OPEN ACESS
Advances in Health and Exercise

REVIEW ARTICLE

Physiological basis of adaptation through super-compensation for better sporting result

Kishore Mukhopadhyay

[1] Department of Physical Education, Union Christian Training College, Berhampore, Murshidabad, West Bengal, India.

Received:
November 05, 2021

Accepted:
December 12, 2021

Published:
December 30, 2021

Abstract

Every sports person tries to excel in competitive events. Excellence in any sport largely depends upon scientific training based on physiological principles. Upsetting the homeostasis of an individual regularly through external stimuli or training load adaptation takes place based on load fatigue theory and other factors. The importance of recovery after a workout session plays a vital role in super compensation of stresses. Super compensation is only possible when the body has fully recovered from the previous session following Selye's theory of "GASS". The higher the degree of adaptation to a training process, the higher will be the potentiality of the performance of an athlete. The present research article deals with the various aspects associated with super compensation and the process of adaptation of training load for betterment in sporting outcomes.

Keywords: Adaptation, sport, super compensation.

Introduction

In the time of modern technologies dominated by an in modern global sporting events, every athlete is striving hard for excellence. Excellence in sports largely depends upon the scientific administration of sports training principles. In sports training, the core concern is the appropriate loading procedure. A variety of factors affect the body's response to training load. Excessive training leads the athlete at risk for injury, illness, and decreased performance. In some cases, under-training for the expected demands of an activity and/or competition may also leave an athlete susceptible to injury and decreased performance. Overload occurs when the balance between external load and internal load is altered so that the body's adaptive capacity is inadequate, resulting in manifestations of altered performance and injury and/or illness.

Current concepts explain more accurately the features related to loading, overload, and recovery. Athletes respond individually with an internal and external load; it, therefore, determines the magnitude of the stimulus of load (Herring et al., 2015; Stanley et al., 2019).

External responsibility is the active participation of the athlete during training and competition. External loads create physical, physical, psychological, and social demands, which are affected by frequency, intensity, and duration of exercise, among other factors. These requirements may apply to certain games (Table 1).

Internal loads are individual, physical, psychological, and social factors that respond to external loading. The same external stimulus may trigger very different internal loads in two athletes with different internal characteristics (e.g., age, gender, body composition, fitness, range of motion (ROM), and history of previous injuries, mental health, and well-being). Internal loads vary over time, requiring continuous monitoring (Table 2).

Rehabilitation is the process of making the top condition of an athlete's internal balance reach homeostasis. The goal of the recovery process is to improve physical, physical, and mental adaptation to internal and external loads (Table 3).

Medical problems may arise due to too much load, too intense training, or increased training intensity of athletes who are not well prepared or conditioned to accept the loading stimuli. Athletes at different levels respond to the same stimuli in different ways.

✉ M. Mukhopadhyay, e-mail: kishore.km2007@gmail.com
To Cite: Mukhopadhyay, K. (2021). Physiological basis of adaptation through super-compensation for better sporting result. *Adv Health Exerc*, 1(2), 30-42.

Fitness-Fatigue Theory:

The prevailing theory of adaptation depends upon the fitness-fatigue paradigm. According to this theory; the sports person's preparedness in any sporting event is determined by the summation of two after effect phenomena i.e. after- effects of training: fatigue and fitness. The super-compensation theory is based on the cause and effect relationship of these important factors. The response of fitness and fatigue indicates the opposite effect on an athlete. This can be minimized through programme design and implication. A proper strategic framework is necessary for optimizing the preparedness of an athlete that maximus the fitness responses to training load with minimizing fatigue. An athlete's preparedness is determined by the positive (fitness) and negative (fatigue) responses based on the theory of super-compensation as described by Zatsiorsky (1995). This theory is termed the two-factor theory of supercompensation of training load.

Table 1

External loads create physical, physiological and psychosocial demands (Stanley et al., 2019).

Exercises	Physiological	Psychosocial
Selected examples		
Jumping	Aerobic/anaerobic	Aesthetic sports
Running	Competition frequency	Coaching dynamic
Throwing	Anaerobic	Sociocultural context
Off-season conditioning	Environmental	Performance pressure

Table 2

Internal loads are individual characteristics that respond to external load (Stanley et al., 2019).

Exercises	Physiological	Psychosocial
Selected examples		
Endurance-Fatigue	Aerobic/anaerobic	Mood states
Strength	Heat response, Metabolic function	Stress responses
ROM	Endocrine status	Self-efficacy
Skill mechanics	Heart rate	Stress responses

Table 3

Recovery optimizes adaptation to internal and external loads (Stanley et al., 2019).

Exercises	Physiological	Psychosocial
Selected examples		
Joint stability	Heart rate/HR variability	Mood states
Tissue healing	Blood markers	Motivation
ROM	Hormonal changes	Sleep patterns
Strength	Rate of perceived exertion Immunologic markers Nutritional status	Social support

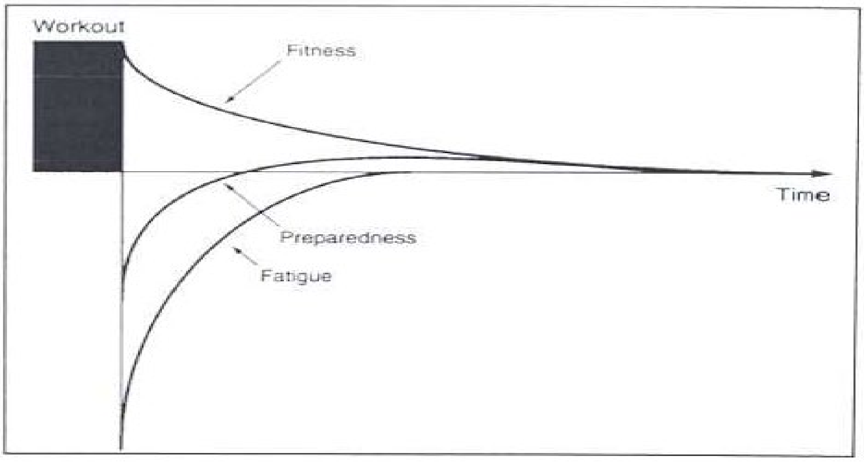

Figure 1. The Athlete's preparedness is determined by the summation of positive (fitness) and Negative (fatigue) responses (Zatsiorsky, 1995).

Super- Compensation

According to Selye this is achieved through a three-stage process. The first reaction in stressor is called an alarm/reaction stage in which the athlete may experience stiffness, pain, or a slight decrease in performance due to fatigue after a training session. The second stage is called the resistance phase and is when the body responds to the stressor by adapting to new stress with less pain, stamina, increased endurance at work, and improved performance. This is considered to occur at a higher rate than that required by the stressor and is termed "supercompensation". The final stage occurs when stress lasts longer than the body is able to adapt, as well as the effects of fatigue, in which the athlete may experience stiffness in training or experience symptoms of over-training (Selye, 1974) In contrast to Selye, the fitness-fatigue model considers occasional performance as a balancing act between fitness and fatigue (Rhea & Alderman, 2004). Thus a person's level of readiness is the result of an interaction between level of intensity and amount of fatigue (Plisk & Stone, 2003), while reducing the resulting fatigue (Plisk & Stone, 2003). For the neuromuscular system to be more adaptable to training or stress, volume adjustment and stamina are needed (Lorenz et al., 2010). Rising demands make the neuromuscular system adapt to increased muscle function but also have a similar increase in physical, mental, and metabolic costs of recovery. Without the corresponding changes in overload, the system does not need to adapt to pressures. Therefore, no further adaptations are needed and increases in the desired outcome will eventually stop (Rhea et al., 2002; Rhea et al., 2003). On the other hand, if the load is too high, the physiological costs will be too high and the physical readiness of the athlete's training will be included. The periodized program helps to avoid these problems because the load on the neuromuscular system varies to drive to adapt while minimizing fatigue. Making times may be beneficial because of the different additions to the performance by changing sets, repetition, exercise routine, amount of exercise, resistance, breaks, type of access, or frequency of training (Rhea & Alderman, 2004; Lorenz et al., 2010). Another additional benefit is to avoid training plains or boredom (Rhea et al, 2002; Rhea et al., 2003; Lorenz et al., 2010). A configurable planning approach based on the appropriate feedback is continuous exercise program (DAPRE) that allows for more flexible use than most traditional methods (Knight, 1979; Siff et al., 1999).

This modified protocol is an area-based approach designed to focus on strength/power, strength/hypertrophy, and hypertrophy. This method has been used successfully in both refreshments and performance-based settings and has been shown to surpass conventional methods of setting times in some cases (Mann et al., 2010). The Use of Rating of Perceived Exertion (RPE) has been shown to be a reliable session measure of intensity and special intensity of exercise within the training session (Day et al., 2004; Lins-Filho et al., 2012) The use of RPE provides a great advantage to the rehabilitation specialist as it allows the intensity of monitoring without establishing a real single rate of repetition (1RM), which is often opposed due to the healing stages. Other models exist to measure 1RM without proposing real 1RM such as Oddvar Holten Curve (Holten Institute, 2015) and other Baechle et al models used to establish a 1RM standard based on small loads led to failure (Baechle et al., 2000; Gamble, 2006).

The real basis for the periodization theory was general adaptation syndrome (Seyle, 1956). According to this model, the first stage of response to any stress is seen as a shock or alarm (Wathen et al., 2000; Brown & Greenwood, 2005). Following this stage, a new phase of supercompensation arises, in which the body adapts to the conditions to develop certain abilities affected by a particular stressor (Wathen et al., 2000; Brown & Greenwood, 2005). Over time if the stressor persists the body may enter a terminal phase, called maladaptation or fatigue (Wathen et al., 2000; Brown & Greenwood, 2005). Periodization was developed with the intention of counteracting these effects of addiction and avoiding the maladaptation phase, which could put the athlete in a state of over-training (Stone et al., 1981; Wathen et al., 2000).

This GAS paradigm has always been refined and successfully replaced by a fitness fatigue model (Chiu & Barnes, 2003; Plisk & Stone, 2003). An important difference is that the model of fitness fatigue distinguishes between the stressor actions provided in the individual neuromuscular and metabolic systems (Chiu & Barnes, 2003). Another major development is that the model describes a dual dynamic response that leads to both fitness and fatigue outcomes, as opposed to one common response defined by GAS. It is the net result of these two opposing effects that determine the athlete's neuromuscular and metabolic system at a given time (Chiu & Barnes, 2003) leads to both fitness and fatigue outcomes, as opposed to one common response described by GAS. These responses to fitness and fatigue work in opposition and are defined as having defined characteristics, with differences in both magnitude and duration (Chiu & Barnes, 2003).

The higher the degree of adaptation to a training process, the higher will be the potentiality of the performance of an athlete. Therefore, the objective of any well-organized training plan is to induce adaptations that improve performance. Improvement is possible only if the athlete follows the following sequence.

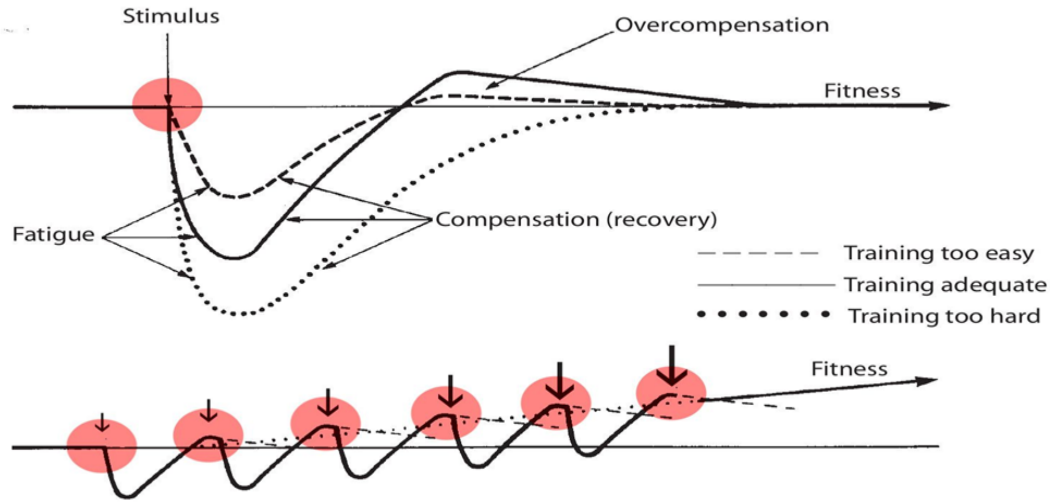

Figure 2. Stimulus-Fatigue and over-compensation (https://useful.coach/articles/heart-rate-variability-hrv-for-training-and-recovery/)

Increasing stimulus (load) ⟹ adaptation ⟹ performance improvement.

If the load is always at the same level, adaptation occurs in the early part of training, followed by a plateau (stagnation) without any further improvement:

Lack of stimulus ⟹ plateau ⟹ lack of improvement.

If the stimulus is excessive or overly varied, the athlete will be unable to adapt and Mal-adaptation will occur:

Excessive stimulus ⟹ mal-adaptation ⟹ decrease in performance.

Therefore, the objective of training is to progressively and systematically increase the training stimulus (the intensity, volume of training loads, and frequency of training) to accelerate adaptation and, as a result, improve the performance capacity. These alterations in the training stimulus must include training variation to maximize the athlete's adaptation in training (Bompa & Haff, 2009).

When adequate Training Load (TL) is regularly applied (frequency), along with adequate recovery (compensation), positive metabolic, cardiovascular, and neuromuscular adaptation occurs. Progressive increases in TL are required to stimulate progressively greater adaptation over time. Overcompensation (also called supercompensation) results from consistently applying TL above previous adaptation levels.

Apart from specific training blocks where the object is to acutely overreach (via excessive TL relative to recovery), TL is applied following adequate recovery, which provides greater training capacity, allowing for greater training volume, leading to greater adaptation over time (Figure 2) (https://useful.coach/articles/heart-ratevariability-hrv-for-training-and-recovery/).

Supercompensation is only possible when the body has fully recovered from the previous session. And that's where periodization training gets important.

Reducing the workload in the days after a hard training session gives the body time to adapt so that in the next cycle it can tolerate a bigger load. This allows athletes to build on the adaptation effect and grow stronger over time – in an 'upward spiral' kind of way.

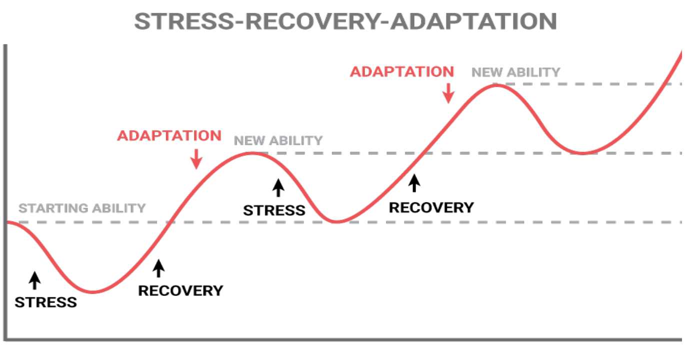

Figure 3. Stress-Recovery and Adaptation Cycle (https://fytt.io/blog/the-athletic-performance-curve/)

One of the most amazing things about humans (and all organisms really) is our ability to adapt. Our tissues are malleable and reactive, and our DNA provides our bodies with biological algorithms that can respond to a wide array of circumstances. This adaptive capability is the core mechanism of fitness. In order to promote adaptation and improve fitness, we need to create the right physical conditions that will trigger the body's adaptive processes. But one can't force or "confuse" the body to drive improvement. One must follow a logical program that progresses along the athletic performance curve to properly optimize the stress-recovery-adaptation cycle (https://useful.coach/articles/heart-rate-variability-hrv-for-training-and-recovery/).

Phases of Supercompentation

The supercompensation cycle comprises of four stages:

Phase 1. Exercise-induced fatigue Stage

Reducing neural muscle function, which is often associated with central fatigue, may be possible in response to exercise (Davis et al., 2003).

• Exercise induces central fatigue caused by exercise may increase serotonin levels in the brain, which can lead to mental fatigue (Garrandes et al., 2007).

• Exercise can cause impairment in neuromuscular transmission, decrease release of Ca2 + by sarcoplasmic reticulum, substrate depletion, and other factors that disrupt the contraceptive process and are associated with peripheral fatigue caused by exercise (https://useful.coach/articles/heart-rate-variability-hrv-for-training-and-recovery/).

• Substrate use caused by exercise occurs due to intensity, volume, and duration of exercise. The most affected substrates include muscle glycogen stores and phosphocreatine. Muscle glycogen can be significantly reduced in response to high-intensity interval training (Dal Monte, 1983; Burgomaster et al., 2006), resistance training (Haff et al., 2000; Stone et al., 2004), and endurance training (Costill et al., 1973; Macdougall et al., 2009). Phosphocreatine stores can be significantly reduced by 5 to 30 seconds and can be eliminated after a completely exhaustive workout (Hirvonen et al., 1987; Coyle, 2000).

• Ancient literature suggests that increased levels of lactic acid as a result of exercise are a major factor in fatigue formation (Karlsson et al., 1972). It is thought that high levels of lactic acid formation cause acidosis, which may reduce energy production due to changes in contractile properties (Karlsson & Ollander, 1972; Westerblad et al., 2002)

• During prolonged exercise there is an increase in glucose intake despite a decrease in circulating insulin levels (Tomlin & Wenger, 2001). Glucose uptake is thought to be facilitated during exercise as a result of glucose transporter-4 (GLUT4) (Westerblad et al., 2002). GLUT is sensitive to accessibility and facilitates glucose uptake by active tissues (Kjaer et al., 1991).

• During exercise, whether endurance training or resistance training, important components of eccentric exercise can cause muscle damage (Suh et al., 2007). Examples of strength exercises that increase muscle damage, leading to early onset of muscle pain (Kjaer et al., 1991; Suh et al., 2007), have been hypothesized that inflammation associated with muscle injury plays a role in muscle repair (Garcia-Lopez et al., 2006) and cause of muscular hypertrophy.

Phase II. Compensation phase:

As soon as the training is completed the compensation phase (rest) begins. During the compensation phase, the following occurs:

• Within 3 to 5 minutes of stopping exercise, ATP stores are completely restored (Macintyre et al., 2001; Close et al., 2005), and within 8 minutes PCr is fully reassembled (Heidt et al., 2000). Extreme exercise may require up to 15 minutes of recovery after exercise for PCr to be fully restored (Macintyre et al., 2001). Depending on the volume, intensity, and type of training, ATP and PCr pool may be increased above normal levels (Hultman & Sjøholm, 1986; Mccann et al., 1995).

• Within 2 hours after exercise with large components stretch-shortening cycle (SSC), such as jumping and maximum voluntary contraction (MVC) are slowly restored (Abernethy et al., 1994). However, fatigue caused by SSC induced as indicated by depressed EMG and MVC show bimodal recovery, the first recovery occurs within 2 hours and the final recovery takes 6 to 8 days (Abernethy et al., 1990).

• Muscle glycogen is usually restored to baseline levels within 20 to 24 hours (Burke & Deakin, 2000; Nicol et al., 2006). In the event of severe muscle damage, more time is needed to acquire muscle glycogen (Coyle, 1991). The rate at which muscle glycogen is restored is directly related to the amount of carbohydrate consumed during compensation (Costill et al., 1990). Increased oxygen consumption following exercise, known as post-exercise oxygen consumption (EPOC), occurs in response to an exercise bout (Nicol et al., 2006). Depending on the method and intensity of the exercise bout, EPOC can remain elevated 24 to 38 hours after stopping exercising (Costill et al., 1981; Laforgia et al., 2006).

• The resting energy expenditure increases as a result of resistance training or endurance training. This increase in energy expenditure can be expected to take 15 to 48 hours depending on the size of the training bout (Burleson et al., 1998; Mcmillan et al., 1993). Although the exact mechanism of action to promote elevation in the use of resting energy is unknown, some authors have suggested that increased protein synthesis (Burleson et al., 1998), increased thermogenesis from the thyroid hormones (Jamurtas et al., 2004), and increased activity of the sensory nervous system (Melby et al., 1993) play a role for increment of the rate of energy expenditure after exercise.

• After resistance training the rate of protein synthesis occurs (Macdougall et al., 1995; Lebon et al., 2001). 4 hours after exercise the level of muscle protein synthesis increases by 50%, and in 24 hours it increases by 100%. The level of protein resynthesis returns to baseline by 36 (Pratley et al., 1994) hours. Therefore, it is thought that this phase of the supercompensation cycle is the initiation of the anabolic phase.

Phase III. Rebounding Phase

This phase of training is marked by a rebounding or supercompensation of performance.

•Force-generating capacity and muscle soreness have returned to baseline by 72 hr post exercise (Chesley et al., 1992).

•Psychological supercompensation occurs, which can be marked by high confidence, feelings of being energized, positive thinking, and an ability to cope with frustrations and the stress of training.

•Glycogen stores are fully replenished, enabling the athlete to rebound (Zainuddin et al., 2006).

Phase IV. Involution Phase

If the athlete does not use another stimulant at the right time (during supercompensation phase), and then the natural occurrence, which is a decrease in the physical benefits obtained during the main compensation phase. At 6 to 8 days after the activation of the post-stretch shortening cycle (SSC), a second replication of electromyographic compression strength and greater voluntary stretch strength occurred (Burke, 1996; Nicol et al., 2006).

Following the maximum motivation for the training session, the recovery time, which includes the main compensation phase, is approximately 24 hours. The difference in the duration of the compensation phase depends largely on the nature, intensity and volume of the training. For example, following a moderate aerobic endurance training session, maximum compensation may occur in about 6 to 8 hours. On the other hand, great work that places great demand on the central nervous system may require more there are 24 hours, sometimes 48 hours, for maximum compensation. A typical super compensation model is shown in Figure 4.

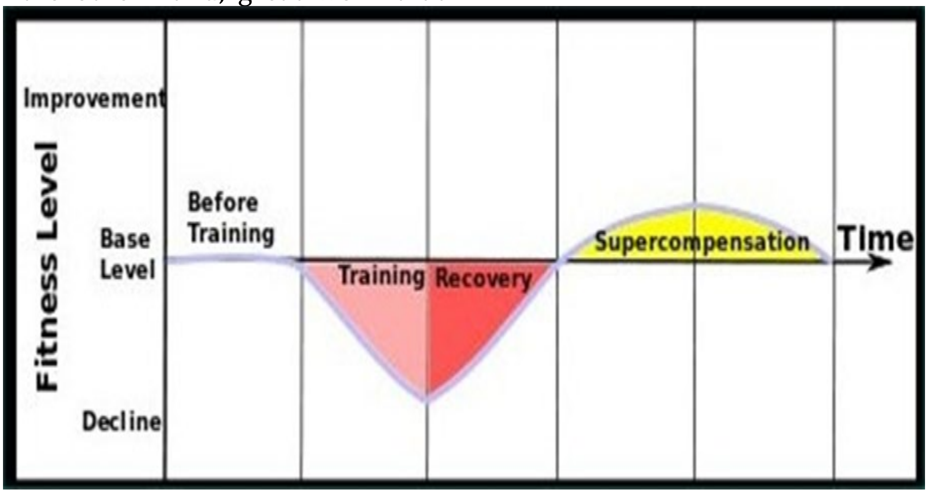

Figure 4. Super-compensation Model (Olympia Training Systems, 2011).

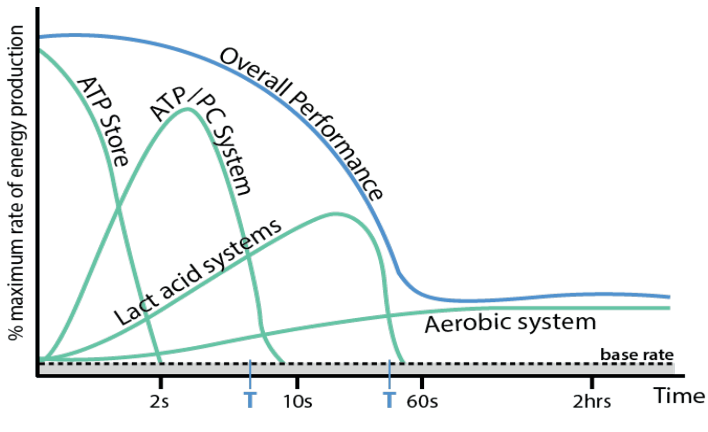

Figure 5. Time-Energy expenditure relationship (WJEC CBAC, 2021).

Physiological Consideration of Adaptation

I. Energy System :

The Carbohydrate, fat and protein breaks down in the process of digestion into simple compounds -- glucose, amino acids and fatty acids -- which are transported to various cells throughout the body. Within these cells energy nutrients converted into adenosine triphosphate (ATP) to provide fuel. The body uses 3 different systems to supply cells with the necessary ATP to fuel energy needs. Most of the body's activities use a continuum of all three energy systems, working together to ensure a constant supply of energy (Live Strong, 2021). Figure 5 represents the duration of activity corresponding to the energy system.

i. ATP-PC System

ii. **Lactic Acid System**

iii. **Aerobic System**

When someone are performing any activity, ATP-CP system is first to respond. Among other energy systems, it's the fastest, and the one most prepared for emergencies. ATP is the main source of energy, the breakdown of this highly energy compound releases energy, ADP and inorganic phosphate. This energy is required for mechanical functions of the body. In fact, the ΔG for the hydrolysis of one mole of ATP in a living cell is almost double the value at standard conditions:

i. ATP-PC System

Phosphocreatine (PCr) is another high-energy compound containing a high-energy phosphate bond that can be hydrolyzed to provide energy and resynthesize ATP with the help of creatine kinase. However, of greater quantitative importance is the utilization of phosphocreatine stored in the muscle. The millisecond you start, all three energy systems are geared to work. But the first to burn is the ATP-CP system, which burns in faster and faster rate, including adenosine triphosphate stored within your active muscles (WJEC CBAC, 2021).

This fast-moving energy system in the skeletal muscle uses a number of integrated chemical compounds to release the active cellular energy in an explosive, fast, but fast-acting form of ATP again. It does not need oxygen (anaerobic) and does not produce lactate (like glycolysis). Instead, the system incorporates ATP and creatine phosphate stored within muscle tissue. With a few enzymatic steps, the system will release energy from ATP and recombine it using creatine phosphate to produce ATP and creatine. The total dose of this one method is limited, so that during exercise, the energy from this system can continue until the creatine phosphate stores are depleted, which is likely in about ten seconds. The limiting factor of this program is less dependent on creatine phosphate that is why athletes often combine it with creatine.

i) Glycolytic System:

One alternative source is the adenylate kinase reaction, which results in ATP production from the conversion of two molecules of adenosine diphosphate (ADP) to adenosine monophosphate (AMP) and ATP. The product of glycolysis is pyruvate, and this is where the glycolytic system can be alactic, or lactic. That is, in situations where the products of glycolysis (pyruvate molecules) are exceeding the rate at which they can be shuttled into the citric acid cycle (the next phase of the energy systems), the body will bind a hydrogen to each pyruvate molecule to form lactate, which will then be synthesis back to the beginning of glycolysis to be reused. The lactate production, therefore, is both a coping mechanism (handle the excess hydrogen), and a way to create ATP in situations where the slower, more efficient system can't run its course but the demands of the body are too intense. It last for 10 seconds–75 seconds.

ii) Oxidative system

The Oxidative System comes to prominence during lower intensity, sustained exercise wherein ATP needs can be met almost indefinitely, but the production rates are not as rapid as glycolysis. Unlike glycolysis, this system is aerobic, and can be powered not only by glucose and glycogen, but by fatty acids.

This energy system is rather profound, and given that adequate substrate is available–as in, you've eaten enough–the production of ATP can last for long durations. The Oxidative System is powered by what are referred to as "high energy electron carriers," which are molecules that bond with hydrogen (threat reduction) and then create a hydrogen gradient inside mitochondrial inner membranes to power the electron transport chain–which ultimately provides the energy to resynthesize a large amount of ATP. Of all the systems, this one is most efficient at coping with hydrogen and regenerating ATP. It last for longer duration (Experience Life, 2021).

II. Muscle Fiber Type:

Each one of your skeletal muscles is made up of hundreds to thousands Trusted Source of muscle fibers that are tightly wrapped together by connective tissue.

Our muscles are comprised of different types of fibres, each with different mechanical and biological properties. These muscle fibres have been separated into two main categories based on how quickly they contract: Type I, slow twitch or Type II, fast twitch. Fast twitch muscle fibres can then be further divided into Type IIa and Type IIx fibres. To keep it simple we'll stick to the main two categories (Jason & Karp, 2004).

Each muscle fiber contains smaller units made up of repeating thick and thin filaments. This causes the muscle tissue to be striated, or have a striped appearance.

Skeletal muscle fibers are classified into two types: type 1 and type 2. Type 2 is further broken down into subtypes.

- **Type 1.** These fibers utilize oxygen to generate energy for movement. Type 1 fibers have a higher density of energy-generating organelles called mitochondria. This makes them dark.
- **Type 2A.** Like type 1 fibers, type 2A fibers can also use oxygen to generate energy for movement. However, they contain less mitochondrion, making them light.
- **Type 2B.** Type 2B fibers don't use oxygen to generate energy. Instead, they store energy that can be used for short bursts of movement. They contain even less mitochondria than type 2A fibers and appear white.

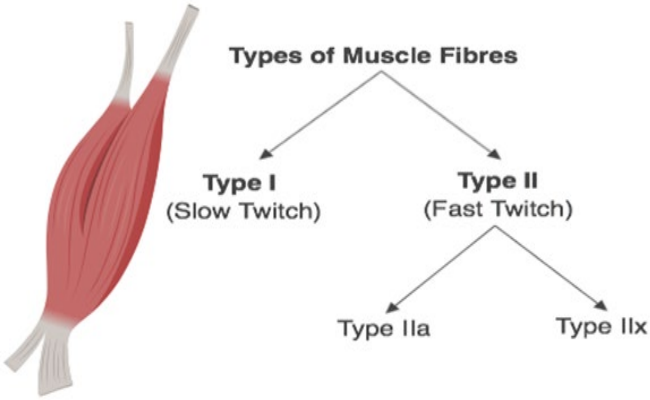

Figure 6. Types of muscle Fiber

FT and ST refer to skeletal muscle fibers. Types 2A and 2B are considered to be FT while type 1 fibers are ST. FT and ST refers to how fast muscles contract. ATP is a molecule that releases energy when it's broken down. FT fibers break down ATP twice as fast as ST fibers.

ST fibers are good for long lasting activities. These can include things like holding a posture and stabilizing bones and joints. They're also used in endurance activities, such as running, cycling, or swimming.

FT fibers produce shorter, more explosive bursts of energy. Because of this, they're good in activities involving bursts of energy or strength. Examples include sprinting and weightlifting.

Everyone has both FT and ST muscles throughout their body. However, the overall amount of each varies greatly between individuals.

FT versus ST composition can also influence athletics. Generally speaking, endurance athletes often have more ST fibres, while athletes like sprinters or power-lifters often have more FT fibres.

Table 4. Characteristics of different types of muscle fibre.

Characteristics	Muscle Fiber Types Slow Twitch	Fast Twitch a	Fast Twitch x
Aerobic (oxidative) Capacity	High	Moderate	Low
Anaerobic (glycolytic) capacity	Low	High	Highest
Myosin ATPase	Low	High	Highest
Creatine Phosphate	Low	High	Highest
Buffering capacity	Low	Highest	High
Lactate removal rate	Low	Highest	HIgh
Capillaries per fiber	High	Moderate	Low
Contractile velocity	Slow	Fast	Fastest
Fibers per motor unit	<300	>300	>300
Contractile force	Low	High	Highest
Power	Low	High	Highest

Most humans are born with approximately equal proportions of fast twitch and slow twitch muscle fibers. The FTa fibers tend to predominate within the fast twitch category, and the percentage of FTx fibers is generally lower.

III. Metabolic Adaptation:

- Increases the size and number of mitochondria in the trained muscle
- The myoglobin content may sometimes increase, thus the oxygen storage capacity increases.
- Trained muscles glycogen storage capacity increases, and the ability to use fat as an energy source (Patel & Zwibel, 2019).

IV. Cardiac Function:

When healthy individuals participate in a long term aerobic exercise programme they undergo positive cardiac adaptions, both morphologically and physiological (Joseph, 1928; Pescatello et al., 2004).

- Increased early diastolic filling and increased contractile strength.
- Morphological changes appear in both the left and the right ventricle.
- Cardiac adaptations lead to increased cardiac output while exercising, and a higher VO2max after exercise (Lavie et al., 2015).
- Post-training heart rate is decreased at rest and during sub-maximal exercise.

Stroke volume increases through long term endurance training.

- Endurance training increases plasma volume, which elevates the blood volume that returns to firstly the right heart and after that to the left ventricle.
- The greater amount of blood in circulation causes an increase in the amount of blood in the left ventricle when the end-diastolic phase is reached. The end-diastolic phase is the phase in which the passive filling (diastole) of the heart finishes.
- The left ventricle is fully filled and its wall is stretched.
- The passive stored energy in the wall helps to a forceful contraction in the emptying phase (systole).
- As a result, the heart muscle is hypertrophied.
- Each heart muscle fiber increases in size. The hypertrophy refers to the ventricle and the posterior and septal walls.

According to the American College of Sports medicine, dynamic aerobic training reduces blood pressure (BP) in individuals with hypertension.

- Hypertension is a risk factor for cardiovascular events.
- Endurance exercises lower arterial blood pressure for some hours after a bout of exercise: this phenomenon is the post-exercise hypotension.
- Post-exercise hypotension seems to be greater in people with higher pre-exercise blood pressure values.
- Blood pressure reductions occur after short bouts of exercises of 3 minutes duration and an intensity of 40% VO_2max.
- Morphological cardiac adaptations are less in people with cardiovascular disease than when compared to younger, healthy people (Lavie et al., 2015).

V. Respiratory System:

- The blood flow in the upper regions of the lungs increases after prolonged endurance training and the respiration rate increases (Spruit et al., 2016).
- Increased strength of diaphragm and intercostal muscles.
- Greater number of alveoli.
- Increased ability of the lungs to extract oxygen from the air.

- Increased vital capacity.
- Increased amount of oxygen delivered to, and carbon dioxide removed from, the body (https://www.bbc.co.uk/bitesize/guides/zmgk7ty/revision/4).

VI. Nervous System

- Long-term exercise induces significant elevation of norepinephrine and serotonin levels in different brain areas, compared to the sedentary controls.
- Exercise-induced changes in the brain morphology, chemistry and functions seem to be responsible for the beneficial effects of exercise, like improved learning and memory, anti-depressant like and anxiolytic effects, reduced cognitive decline related to ageing and improvements in symptoms of neurodegenerative diseases (Cırrık & Hacıoğlu, 2016).

Training Dose and Response

The principle of adaptation refers to the process of the body getting accustomed to a particular exercise or training program through repeated exposure. As the body adapts to the stress of the new exercise or training program, the program becomes easier to perform and explains why beginning exercisers are often sore after starting a new routine, but, after doing the same exercise for weeks and months at the same intensity, the exerciser experiences little, if any, muscle soreness. This reinforces the need to constantly vary the exercise and training routine if you want to maximize your results (Studio Pilates, 2021).

The basic rule about getting better at anything is to keep progressing the level of difficulty of the training without getting hurt or overtired. Very simple concept in theory, but it can be hard to apply in practice. Performance tends to plateau when the difference between too much and too little is so small.

Figure 7 indicate the dose response relationship can be generally (Haskell, 2001; Bouchard, 2001) suggests that most of the benefits are attained at low to moderate levels of activity. The principle of overload suggests that for students to experience an improvement in fitness (i.e., a response) the training load (i.e., the dose) must exceed that to which the individual is already accustomed. Known as the FITT Principle, the dose of physical activity is controlled by the manipulation of frequency, intensity, time (duration), and type (mode) of exercise. This principle suggests there is a dose- response relationship between physical activity and physical fitness and in order for a response to occur a specific dose threshold must be surpassed.

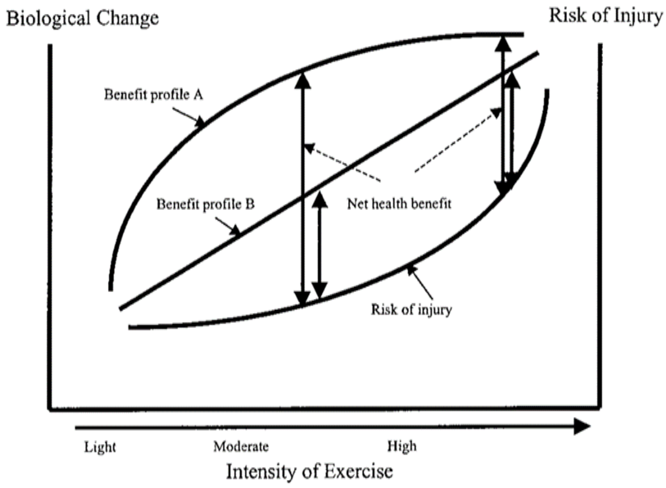

Figure 7. The relationship of exercise intensity to biological change (for two dose-response profiles) and risk injury. Net health benefit for moderate- and high-intensity exercise (96)

Conclusion

The key components of any training programme are the volume (how much), intensity (how hard) and frequency (how often) of exercise sessions. These 'training impulses' determine the magnitude of adaptive responses that either enhance (fitness) or decrease (fatigue) exercise capacity (Hawley, 2002). A long held view is that the training response/adaptation is directly related to the volume of exercise undertaken (Fitts et al., 1975). determinants of the underlying biological mechanisms that result from a wide variety of divergent exercise training protocols in association with appropriate functional outcome measures of exercise capacity is crucial in order to define the precise variations in physical activity which result in the athlete's potentiality and elevate sporting performance. Careful consideration of the use of recovery in different phases of the training program may result in optimal performance outcomes for individual and team sport athletes.

References

Abernethy, P.J., Jurimae, J., Logan, P. A., Taylor, A. W., Thayer, R. E. (1994). Acute and chronic response of skeletal muscle to resistance exercise. *Sports Med*, 17, 22-38.

Abernethy, P.J.,Thayer, R., Taylor, A. W. (1990). Acute and chronic responses of skeletal muscle to endurance and sprint exercise: A Review. *Sports Med*, 10, 365-389.

Baechle, T. R., Earle, R. W., Wathen, D. (2000). Resistance training. In: Baechle TR, Earle RW, eds. Essentials of Strength Training and Conditioning. 2nd ed. Champaign, IL: *Human Kinetics*, 395-425.

Bompa, T. O., Buzzichelli, C. A. (1973). Periodization: theory and methodology of training, *Human Kinetics*.

Bouchard, C. (2001). Physical activity and health: introduction to the dose-response symposium. *Medicine & Science in Sports & Exercise*, 33(6), 347- 350.

Brown, L. E., Greenwood, M. (2005). Periodization essentials and innovations in resistance training protocols. *Strength Cond J,* 27(4), 80–85.

Burgomaster, K. A., Heigenhauser, G. J., Gibala, M.J (2006). Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *J Appl Physiol,* 100, 2041-2047.

Burke, L. M. (1996). Nutrition for post-exercise recovery. *Aus J Sci Med Sport,* 29, 3-10.

Burke, L., & Deakin, V. (2000). Clinical sports nutrition. Roseville, Australia: *Mcgraw-Hill Australia*.

Burleson, M. A., Jr., O'bryant, H. S., Stone, M. H., Collins, M. A., Triplett-Mcbride, T. (1998). Effect of weight training exercise and treadmill exercise on post- exercise oxygen consumption. *Med Sci Sports Exerc,* 30, 518-522.

Chesley, A., Macdougall, J. D., Tarnopolsky, M.A., Atkinson, S.A., Smith, K. (1992). Changes in human muscle protein synthesis after resistance exercise. *J Appl Physiol,* 73, 1383-1388.

Chiu, L. Z. F., Barnes, J.L. (2003). The fitness-fatigue model revisited: Implications for planning short- and long term training. *Strength Cond J,* 25(6), 42–51.

Cırrık, S., & Hacioglu, G. (2016). Neurophysiological Effects Of Exercise. *Fitness Medicine*, Http://Dx.Doi.Org/10.5772/64801 .

Close, G. L., Ashton, T., Mcardle, A., Maclaren, D. P. (2005). The emerging role of free radicals in delayed onset muscle soreness and contraction-induced muscle injury. *Comp Biochem Physiol,* 142, 257-266.

Costill, D. L., Gollnick, P. D., Jansson, E. D., Saltin, B., Stein, E. M. (1973). Glycogen depletion pattern in human muscle fibres during distance running. *Acta Physiol Scand,* 89, 374-383.

Costill, D.L., Pascoe, D. D., Fink, W. J., Robergs, R. A., Barr, S. I., Pearson, D. (1990). Impaired muscle glycogen resynthesis after eccentric exercise. *J Appl Physiol,* 69, 46-50.

Costill, D.L., Sherman, W. M., Fink, W. J., Maresh, C., Witten, M., Miller, J.M. (1981). The role of dietary carbohydrates in muscle glycogen resynthesis after strenuous running. *Am J Clin Nutr,* 34, 1831-1836.

Coyle, E. F. (1991). Timing and method of increased carbohydrate intake to cope with heavy training, competition and recovery. *J Sports Sci* 9(Spec No): 29-51, Discussion 51-22.

Coyle, E. F. (2000). Physical activity as a metabolic stressor. *Am J Clin Nutr,* 72, 512-520.

Dal monte, A., (1983). The functional values of sport. Firente, Sansoni.

Davis, J. M., Zhao, Z., Stock, H. S., Mehl, K.A., Buggy, J., G.A. Hand, G. A. (2003). Central nervous system effects of caffeine and adenosine on fatigue. *Am J Physiol Regul Integr Comp Physiol,* 284, 399-404.

Day, M. L., McGuigan, M. R., Brice, G., et al. (2004). Monitoring exercise intensity during resistance training using the session RPE scale. *Journal of Strength and Conditioning Research,* 18(2), 353-358.

Fitts R. H, Booth, F. W., Winderww Holloszy J. O. (1975). *Am J Physiol* 228, 1029-1033.

Fleck, S. J., Kraemer, W. J. (2004). Designing Resistance Training Programs, ed 3, Champaign, IL. *Human Kinetics.*

Gamble, P. (2006). Periodization of training for team sports athletes. National *Strength and Conditioning Association,* 28(5), 56–66.

Garcia-Lopez, D., De Paz, J. A., Jimenez-jimenez, R., Bresciani, G., De Souza-Teixeira, F., J.A., Herrero, J. A., Alvear-Ordenes, I., Gonzalez-Gallego, J. (2006). Early explosive force reduction associated with exercise-induced muscle damage. *J Physiol Biochem,* 62(3), 163-169.

Garrandes, F., Colson, S. S., Pensini, M., Seynnes, O., Legros, P. (2007). Neuromuscular fatigue profile in endurance-trained and power-trained athletes. *Med Sci Sports Exerc,* 39, 149-158.

Haff, G. G., Koch, A. J., Potteiger, J.A., Kuphal, J.A., Magee, L. M., Green, S. B., Jakicic, J. J. (2000). Carbohydrate supplementation attenuates muscle glycogen loss during acute bouts of resistance exercise. *Int J Sport Nutr Exerc Metab,* 10, 326-339.

Haskell, W. L. (2001). What to look for in assessing responsiveness to exercise in a health context. *Med Sci Sports Exerc,* 33, 454–458.

Hawley J. A. (2002). Clin. Exp Pharmacol Physiol, 29, 218–222.

Heidt, R. S., Jr., Sweeterman, L. M., Carlonas, R. L., Traub, J. A., Tekulve, F. X. (2000). Avoidance of soccer injuries with preseason conditioning. *Am J Sports Med,* 28(5), 659-662.

Herring, S. A., Bergfeld, J. A., Boyajian-O'Neill L, et al. (2015). The team physician and strength and conditioning of athletes for sports: A consensus statement. *Med Sci Sports Exerc,* 47, 440-45.

Herring, S. A., Kibler, W. B., Putukian, M., et al. (2013). Selected issues for nutrition and the athlete: a team physician consensus statement: Update. *Med Sci Sports Exerc,* 4, 2378-86.

Herring, S. A., Kibler, W. B., Putukian, M., et al. (2016). Selected issues in injury and illness prevention and the team physician: a consensus statement. *Med Sci Sports Exerc,* 15(1) 48-59.

Hirvonen, J., Rehunen, S., Rusko, H., Härkönen, M. (1987). Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. *Eur J Appl Physiol*, 56(3), 253-259.

Holten Institute (2015). www.holteninstitute.com. Downloaded 5/29/2015.

Experience Life, 2021). https://experiencelife.lifetime.life/article/all-about-your-metabolic-energy-systems/

https://fytt.io/blog/the-athletic-performance-curve/

https://opentextbc.ca/anatomyandphysiologyopenstax/chapter/types-of-muscle-fibers/

https://pureperformancetraining.com/energysystems/

Olympia Training Systems (2011, november 06). https://sites.google.com/a/olympiatrainingsystems.com/olympia-training-systems/2--physiology-of-running/supercompensation

https://useful.coach/articles/heart-rate-variability-hrv-for-training-and-recovery/

https://www.bbc.co.uk/bitesize/guides/zmgk7ty/revision/4.

https://www.healthline.com/health/musclefibers#function

live strong, 2021). https://www.livestrong.com/article/131444-3-energy-systems-body/

https://www.slideserve.com/quynh/typicalsupercompensation-model

Studio Pilates. (2021). https://www.studiopilates.com/the-adaptation-cycles-what-they-are-and-why-theyre-important/

Hultman, E., & Sjøholm, H. (1986). Biochemical causes of fatigue. in: human muscle power. Ed. Jones, N.L. Champaign, Il: *Human Kinetics*, p. 343-363.

Jamurtas, A. Z., Koutedakis, Y., Paschalis, V., Tofas, T., Yfanti, C., Tsiokanos, A., Koukoulis, G., Kouretas, D., Loupos, D. (2004). The effects of a single bout of exercise on resting energy expenditure and respiratory exchange ratio. *Eur J Appl Physiol,* 92, 393-398.

Jason, R., & Karp, M. S. (2004). Muscle fiber types and training types of muscle fibers, track coach-4943, http://www.hurdlecentral.com/docs/strength&conditioning/karp_musclefibertypes&training.pdf

Karlsson, J., & Ollander, B. (1972). Muscle metabolites with exhaustive static exercise of different duration. *Acta Physiol Scand,* 86, 309-314.

Karlsson, J., Nordesjö, L. O., Jorfeldt, L., Saltin, B. (1972). Muscle lactate, ATP, and CP levels during exercise after physical training in man. *J Appl Physiol*, 33(2), 199-203.

Kjaer, M., Kiens, B., Hargreaves, M., Richter, E. A. (1991). Influence of active muscle mass on glucose homeostasis during exercise in humans. *J Appl Physiol,* 71, 552-557.

Knight, K. L,. (1979). Knee rehabilitation by the daily adjustable progressive resistive exercise technique. *Am J Sports Med*, 7(6), 336-337.

Kraft, J. A., Green, J. M., Gast, T. M. (2014). Work distribution infl uences session ratings of perceived exertion response during resistance exercise matched for total volume. *J Strength Cond Res,* 28(7), 2042-2046.

Laforgia, J., Withers, R. T., Gore, C. J. (2006). Effects of exercise intensity and duration on the excess post-exercise oxygen consumption. *J Sports Sci,* 24, 1247-1264.

Lavie, C. J., Arena, R., Swift, D. L., Johannsen, N. M., Sui, X., Lee, D. C., Earnest, C. P., Church, T. S., O'keefe, J. H., Milani, R. V., Blair, S. N. (2015). Exercise and the cardiovascular system: Clinical science and cardiovascular outcomes. *Circulation Research,* 117(2), 207-19.

Lebon, V., Dufour, S., Petersen, K. F., Ren, J., Jucker, B. M., Slezak, L. A., Cline, G. W., Rothman, D. L., Shulman, G. I. (2001). Effect of triiodothyronine on mitochondrial energy coupling in human skeletal muscle. *J Clin Invest,* 108, 733-737.

Lins-Filho, O de L., Robertson, R. J., Farah, B. Q., et al. (2012). Effects of exercise intensity on rating of perceived exertion during a multiple-set resistance exercise session. *J Strength Cond Res*, 26(2), 466-472.

Lorenz, D. S., Reiman, M. P., Walker, J. C. (2010). Periodization: Current review and suggested implementation for athletic rehabilitation. *Sports Health*, 2(6), 509- 518.

Lorenz, D. S., Reiman, M. P., Walker, J. C. (2010). Periodization: Current review and suggested implementation for athletic rehabilitation. *Sports Health*, 2(6), 509-518.

Macdougall, J. D., Gibala, M. J., Tarnopolsky, M. A., Macdonald, J. R., Interisano, S. A., Yarasheski, K. E. (1995). The time course for elevated muscle protein synthesis following heavy resistance exercise. *Can J Appl Physiol,* 20, 480-486.

Macdougall, J.D., Ray, S. D.G. Sale, D. G., Mccartney, N., Lee, P., Garner, S., (1999). Muscle substrate utilization and lactate production during weightlifting. *Can J Appl Physiol,* 24, 209-215.

Macintyre, D. L., Sorichter, S., Mair, J., Berg, A., Mckenzie, D.C. (2001). Markers of inflammation and myofibrillar proteins following eccentric exercise in humans. *Eur J Appl Physiol*, 84, 180-186.

Mann, J. B., Thyfault, J. P., Ivey, P. A., et al. (2010). The effect of autoregulatory progressive resistance exercise vs. linear periodization on strength improvement in college athletes. *J Strength Cond Res,* 24(7), 1718- 1723.

Mccann, D. J., Mole, P. A., Caton, J. R. (1995). Phosphocreatine kinetics in humans during exercise and recovery. *Med Sci Sports Exerc,* 27, 378-389.

Mcmillan, J. L., Stone, M. H., Sartin, J., Keith, R. , Marple, D., Brown, C., Lewis, R. D. (1993). 20-Hour physiological responses to a single weight-training session. *J Strength Cond Res,* 7, 9-21.

Melby, C., Scholl, C., Edwards, G., Bullough, R. (1993). Effect of acute resistance exercise on postexercise energy expenditure and resting metabolic rate. *J Appl Physiol,* 75, 1847-1853.

Nicol, C., Avela, J., Komi, P. V. (2006). The stretch-shortening cycle: A model to study naturally occurring neuromuscular fatigue. *Sports Med,* 36, 977-999.

Nicol, C., Avela, J., Komi, P. V., (2006). The stretch-shortening cycle: A model to study naturally occurring euromuscular fatigue. *Sports Med,* 36, 977-999.

O'halloran, P., Bhogal, G. (Eds). Exercise medicine for students: A one-stop resource for the knowledge and promotion of physical activity. Motivate2move

Patel, P. N., Zwibel, H. (2019). Physiology, exercise. instatpearls [Internet] May 5. Statpearls Publishing.

Pescatello, L. S., Franklin, B. A., Fagard, R., Farquhar, W. B., Kelley, G. A., Ray, C. A. (2004). American College of Sports Medicine position stand. Exercise and hypertension. *Med Sci Sports Exerc*, 36(3), 533-553.

Plisk, S. S., Stone, M. H. (2003). Periodization strategies. *Strength Cond J*, 25(6), 19-37.

Plisk, S. S., Stone, M. H. (2003). Periodization strategies. *Strength Cond J,* 25(6), 19–37.

Pratley, R., Nicklas, B., Rubin, M., Miller, J., Smith, A., Smith, M., Hurley, B., Goldberg, A. (1994). Strength training increases resting metabolic rate and norepinephrine levels in healthy 50- to 65-yr-old men. *J Appl Physiol,* 76, 133-137.

Rhea, M. R., Alderman, B. L. (2004). A meta-analysis of periodized versus nonperiodized strength and power training programs. *Res Q Exerc Sport,* 75. 413-423.

Rhea, M. R., Alderman, B. L. (2004). A meta-analysis of periodized versus nonperiodized strength and power training programs. *Res Q Exerc Sport,* 75, 413-423.

Rhea, M. R., Alvar, B. A., Burkett, L. N., Ball, S. D. (2003). A metaanalysis to determine the dose response for strength development. *Med Sci Sports Exerc*, 35, 456–464.

Rhea, M. R., Ball, S. D., Phillips, W. T., Burkett, L. N. (2002). A comparison of linear and daily undulating periodized programs with equated volume and intensity for strength. *J Strength Cond Res*, 16(2), 250-255.

Selye, H. (1974). Stress without distress. New York: J.B. Lippincott.

Seyle, H. (1956). The Stress of Life. New York: *McGraw-Hill.*

Siff, M. C., Verkoshansky, Y. V. (1999). Supertraining. Denver, CO: *Supertraining International*, 4th ed.

Spruit, M, A., Burtin, C., De Boever, P., Langer, D., Vogiatzis, I., Wouters, E. F., Franssen, F. M. (2016). Copd and exercise: does it make a difference? *Breathe,* 12(2), 38-49.

Spruit, M. A., Singh, S. J., Garvey, C., Zuwallack, R., Nici, L., Rochester, C., Hill, K., Holland, A. E., Lareau, S. C., Man, W. D., Pitta, F. (2013). An official American thoracic society/European respiratory society statement: Key concepts and advances in pulmonary rehabilitation. *American Journal of Respiratory and Critical Care Medicine*, 188(8), 13-64.

Stanley, A., Herring, W., Kibler, B., Putukian, M. (2019) Load, overload, and recovery in the athlete: select issues for the team physicianva consensus statement. *Current Sports Medicine Reports by the American College of Sports Medicine*, 1537-890/1804/141-148.

Stone, M. H., O'bryant, H. S., Garhammer, J. (1981). A hypothetical model for strength training*. J Sports Med Phys Fitness,* 21, 342–351.

Stone, M. H., Sands, W.A., Stone, M. E. (2004). The downfall of sports science in the United States. *Strength and Cond J,* 26, 72-75.

Suh, S. H., Paik, I .Y. Jacobs, K. (2007). Regulation of blood glucose homeostasis during prolonged exercise. *Mol Cells,* 23, 272-279.

Tomlin, D. L., & Wenger, H. A. (2001). The relationship between aerobic fitness and recovery from high intensity intermittent exercise. *Sports Med,* 31, 1-11.

Tudor, O., Bompa, G., Gregory, H. (2009). Periodization: Theory and methodology of training, *Human Kinetics.*

Wathen, D., Baechle, T. R., Earle, R. W. (2000). Training variation: Periodization. In: Essentials of Strength Training & Conditioning (2nd. ed.). Baechle and Earle (eds.). Champaign, IL: *Human Kinetics*. pp. 513–527.

WJEC CBAC (2021). http://resource.download.wjec.co.uk.s3.amazonaws.com /vtc/2015-16/15-16_30/eng/02-during-the-game/unit2-energy-systems-and-their-application%20.html)

Wearn, J. T. (1928). The extent of the capillary bed of the heart. *J Exp Med,* 47(2), 273–290.

Westerblad, H., Allen, D. G. Lannergren, J. (2002). Muscle fatigue: lactic acid or inorganic phosphate the major cause? *News Physiol Sci,* 17, 17-21.

Zainuddin, Z., Sacco, P., Newton, M., Nosaka, K. (2006). Light concentric exercise has a temporarily analgesic effect on delayed-onset muscle soreness, but no effect on recovery from eccentric exercise. *Appl Physiol Nutr Metab,* 31, 126-134.

Zatsiorsky,V. M. (1995). Science and practice of strength training. *Champaign II, Human Kinetics*.