **Increased fat oxidation during exercise**
- Enhanced endurance performance
- Improved insulin action
    - They have benefits during rest but more so during exercise

This occurs due to changes in the stimuli e.g. Ca2+ and energy status, hormones etc, signalling and transcriptional regulation

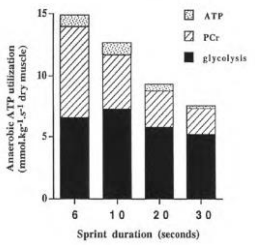
When muscle contracts, there is changes in energy status that increased AMPK pathway and increases in Ca2+ (which acts on Ca2+ sensitive kinases) which changes protein transcription which can increase GLUT4 expression

- **Muscle can also release protein which can bind and have effects elsewhere in the body** e.g. metabolites which can regulate mood states.

The capacity and power of the energy systems varies according to the intensity and duration of the exercise e.g. in sprinting you rely on creatinine phosphate and glycolysis where in endurance, you rely mostly on CHO and FFA



- ATP generation is higher when using creatinine phosphate and glycolysis and lower with CHO oxidation and lowest with FFA oxidation
- The capacity is inverse to this; there is **infinite store of fat for energy, then moderate availability of CHO for oxidation** (but the creatinine phosphate and glycolysis have a low capacity)
- CHO has a greater power output than fat so when you run out of CHO, you slow down because you cannot maintain the same rate of FFA oxidation

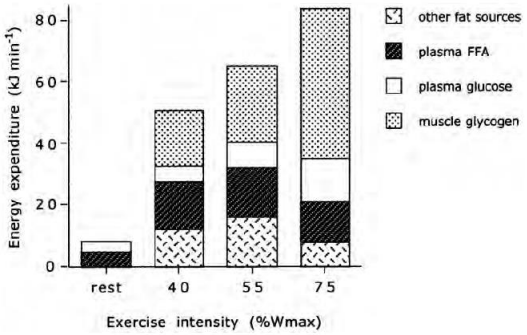


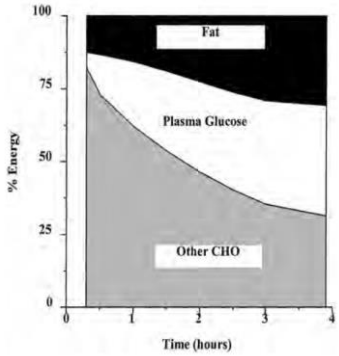
During sprinting, you tend to burn more high energy fuels e.g. **creatine phosphate and glycolysis** and this decreases quickly

- However, with increased duration, the relative contribution of these will go down and you will rely more on glycolysis
- With time, glycolysis declines and **aerobic pathway picks up; at 1 min exercise, it will be 50% aerobic and 50% non-aerobic sources**

In endurance, you tend to use more fat at the lower intensities (with some CHO utilised)

- At about **55% of maximal work load, you are oxidising maximum amount of fat**
- **At higher intensities and increase energy expenditure, you become more reliant on CHOs** (which is relatively finite because most energy is stored at fat, this becomes a factor later)





For example, **after 4 hours there is increased reliance on fat but still 70% comes from CHO as muscle glycogen (=other CHO) and lactate** (but you have to stop this intensity because the level of CHO oxidation is insufficient to maintain the exercise intensity e.g. **glycogen in muscle is deprived; this is hitting the wall)**

Factor influencing exercise metabolism: the substrates used for energy are mediated by the **substrate availability** i.e. high CHO diet will mean you burn more CHO and same for fat, **hormone levels** and **characteristics of the skeletal muscle**

- Exercise intensity, duration, diet, training, environmental temperature (burn more CHO when hot),
- **Age**: VO2 max decreases with age, which means relative exercise intensity at given power output increase so you tend to **burn more CHO**
- **Females tend to burn more fat at given power** (due to **oestrogen**)

Fatigue

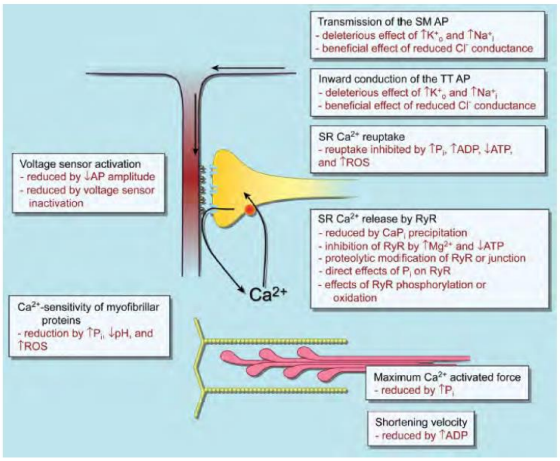
This is defined as a **reduction in force and power generating capacity due to something going on in the muscle or the inability to maintain the required or expected force or power output**. There is a lot of central nervous system involvement.

- It is **induced by activity and reversible** (compared to muscle weakness which is more long-term)
- The is a **CNS output** to this which can increase **motivation** depending on the circumstances; but the complexity of the fatigue process will eventually overwhelm this
- Risk factors for exercise fatigue: being elite because of higher workload and competitiveness which can drive them into trouble (and ignore the central response to fatigue)

Study: Cyclists are told to go as hard as possible when given Fentanyl to **block the afferent response** from muscles

- **When feedback is reduced, they go much harder initially** (compared to placebo with afferent response form muscle to brain) and then they fatigue due to lactate increase (but they could still kick at the end like normal person which highlights importance of brain in this regard)
- Without the afferent response from muscle, it releases a sort of "brake" in place by the brain because the brain does not know the muscle is fatigued without the afferent input

The activation of the membrane potential (depolarisation); the N+/K+ pump is working to restored RMP but because the muscle activation is happening so rapidly, **there is increased loss of K+** because it cannot keep up. This can cause **exertional hyperkalaemia**.



- This can change excitability and reduce Ca2+ release from SR and reduced force
- There can cause **increased risk of cardiac events**
- Losing ATP will also mean you cannot maintain energy turnover

During strenuous exercise (1.5 hours): muscle glycogen drops, lactate increases then decreases, increased AMP because it cannot be re-synthesised into ATP, causes activation of an enzyme resulting in **production of IMP**)

- The **type I fibres have run out of glycogen after 40 minutes**, so you now rely on type II fibres which are fatigable
- Endurance athletes tend to glycogen load to maintain muscle use by increasing the finite amount
- **CHO ingestion also improves exercise capacity (it can delay but not prevent fatigue);** even though blood glucose is normal, they will still fatigue

Strategies to enhance fatigue resistance:

- Training – physical, technical, mental
- Nutrition – CHO, fluids, protein
- Heat acclimatisation / pre-cooling
- Drugs / supplements / gene doping ?