

## Pre-exercise Screening

<b>Pre-exercise screening</b>	
Evidence based system identifying + managing health risks for exercise. 3 stages	
<b>Stage 1</b>	Identify individuals w/ known diseases, signs/symptoms disease who may be at risk adverse events during exercise. Self-administered questionnaire
<b>Stage 2</b>	Administered by health professional, identifies individuals w/ risk factors/ conditions to assist correct exercise prescription . measures family history, smoking status etc
<b>Stage 3</b>	Administered by health professional, used to calculate risk factor, measures things like BMI, BP etc
<b>Risk factors</b>	Age, smoking status, family history, physical inactivity, other (BP, BMI, cholesterol, blood sugar) If overall score less 2= low risk= continue exercise

<b>Exercise intensity guidelines</b>			
Intensity	HR	Perceived exertion RPE	Description
Sedentary	<40% max	RPE <1	Sitting/lying, little movement
Light	40 <55% max	RPE 1-2	Aerobic activity causing no change breathing rate, intensity sustain 60 min
Moderate	55 < 70% max	RPE 3-4	Aerobic activity has convo without un-interruption, intensity between 30-60 min
Vigorous	70 < 90% max	RPE 5-6	Aerobic activity convo cannot be maintained, intensity sustained up to 30 min
High	➤ 90% max	RPE > 7	Intensity cannot be maintained longer than 10 minutes

## Skeletal muscle: structure and function

<b>Functions</b>	Force production for movement+ breathing + postural support. Heat production, endocrine role
<b>Connective tissue</b>	<b>Epimysium:</b> surrounds entire muscle <b>Perimysium:</b> surrounds bundles of muscle fibers <b>Endomysium:</b> surrounds individual muscle fibers <b>Sarcolemma:</b> muscle cell membrane
<b>Satellite cells</b>	role muscle growth + repair (increase # nuclei in damaged fiber). More nuclei= bigger size= hypertrophy

<b>Microstructure skeletal muscle</b>	
<b>Muscle fiber</b>	Long cylindrical cell containing sarcoplasm, myofibrils
<b>Z disc</b>	Anchor thin filaments, separate one sarcomere from next

<b>a-band</b>	Thick filaments
<b>I band</b>	Made actin, z line passes through middle
<b>M line</b>	Protein structure holds thick filaments together at centre sarcomere

<b>Neuromuscular junction</b>	
Gap between motor neuron + muscle fiber, site where actin + myosin interaction occurs	
<b>Sarcoplasmic reticulum + transverse tubules</b>	
SR houses calcium + transverse tubules= tubes allow travelling AP to contract/relax muscle fibers	

<b>Sliding filament model</b>	
Muscle shortening= movement actin filament over myosin forming cross bridges between the 2 filaments= reduces distance between z-lines of sarcomere	

<b>Relationship between troponin, tropomyosin, myosin and calcium</b>	
Calcium= needed to bind + move troponin away from myosin Hydrogen ions can impede calcium binding process by competing for site of binding= myosin can't attach = loss of power/no contraction	
<b>Energy for muscle contraction</b>	
ATP required for muscles to contract, myosin ATPase breaks down ATP as fibers contract producing ADP + phosphate	
<b>Sources ATP for contraction</b>	
Phosphocreatine (stored muscle), glycolysis (breakdown glycogen stored in liver), oxidative phosphorylation (breakdown carbs fats proteins).	

<b>Muscle excitation, contraction and relaxation</b>	
<b>Excitation</b>	Nerve impulse arrives NMJ travels down transverse tubules to SR
<b>Contraction</b>	AP reaches SR, calcium is released + diffuses into troponin Tropomyosin removed from active sites on actin, cross bridge of myosin + actin formed
<b>Relaxation</b>	Absences nerve impulse NMJ= $Ca^{2+}$ pump removes calcium back to SR= tropomyosin moves and covers actin, cross bridge formation ceases

<b>Muscle fatigue</b>	
Decline in power output. At high intensity= accumulation hydrogen ions= diminished corss bridges= cannot maintain power output Low intensity/long durations= accumulation free radical, electrolyte imbalances, glycogen depletion	
<b>Exercise related muscle cramps</b>	
Spasmodic involuntary contractions. 2 possible causes: Electrolyte depletion Neuromuscular control theory= afferent signals do not come back to tell muscles to relax= continuous contractions= spasms	

<b>Characteristics muscle fibers</b>	
<b>Biomechanical properties:</b>	Oxidative capacity= # capillaries surrounding muscle fibre, # mitochondria, # myoglobin Type myosin ATPase= regulates speed of ATP degeneration i.e. type 1= break down is slow
<b>Contractile properties:</b>	Maximal force production, speed of contraction, muscle fiber efficiency
<b>How are skeletal muscle fibers typed</b>	
Muscle biopsy: Small piece tissue removed, then is stained for type of myosin ATPase (T1= darkest colour).	
<b>Characteristics individual fibers</b>	
Shifts in fiber type may occur, resistance training increase amount Type 2 X and reduce type 1. Detraining can reduce % type 1	
<b>Type 1</b>	Slow twitch, slow oxidative
<b>Type 2 A</b>	Intermediate fibers, fast-oxidative glycolytic fibers
<b>Type 2 X</b>	Fast twitch, fast glycolytic fibers

<b>Muscle actions</b>	
<b>Isometric</b>	Exerting force without changing muscle length
<b>Isotonic</b>	Shortening of muscle (bicep in bicep curl) Lengthening of muscle during movement (tricep in bicep curl)
<b>Concentric</b>	
<b>Eccentric</b>	

<b>Speed of muscle actions</b>	
<b>Twitch</b>	Single stimulus, time phases vary among people
Latent period	1 <sup>st</sup> stage, lasts approx 5ms
Contraction	2 <sup>nd</sup> phase, lasts 40ms
Relaxation	Final phase, lasts 50ms
Speed shortening greater in fast fibers= SR releases Ca <sup>++</sup> at faster rate + higher ATPase activity in comparison	

<b>Force regulation in muscle</b>	
<b>#/type motor units</b>	More units recruited- greater force. Fast fibers exert greater specific force than slow
<b>Initial length</b>	Ideal length for force, 60% lengthening greater force than 100% stretched
<b>Natural stimulation</b>	3 stimulations: simple, summation and tetanus Simple= first few contractions Summation= increase neural stimulation decreasing relaxation period Tetanus= further increase stimuli, individual contractions blended into 1 sustained contraction

**Contractile history**

If muscle first performs bouts of fatiguing movements=  
subsequent muscle force decreased

**Force-velocity relationship**

Force exerted by muscle, velocity is greater in muscle containing higher % fast fibers

**Force power relationship**

Peak power generated greater in muscle that contains high % fast fibers. Increases with velocities of movement up to 200-300 degrees/ second

**Diseases + ageing on muscle function**

Aging associated w/ muscle loss + mass. Aging also results loss fast fibers, they take on type 1 characteristics instead

Diabetes, cancer, muscular dystrophy all related with decrease muscle mass