

THE EFFECT OF *PHOSPHORICUM ACIDUM 200CH* ON THE ADVERSE PHYSIOLOGICAL EFFECTS INDUCED BY EXERCISE IN CYCLISTS

By

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Technology: Homoeopathy in the Faculty of Health Sciences at the Durban University of Technology.

I Giovanni Pantalone do hereby declare that this dissertation represents my own work in concept and
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To my family that always supported me in all my dreams and endeavours, my family for their patience and financial support

- Thank you -

ABSTRACT

Introduction

The treatment with homoeopathic remedies, namely *Phosphoricum acidum 200CH* has proved to be effective in many clinical situations; however confirmation of its use within the sporting industry is limited. This study aimed to test *Phosphoricum acidum 200CH* efficacy in treating highly trained cyclists. This study was a superiority trial, in which the homoeopathic intervention was hypothesized to be superior to the placebo control group, in a statistically significant way ($p < 0.05$).

Aim

This study aimed to test the use of *Phosphoricum acidum 200CH* in treating the adverse physiological and psychological symptoms induced by exercise on cyclists. The aim for treating these adverse symptoms, induced by exercise, is to enhance performance and recovery of cyclists. Performance and recovery were tracked by assessing blood lactate concentration, oxygen consumption rate, heart rate, peak power output and emotional status.

Methodology

The study was a randomized controlled clinical trial, parallel group design. Participants were selected using convenience sampling of male road and mountain bike cyclists in the Western Cape. This study followed an explanatory Randomized Controlled Trial test, where the efficacy of the homoeopathic remedy (*Phosphoricum acidum 200CH*) was under investigation. The participants were selected with great care and testing was completed under highly controlled conditions.

Thirty competitive male cyclists volunteered for this study. All participants were required to complete two cycling power to exhaustion interval tests, consisting of a ten minute warm-up at 100 Watts, followed by a five minute constant load at 150 Watts. The purpose of the constant load was to test cycling economy (CE). Thereafter the workload was increased to 200 W for 30 seconds and then the workload was increased by 20 Watts every 30 seconds. The test continued until the participant could no longer maintain the set repetitions per minute for that workload. The peak power output attained

was recorded. The first test served as a baseline, after which a single dose of *Phosphoricum acidum 200CH* or identical placebo was administered, a 30 minute recovery period allowed for the remedy to take effect. The test interval was then repeated, the second test results were then compared to the first baseline test to determine the effect of treatment. The study took many different aspects of the remedies action on the cyclists into account, including mental and physiological effects.

Breath-by-breath gases were continuously recorded. Expired gases, volumes and air flow were sampled through a flow meter and gas sampling line and heart rate was measured through telemetry (*Polar®*, Polar Electro, Oy, Finland) and analyzed by a cardio-pulmonary metabolic system (*Quark CPET®* Cosmed, Rome, Italy, 2009). Data recorded was filtered for values outside the normal ranges and averaged for every five seconds. Oxygen consumption ($\dot{V}O_2$ mL.min⁻¹) and heart rate (bpm) at different stages of each test interval were recorded. More specifically oxygen consumption and heart rates were averaged over the 5 minute section following the warm-up to determine cycling economy, directly after exhaustion for one minute and two minutes following exhaustion for another one minute period. Maximum attained heart rate was recorded for each interval test. Maximum oxygen consumption ($\dot{V}O_{2Max}$ Absolute) was calculated as the mean of the highest three values attained, this mean was then divided by the participants body mass to determine maximum aerobic capacity ($\dot{V}O_{2Max}$ Relative). Blood lactate levels were tested before, 15 minutes into and directly after each interval, to assess resting lactate status, cycling economy and to determine maximum lactate accumulation.

Psychological testing included mood analysis, using a Stellenbosch mood scale (STEMS) questionnaire and further symptoms were analyzed using a Numerical rating scale (NRS) with symptomatic questions.

Results

From the results, it was clearly apparent that the control group proved to be of a higher calibre when comparing performance variables of the two groups namely:

- Higher peak power output
- Higher $\dot{V}O_{2Max}$
- Lower economy lactate

- Lower resting lactate

Despite the treatment group being the weaker of the two groups, they showed improvement in performance after administration of the remedy. This improvement was manifest through physiological alteration in the second test. More exclusively is the acceptance of the hypothesis concerning heart rate and oxygen consumption, whereby results showed that the administration of *Phosphoricum acidum 200CH* decreased heart rate and submaximal oxygen consumption rates during performance and recovery. There was no observable psychological effect during this study. The results suggest that *Phosphoricum acidum 200CH* primarily demonstrated physiological effects on the cyclists. The researcher believes that this is due to insufficient time given for psychological alterations.

Conclusion

The *Phosphoricum acidum 200CH* has proven to be effective in enhancing cycling economy, reducing maximum heart rate and enhancing recovery to a large degree for the first minute following exhaustion. These positive effects are of great importance as the treatment group was the weaker of the two groups. Resulting in the possibility of even larger results being observable in repeated studies where both groups have similar performance abilities.

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LIST OF ABBREVIATIONS

Upcase ^N	:	Negligible Effect
Upcase ^S	:	Small Effect
Upcase ^M	:	Medium Effect
Upcase ^L	:	Large Effect
Upcase ^{VL}	:	Very Large Effect
Upcase ^T	:	Treatment Group
Upcase ^C	:	Control Group
%Δ	:	Percentage Change
*	:	Significant Difference ($p < 0.05$)
0-1 min	:	Average One Minute Post Exhaustion
2-3 min	:	Average Two To Three Minutes Post Exhaustion
a-vO ₂	:	Arterial Venous Difference
ADP	:	Adenosine Diphosphate
ANOVA	:	Analysis Of Variance
ATP	:	Adenosine Triphosphate
BDC	:	Bottom Dead Centre
BMI	:	Body Mass Index
Bp	:	Blood Pressure
bpm	:	Beats Per Minute
CE	:	Cycling Economy
CH	:	Dilution Scale Using The 1:100 Ratio
cm	:	Centimetres
CNS	:	Central Nervous System
CO	:	Cardiac Output
°C	:	Degrees Celsius
<i>d</i>	:	Cohen's <i>d</i>
DOMS	:	Delayed Onset Of Muscle Soreness
Econ	:	Economy
ECW	:	Extra-Cellular Water
e.g.	:	For Example
<i>et al.</i>	:	And Others
GH	:	Growth Hormone
HR	:	Heart Rate
H ⁺	:	Hydrogen Ions
hr(s)	:	Hour(s)
kg	:	Kilogram(s)
km	:	Kilometre(s)
LBM	:	Lean Body Mass
m	:	Meters
M	:	Deconcentration level where 10 ⁻²⁰⁰⁰ of the original substance

	:	is present in the potency
METS	:	Metabolic Equivalents
Max	:	Maximum
ml	:	Millilitres
n	:	Number Of Participants
NRS	:	Numerical Rating Scale
NSAIDS	:	Non-Steroidal Anti-Inflammatory Drugs
O ₂	:	Oxygen
OBLA	:	Onset Of Blood Lactate Accumulation
OTS	:	Over Training Syndrome
p	:	Probability Value
PCr	:	Phosphocreatine
pH	:	Hydrogen Ion Concentration
P _i	:	Inorganic Phosphate
POMS	:	Profile of Mood States
PPO	:	Peak Power Output (W)
PQRS	:	Peculiar, Queer, Rare Or Strange Symptoms
RBC	:	Red Blood Cell(s)
RER	:	Respiratory Exchange Ratio
rpm	:	Revolutions Per Minute
SD	:	Standard Deviation
SEM	:	Standard Error of Measurement
STEMS	:	Stellenbosch Mood Scale
TDC	:	Top Dead Centre
VAS	:	Visual Analogue Scale
vO ₂	:	Volume Of Oxygen Consumed (ml.min ⁻¹)
vO _{2Max}	:	Maximum Oxygen Consumption Volume (ml.min ⁻¹ ; ml.kg ⁻¹ .min ⁻¹)
% vO _{2Max}	:	Fractional Utilization Of Oxygen (%)
W	:	Watt (s)
\bar{x}	:	Mean

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CHAPTER ONE: INTRODUCTION

In striving to be the best, athletes push themselves harder to improve performances, to achieve faster times and to acquire more power. Various strategies have been employed to improve performance and enhance recovery, allowing athletes to push their bodies harder during training and competitions (Gardner & Moore, 2004).

Various homoeopathic remedies, namely *Arnica montana*, *Rhus toxicodendron* and *Ruta graveolens*, have been successfully used to treat sports related injuries, and act as recovery aids. However, limited scientific (Inch, 2001) research has been conducted using the homoeopathic remedy *Phosphoricum acidum* and its effects on improving recovery and enhancing performance on cyclists. This study endeavoured to investigate the possible influence of *Phosphoricum acidum 200CH* on highly trained cyclists during and after, two cycling interval tests. This study took many different aspects of the remedy's effect on the cyclists into account, including mental and physiological effects.

Thirty competitive male cyclists (mean age: 30 years; mean height: 175 cm; mean body mass: 76 kg; mean $\dot{V}O_{2\text{Max}}$: $55.8 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) volunteered for this study. All participants were required to complete a warm-up at 100 Watts for ten minutes, followed by a five minute constant load at 150 Watts. The purpose of the constant load was to test cycling economy (CE). Thereafter the workload was increased to 200 W for 30 seconds and then the workload was increased by 20 Watts every 30 seconds. The test continued until the participant could not maintain the set repetitions per minute for that workload. The peak power output attained was recorded. Each cyclist acted as his own control, and repeated the testing protocol after a 30 minute passive recovery. Half the group received a placebo to act as a further control and half received the homoeopathic intervention *Phosphoricum acidum 200CH*.

Testing included mood analysis, using a Stellenbosch Mood Scale (STEMS) questionnaire. Further symptoms were analyzed using a Numerical rating scale (NRS) with symptomatic questions. Blood lactate levels were tested 15 minutes into each interval, to assess cycling economy and directly after each interval, to determine maximum lactate accumulation.

Aim:

- To test the effect of *Phosphoricum acidum 200CH* on the recovery rate of cyclists.
- To test the effect of *Phosphoricum acidum 200CH* on the blood lactate concentration of cyclists.
- To test the effect of *Phosphoricum acidum 200CH* on oxygen consumption of cyclists.
- To test the effect of *Phosphoricum acidum 200CH* on peak power output of cyclists.
- To test the effect of *Phosphoricum acidum 200CH* on the emotional status of cyclists.

Hypotheses:

- The administration of *Phosphoricum acidum 200CH* would decrease blood lactate accumulation in the body.
- The administration of *Phosphoricum acidum 200CH* would increase peak power output of cyclists.
- The administration of *Phosphoricum acidum 200CH* would decrease submaximal oxygen consumption during performance and recovery.
- The administration of *Phosphoricum acidum 200CH* would increase maximal oxygen consumption during performance.
- The administration of *Phosphoricum acidum 200CH* would decrease heart rate during performance and recovery.
- The administration of *Phosphoricum acidum 200CH* would decrease lower limb muscle pain, lower limb muscle stiffness, nausea, headache and dizziness symptom severity.
- The administration of *Phosphoricum acidum 200CH* would decrease negative emotions (anger, confusion, depression, fatigue, tension) and increase positive emotions (vigour) induced by cycling.
- The administration of *Phosphoricum acidum 200CH* would improve the recovery rate of cyclists.

Assumptions:

- Athletes would perform at their peak ability.
- Athletes would answer all questions truthfully.
- Athletes are physically fit to complete the test.
- Athletes would be adequately fuelled to complete the test.

- Athletes would adhere to the guidelines requested for the study, e.g. refraining from certain substances that could affect the outcome variables of the study.
- Performance changes as a result of *Phosphoricum acidum* experienced by the test subjects will be similar in all cyclists.

Delimitations:

- Only the physiological and emotional effects experienced during the testing period would be taken into account – long term effects are beyond the scope of this study.
- Physiological and genetic variability between individuals would not be taken into account as this study assumed that all cyclists would experience similar effects.
- This study only tests the effect of *Phosphoricum acidum 200CH* on cyclists. Testing its effect on other sporting disciplines lies beyond the scope of this study.
- Other potencies of *Phosphoricum acidum* will not be tested.

There may be a limitation with regards to the testing period of the study. The testing period of only one day may not be long enough for the remedy to reveal any significant effects on the tested individuals. Homoeopathic remedies are known to cause an immediate change in the body environment, as it is hypothesised that remedies alter the structure of every water molecule in the body instantaneously (Milgrom, 2007). Whether that change within the internal environment is extensive enough for the body's metabolism to respond to the received stimulus immediately is questionable, and might pose a limitation in the study.

Technology, design and development within the cycling industry have advanced far beyond expectations. Modern racing bicycles no longer hamper performance due to their light weight, rigid and aerodynamic design. The athletes themselves have become the major performance limiting factor which can be improved on. For this reason much research is focused on improving the athlete, allowing them to better utilize the technology at their disposal. This study aims to bring some solutions to the challenge of enhancing performance, through the use of the homoeopathic remedy *Phosphoricum acidum 200CH*. The effects on cyclists following this intervention were equated by measuring recovery time, cycling economy, blood lactate concentration and peak performance (Gardner & Moore, 2004).

CHAPTER TWO: LITERATURE REVIEW

A. INTRODUCTION

Cycling is one of the most popular sports in the world particularly among young male adults, transit users and those who are physically active and in good health (Moudon, Lee, Cheadle, Collier, Johnson, Schmid, & Weather, 2005). Most cyclists spend a few hours a week cycling, while highly trained cyclists often ride more than 1000 km per month and complete more than 100,000 pedal stroke repetitions per week (Chapman, Vicenzino, Blanch, & Hodges, 2008). Completing a high number of peddle strokes promotes muscle, metabolic, cardio-respiratory and neuro-endocrine adaptations. However this repetitive movement during prolonged training imposes biomechanical, physiological and psychological stress, which can result in a host of symptoms including fatigue and injury. These symptoms are often ignored or treatment plans are inappropriate (Inch, 2001). Homoeopathy is based on the principle of administering a substance to a diseased individual, which if administered to a healthy individual will cause similar symptoms (Walach, 1997). Therefore a substance that causes similar symptoms as those experienced following prolonged cycling would be the indicative treatment. It is thus suggested as a highly effective method of managing an athlete's health (Mathie, 2003; Owen, 2003).

B. PHYSICAL DETERMINANTS OF CYCLING PERFORMANCE

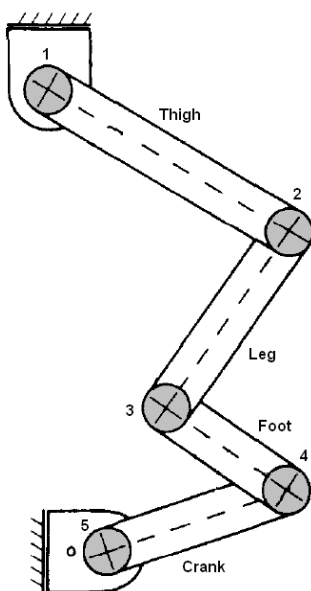


Figure 2.1 Rotational Axes
(Gonzalez & Hull, 1989)

1=hip, 2=knee, 3=ankle, 4=pedal, 5=crank

Various factors distinguish a trained cyclist from an untrained individual. The most notable of these are biomechanics, maximal aerobic capacity ($\dot{V}O_{2Max}$), cycling economy, lactate threshold and peak power output (Gonzalez & Hull, 1989).

1. Biomechanics Of Cycling

There are five rotational axes around which torque is generated during a pedal stroke. The lower extremity of the cyclist constitutes three of these axes, the hip, knee and ankle joint. The bike pedals and cranks are the other two mechanical axes. These axes are connected by the thigh, leg, foot and the crank respectively, as illustrated in *Figure 2.1*.

There are five biomechanical variables that affect cycling efficiency; pedalling rate (cadence), crank arm length, seat tube angle, seat height and longitudinal foot position on the pedal. Four of these factors are geometric variables which influence the joint angles, the fifth variable is cadence. At higher cadence, time for muscle contraction is reduced for each stroke. However, more strokes can be completed in a set time which makes up for the decreased muscular contraction time, inversely at slower cadence, more muscular contraction time but fewer repetitions.

The optimal crank arm length, seat height, and longitudinal foot position on the pedal increases as the size of a rider increases whereas the optimal cadence and seat tube angle decrease as the rider's size increases. These anthropometric parameters emphasize the importance of tailoring bicycle equipment to the individual and fine attention should be given to adjusting bicycle equipment specifically for each cyclist. The optimal cadence rate is in the range 90-105 revolutions per minute at a power output of 200 Watts (Gonzalez & Hull, 1989).

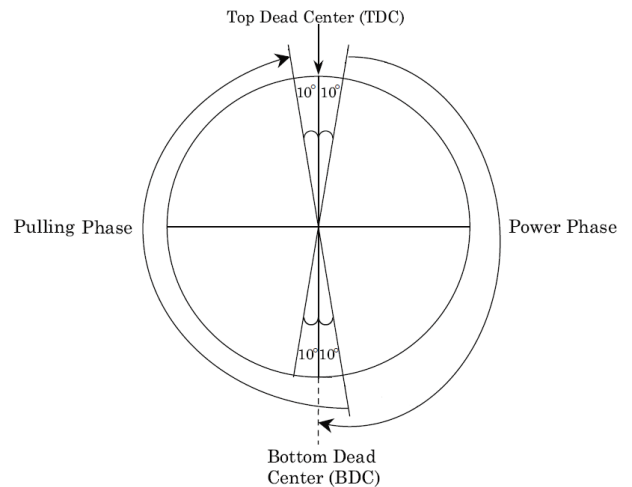


Figure 2.2 Pedal Phases
(So, Ng & Ng, 2005)

There is a well-defined muscle recruitment pattern in cycling due to the perfect circular motion of the pedal, however these patterns can be altered by muscle fatigue, change in cadence and bike setup. A pedal stroke is made up of four components, the Power Phase, the Bottom Dead Centre (BDC), Pulling Phase and the Top Dead Centre (TDC), as illustrated in *Figure 2.2* Extension of the hip, knee and ankle generate most of the power during the Power Phase. During the pull phase agonist and antagonist muscles perform flexion. During the TDC and BDC, there is no active contraction of any muscles, momentum completes this passive movement. The soleus, gastrocnemius and the tibialis anterior, support and stabilize the ankle joints by isometric contraction (So et al., 2005). *Table 2.1* illustrates which muscles are actively contracting at different degrees of motion.

Table 2.1 Patterns Of Muscle Activation During The Cycling (Ryan & Gregor, 1992).

Muscles	Action	Range of action (°) peak activity angle (°)	
Gluteus maximus	Hip extension	340–130	80
Vastus lateralis	Knee extension	300–130	30
Vastus medialis	Knee extension	300–130	30
Rectus femoris	Knee extension/Hip flexion	200–110	20
Soleus	Ankle stabilizer	340–270	90
Gastrocnemius	Ankle stabilizer/Knee flexion	350–270	110
Tibialis anterior	Ankle stabilizer/Ankle flexion	All the range	280
Hamstrings	Knee flexion	10–230	100
Biceps femoris	Knee flexion/Hip extension	350–230	110

2. Maximal Aerobic Capacity (vO_{2Max})

Maximal aerobic capacity is the product of heart rate maximum (HR_{Max}), Stroke Volume (SV) and arterial venous difference maximum ($(a-vO_2)_{Max}$). Arterial venous difference indicates systemic oxygen uptake. Therefore to increase vO_{2Max} one or more of these factors need to be increased.

Formula 2.1 Maximum aerobic capacity (McArdle, Katch & Katch, 2001; Powers & Howley, 2007)

$$vO_{2Max} = HR_{Max} \times SV \times (a-vO_2)_{Max}$$

vO_{2Max} is the maximum volume of oxygen that an individual can absorb, transport and use during maximal intensity exercise. A large portion of this oxygen is used within the active skeletal muscles to metabolize adenosine triphosphate (ATP) to fuel muscular contraction (Valberg, 2008).

The values are obtained as follows: Incremental exercise tests are used to test vO_{2Max} , peak oxygen consumption is measured and the participant's weight is also a factor in the calculation. Various physiological factors determine vO_{2Max} , maximal cardiac output, blood oxygen carrying capacity, number of mitochondria in skeletal muscle cells and ventilation maximum. Maximum cardiac output can be increased by a combination of increasing heart rate and increasing stroke volume, which is achieved by increasing the size of the ventricle, to increase volume of blood that can be accommodated, increasing the force of contraction, resulting in the ejection of a larger volume of blood (Brun, Connes & Varlet-Marie, 2007). These physiological adaptations are achieved through

prolonged endurance training (Giada, Bertaglia, De Piccoli, Franceschi, Sartori, Raviele & Pascotto, 1998).

3. Cycling Economy (CE)

Cycling economy is measured by the rate of oxygen consumption ($\dot{V}O_2$) while pedalling at a constant submaximal work load or constant power. Economy is a useful indicator of the gross efficiency, defined as the ratio of the work accomplished per unit time to the caloric energy expenditure per unit time. Therefore economy is the energy demands placed on the rider, which in turn is the biomechanical demands placed on the muscles (Van Sickle Jr & Hull, 2007). It has been noted that during endurance training, even once $\dot{V}O_{2Max}$ has reached its maximum level possible for that individual, performance does not stop improving. Performance is therefore not limited to maximal aerobic capacity alone. Improving cycling economy results in superior performance which is accomplished by aerobic training. This can explain how athletes, with similar $\dot{V}O_{2Max}$ values performances can be greatly dissimilar (Bassett & Howley, 2000; Shave & Franco, 2006). Performance is therefore determined by a combination of fractional use of $\dot{V}O_{2Max}$ (% $\dot{V}O_{2Max}$) and cycling economy.

The fraction of aerobic capacity (% $\dot{V}O_{2Max}$) that the cyclist uses at a constant submaximal load determines the duration that they are able to maintain that speed. Thus the lower percent $\dot{V}O_{2Max}$ they are using, the better they will perform and the slower the onset of fatigue (Correia, Lakatta, O'Connor, Becker, Clulow, Townsend, Gerstenblith & Fleg, 2002).

1.4 Lactate Threshold

Lactic acid is an end product of glucose metabolism in the glycolytic pathway and is formed at greater rates in conditions of inadequate oxygen (hypoxia) and in muscles with few mitochondria. High intensity exercise is often the aetiology of this hypoxic state in athletes. As intensity and/or duration of exercise are increased the body starts producing more lactic acid than it can metabolise and break down, at this point, where production is higher than removal, onset of blood lactic acid accumulation (OBLA) commences, this point is termed the lactate threshold. Lactic threshold is defined as the power output or % $\dot{V}O_{2Max}$ where, if exceeded will induce OBLA. This point is closely related to the heart rate and speed of cycling. Blood lactate concentration rises above 4mmol at the lactic threshold;

this level seems to be universally similar between different athletes. Exercise can continue for a very limited period of time at plasma lactate concentrations above four mmol, as skeletal muscle fatigue, cramping, discomfort and pain, rapidly set in. These symptoms, induced by high lactic levels, act as a self preservation mechanism, preventing total loss of homeostasis and minimise tissue damage. At maximum intensity exercise plasma lactate levels can rise as high as 20 mmol (Caputo & Denadai, 2009).

1.5 Peak Power Output (PPO)

Power is the product of force generated per stroke and cadence. The term peak power output has been used to describe maximum aerobic or maximum anaerobic power capacity. Aerobic peak power output is measured using a progressive incremental cycling test, where as anaerobic power is determined using an all out effort cycling test. Aerobic peak power output is a good indicator of endurance performance ability, second only to power at plasma lactate turning point, which is the most reliable indicator of endurance performance. According to Jeukendrup, Craig & Hawley (2000) aerobic peak power output for well trained cyclists range from 300 to 450 Watts, as illustrated in *Table 2.2*.

Table 2.2 Classification Of Road Cyclists (Jeukendrup *et al.*, 2000)

Category	Trained cyclists	Well-trained	Elite	World Class
Training and race status				
Training frequency per week	2-3x	3-7x	5-8x	5-8x
Training duration	30-60 min	60-240 min	60-360 min	60-360 min
Training background	1 year	3-5 years	5-15 years	5-30 years
Race days per year	0-10	0-20	50-100	90-110
Physiological variables				
Maximum power (Watts)	250-400	300-450	350-500	400-600
Power to weight (Watts/Kg)	4.0-5.0	5.0-6.0	6.0-7.0	6.5-8.0
vO ₂ (L/min)	4.5-5.0	5.0-5.3	5.2-6.0	5.4-7.0
vO _{2Max} (ml/kg/min)	64-70	70-75	72-80	75-90
Economy (W/L/min)	72-74	74-75	76-77	>78

C. ADAPTATIONS FOR PERFORMANCE ENHANCEMENT

During training athlete's condition their bodies to cycle further and faster, this is achieved by progressive adaptation induced by training. Adaptation is desirable in the musculoskeletal system, the metabolic system, the cardio-respiratory system and the neuro-endocrine system.

1. Musculoskeletal System

1.1 Skeletal Muscle Fibre Type

There are two different types of skeletal muscle fibres in humans, slow twitch (type one) and fast twitch (type two). These fibre types are controlled by gene expression of the myosin heavy chain isoforms. Type two can be further divided into 2A (fast oxidative), 2X (fast intermediate) and 2B (fast glycolytic). These fibres differ in oxidative enzymes and mitochondrial content, the highest content evident in type one and type 2A. Higher concentrations of oxidative enzymes and myoglobin, in type one and 2A, allow for a higher oxygen carrying capacity and metabolic rate. Velocity of contraction is dictated by fibre type, whereas resistance to fatigue is related to oxidative enzyme content. Therefore type one fibres show low velocity of contraction and high resistance to fatigue, type 2B fibres show high velocity of contraction and low resistance to fatigue. Muscle power is determined by the speed of contraction, during activities that require high muscle power for a short period of time, e.g. lifting a heavy weight, type two fibres are mainly used to generate the power, in endurance events where low power is required for a sustained period of time type one fibres are required.

Applying repeated mechanical stress to the muscles causes altered gene expression causing change from type 2B to 2X to 2A and finally type one, whereas inactivity causes the reverse resulting in a high concentration of type 2B in the muscles. During initial training, type two muscle fibre adaptation happens at a faster rate than type one (Schiaffino & Serrano, 2002; MacDougall, 1986).

1.2 Skeletal Muscle Adaptation

Morphological changes in skeletal muscles due to training include increased cross-sectional area of both type one and type two fibres (hypertrophy), Increased expression of MHC-2A and decreased expression of MHC-2X isoforms (Baldwin & Haddad, 2001), as well as various intra-cellular metabolic

changes and altered muscle proteins (Scott, Stevens & Binder-Macleod, 2001). These changes only occur when loads are maximal or near maximal and that the stimulus duration is long enough. Hypertrophy is a delayed phenomenon occurring late after the onset of training and requires rest periods between training for maximal adaptation (Bruton, 2002).

A meta-analysis by Kelley (1996) reviewed the effect of exercise on the number of skeletal muscle cells in animals, the study concluded that hyperplasia (increase in number of cells) does occur in animals, however this has not been shown in human studies. It is suggested that there may be a small fractional increase in the number of muscle cells, especially in young athletes.

There are multiple factors that stimulate morphological muscle changes including, mechanical factors such as change in tension within the muscle, high testosterone levels and altered thyroid levels (Baldwin & Haddad, 2001).

It has been shown that training causes protein breakdown with cellular damage and subsequent repair during a recovery phase. The repair is over expressed, causing an overshoot of protein synthesis. This cycle of damage and repair, if continued, will lead to muscle adaptation and hypertrophy (Talmadge, 2000; MacDougall, 1986).

1.3 Muscle Endurance

Muscle endurance is defined as the ability of a muscle to contract repetitively or to sustain a single contraction for a prolonged period of time. During cycling repetitive contraction is the more desirable component of muscle endurance, as increased muscle endurance allows the cyclist to perform more peddle strokes than an untrained cyclist (Kisner & Colby, 1990).

Muscle endurance can be increased by performing many repeated contractions against a mild resistance, far below maximum strength. Training programs often aim to increase muscle endurance, which acts as a base from which other factors can be developed. These factors include muscle strength, skill training and muscle hypertrophy. Morphological changes following endurance training are as a result of muscle cell organelle conversion. This conversion is accomplished by changes in gene expression of the myosin heavy chain isoform. Morphological changes include:

- Increased number of capillaries surrounding muscle fibre, which increases blood volume to meet the increased metabolic demands of the muscles.
- Increase myoglobin content, which is an oxygen carrier in the muscle.
- Increase size and number of mitochondria.
- Increase activity of enzymes intra cellular, increase glycogen and fat stores in muscle.
- Increased lactate threshold.
- Small increase in cross sectional area of muscle, especially of type one fibres.

The greatest adaptation occurs during the first few weeks of training (Tonkonogi, Walsh, Svensson & Sahlin, 2000).

1.4 Muscle Strength

Muscle strength is defined as the ability of a muscle or group of muscles to contract and produce a maximal force for one effort. Therefore an increase in muscle strength allows the muscle to produce a greater force. Muscle strength can easily be increased by training, provided that the loads applied to the muscles exceeds those of normal daily activities (Komi & Hakkinen, 1991). Muscle response to load is directly proportionate to the magnitude of load, therefore the greater the load the greater the increase in muscle strength. Response to loading is also dependant on the initial status of the muscle. Athletes often have highly adapted muscles, resulting in greater loads and more repetitions needed to cause further adaptation and prevent hypotrophy. Loads for athletes should range between 80 percent and 100 percent of maximal voluntary performance for that individual (Bruton, 2002; Sverdlova & Witzel, 2010).

1.5 Muscular Fatigue

Muscular fatigue is defined as a decrease in the velocity of contraction or as a decrease in force at which the muscle can contract. Potma, van Graas & Stienen (1995) investigated the effect of low pH and accumulation of inorganic phosphate (P_i) on the ATPase enzymatic activity and force of muscular contraction. The study demonstrated that both ATPase activity and force of muscular contraction was decreased in both fast and slow twitch muscle fibres. The study further revealed that low pH and P_i 's effects are not independent of each other and exhibit a synergistic effect to further hinder muscular physiology. Maintaining homeostasis by preventing the accumulation of hydrogen ions (H^+) and inorganic phosphates is crucial to prevent premature muscular fatigue.

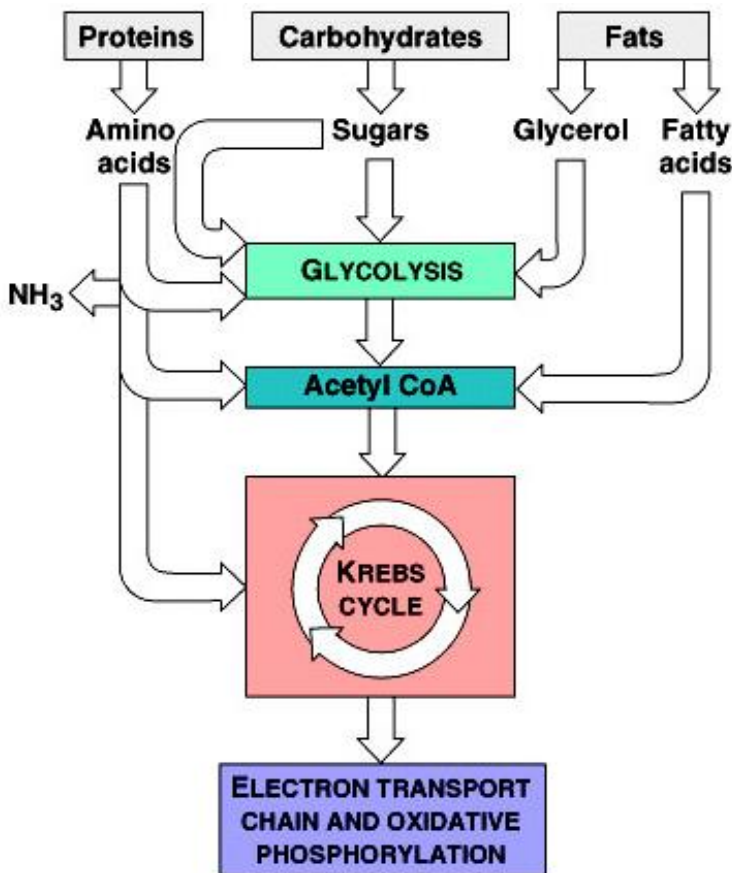
2. Metabolic Systems

2.1 Energy Systems

Adenosine Triphosphate (ATP) is the main energy source of the body. There are three distinct, yet closely interrelated energy systems that operate together to satisfy the ATP requirements of muscles during exercise, namely the Adenosine Triphosphate Phosphocreatine (ATP-PCr), Glycolytic and Oxidative Phosphorylation systems. The ATP-PCr system and Glycolytic system are both anaerobic metabolic pathways that produce ATP in the absence of oxygen, whereas the Oxidative Phosphorylation system is an aerobic metabolic pathway which is only active in the presence of oxygen. The anaerobic pathways are capable of regenerating ATP at high rates yet are limited by the amount of energy that can be released in a single bout of intense exercise.

Figure 2.3 Energy Systems

(Powers & Howley, 2007)



The aerobic energy system can produce sustained energy for extended time periods yet limited to slow production of ATP (Pisani, Leclerc, Jarretou, Marini & Dechesne, 2005; Powers & Howley, 2007).

Aerobic training increases the activity of enzymes, involved in beta-oxidation of fat, within the active muscle fibres. An increase in the activity of oxidative enzymes (Succinate dehydrogenase and citrate synthase) within the skeletal muscle cells is a further metabolic adaptation to exercise (Wadley & Le Rossignol, 1998).

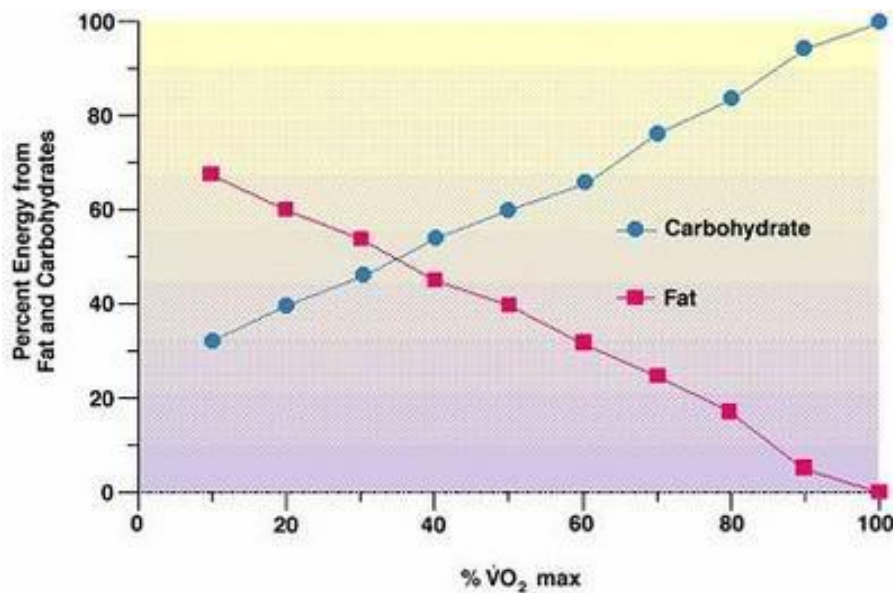
2.2 ATP Production

The metabolism of one glucose molecule yields 38 ATP molecules, however more recent studies indicate that only 32 ATP molecules are released into the cytoplasm and are available as energy. Six ATP molecules are used during aerobic respiration for the breakdown and transport of ATP across the mitochondrial membrane. However this high yield of ATP only happens when oxygen is freely available and ATP demand is relatively low. During periods of high intensity exercise oxygen becomes limited and ATP demands increases dramatically. Aerobic and anaerobic energy systems do not function independently of each other but rather together, with varying proportions, during different intensity exercises (Powers & Howley, 2007).

2.3 Interaction Between Aerobic and Anaerobic Exercise

Aerobic exercise is classified as low intensity exercise (less than 30 percent $\dot{V}O_{2\text{Max}}$) where fat metabolism is dominant, where as anaerobic exercise is high intensity exercise (greater than 70 percent $\dot{V}O_{2\text{Max}}$) where carbohydrate metabolism is dominant. As the fractional use of $\dot{V}O_{2\text{Max}}$ and intensity increases, the rate of aerobic metabolism decreases and anaerobic metabolism increases. During longer bouts of exercise aerobic metabolism is dominant (Powers & Howley, 2007).

Figure 2.4 Crossover Concept



2.4 Fuelling The Energy Systems

The energy systems need to be sustained via fuel sources. Carbohydrates (muscle glycogen and plasma glucose) and lipids play the major role in satisfying this requirement, whereas amino acids are a minimal component. Lipids play a predominant role in sustaining exercise at half or less of aerobic capacity ($\dot{V}O_{2\text{Max}}$). However, higher intensity exercise, depend increasingly on plasma glucose and muscle glycogen as substrates. The switching from fat metabolism to carbohydrate metabolism is referred to as the “crossover concept”. Thus, as exercise intensity increases, fuel selection switches (crosses over) from lipid to carbohydrate. Endocrine and intra-cellular factors play a vital role in determining substrate balance during sustained exercise. However, genetic variances play a fundamental role among different athletes. Phenotypic adaptations in response to training also effect the balance of substrate utilization during exercise (Brooks, 1998).

Average percentages of energy system utilization during different cycling disciplines have been predicted in *Table 2.3*. According to the *Table 2.3* all three energy systems were active during the testing protocol as the intensity and duration is similar to 4000m pursuit. The effect of treatment is thus observable, regardless of the altered energy system. Intensity is not solely responsible for energy system utilization, as duration of the activity and the participant’s fitness also plays a major role (Bompa & Bompa, 1999).

Table 2.3 Energy System Selection Percentages (Bompa & Bompa, 1999)

Cycling	Phosphagen	Glycolytic	Oxidative
200m Track	98	2	0
4,000m Pursuit	20	50	30
Road Racing	0	5	95

2.5 Metabolic Homeostasis

Homeostasis is crucial for tissue functioning especially during periods of increased metabolic rate. Maintenance of ionic levels within small ranges is necessary for sustained performance and optimal functioning of all body systems. During exercise, metabolic rates are greatly increased, resulting in a minor ion imbalance which may have major repercussions (Kronfeld, 2001).

3. Cardiovascular-Respiratory System

The respiratory system and the circulatory system are linked through the capillary systems found around the alveolar sacks in the lungs. Gaseous exchange of oxygen and carbon dioxide take place continuously through diffusion between blood and air. Furthermore, the blood constituents are important for continued function of both these systems. Production of blood components, control of blood pressure (Bp), peripheral resistance and depth of breathing are all controlled by the nervous and endocrine (neuro-endocrine) system. These systems function together and rely heavily on each other for optimum functioning (Morton, 2008).

3.1 Respiratory System

The respiratory system provides the muscles with the oxygen required for ATP production. The efficiency of allowing oxygen to diffuse to the blood is directly proportionate to the athlete's performance. At high intensity exercise (greater than 85 percent $\dot{V}O_{2Max}$) respiratory muscle fatigue can set in and limit performance.

Respiratory fatigue can be further broken down into four mechanisms:

- An inadequate hyperventilatory response to exercise, because respiratory muscles are unable to contract with sufficient force to achieve desired air pressure,
- Tachypnoea, rapid shallow breathing which does not allow sufficient air flow. Elite athletes do not experience tachypnoea, as they have trained their bodies to breathe deep and rhythmically,
- Increased sympathetic vasoconstrictor outflow to limb skeletal muscle, limiting blood supply to the active muscles. This protective mechanism increases the blood supply to the vital organs, ensuring the brain and heart gets adequate blood supply,
- An increased sensation of dyspnoea, which is a conscious sensation of breathlessness. The purpose of dyspnoea is to increase depth of breathing and motivates the use of accessory respiratory muscles to assist the respiratory muscles in maintaining adequate pressure differences within the lungs. Dyspnoea serves as a protective mechanism, limiting strain and fatigue of the respiratory muscles (Wells & Norris, 2009).

3.2 Cardiovascular System

An athlete's heart, according to Atchley & Douglas (2007), shows morphological changes as a physiological adaptive response to exercise. These changes include an increased left atrial and ventricular volume, increased left ventricular mass and right ventricular structural changes. Most of these changes fall within normal reference ranges, however there are significant numbers of athletes who have “abnormal” measurements (outside normal ranges) that can easily be diagnosed as pathological rather than physiological adaptations.

Cardiac changes in cyclists were further investigated by Miki, Yokota, Seo, & Yokoyama, (1994) comparing 104 professional cyclists to a control group of a 104 similarly aged untrained individuals. Measurements taken from participants included heart rate, systolic blood pressure and echocardiographic measurements. The study concluded that heart rate was lower and left ventricle wall mass, thickness and dimension index was increased in professional cyclists versus untrained individuals ($p < 0.001$). There was no significant difference in systolic blood pressure readings between the two groups.

Circulatory demands placed on the heart are very high during exercise, relative to resting demands. It is essential that the blood is of the correct nature to be able to meet this demand. Important blood characteristics include optimal viscosity, haemoglobin concentration, plasma volume and red blood cell count. During prolonged exercise, large amounts of water and electrolytes are excreted from the body through the sweat glands. The main origin of this fluid is the blood plasma, thus resulting in a decreased blood volume, which further causes a decline in performance and dehydration. It is therefore crucial that blood volume during exercise be maintained to ensure optimal cardiovascular functioning (McKenna, 1995; Machado-Moreira, de Castro Magalhães, Vimieiro-Gomes, Lima & Rodrigues, 2005).

3.3 Blood Volume

Blood volume is constantly changing in response to activity level, diet, weather and metabolic rate. Blood viscosity changes as the plasma volume changes. However the total number of red blood cells in the body remains constant. Increased plasma levels results in an insipid composition, whereas decreased plasma levels results in a more viscous composition. Blood with a high viscosity has a

higher oxygen carrying capacity per litre, however because of its increased viscosity it requires a greater blood pressure and increased time for blood to pass through the capillaries. Loss of plasma volume due to dehydration causes a disruption of the circulatory and thermoregulatory systems, yielding a rapid decline in performance. Plasma moves out the capillaries by increased hydrostatic pressure and increased permeability of the cell membranes, induced by higher blood volumes passing through the tissue during exercise. The ratio between pre and post capillary pressure is altered during exercise to meet the demands of the muscles. Plasma volume loss is directly proportionate to intensity of the exercise (Kawabata, Suzuki & Miyagawa, 2004).

Hypovolemia induced by thermal dehydration, which is often the case in exercise, initially elicits vasodilatation, causing blood to pool in the cutaneous tissue, thus increasing radiation of heat through the skin, with a resultant low central blood volume. When central blood volume reaches a critical low level vasoconstriction is activated, less heat is radiated, possibly resulting in hyperthermia, which severely limits performance capability. It has been suggested that hyperthermia is one of the limiting factors of aerobic performance (Nielsen, Hales, Strange, Christensen, Warberg, & Saltin, 1993).

The performance is also limited by the body's ability to generate ATP, which in turn is limited by the amount of oxygen that can be delivered to the muscles, which is limited by oxygen carrying capacity of the blood and finally oxygen loading rate in the lungs is limited by ventilatory capabilities of the lungs. The oxygen carrying capacity is decreased as the blood volume decreases (Salazar Vázquez, Wettstein, Cabrales, Tsai & Intaglietta, 2008).

Sports physiologists have determined that if drugs are taken to increase haemopoiesis, thus increasing circulating red blood cells (RBC) and the oxygen carrying capacity. However these drugs are illegal for use in most sports due to the increased risk of thrombus formation and increased stress on the heart. Due to stringent drug testing in the cycling industry, an alternative method of increasing RBC for a competition is for the athlete to infuse their own previously donated blood, back into their bodies. However this carries the same risk as the drugs and is also illegal (Dunn, Thomas, Swift, Burns & Mattick, 2010).

4. Neuro-Endocrine System

This system is considered to be one of the most important systems influencing performance, especially during longer events. A small change within this system can produce major effects, which can either positively or negatively affect performance and recovery (Yun-tao, 2011).

Skeletal muscle strength gain is achieved by hypertrophy of muscle cells and through neural adaptations. It has been clearly demonstrated that strength increases in the early stages of training are almost solely attained by neural adaptation, with little or no structural changes to the muscles. Neural adaptations include, improved co-ordination, increased inhibition of antagonists and increased activation of agonist (Sale, 1986). Therefore untrained skeletal muscles have the potential to contract with a greater force, provided there is improved nervous stimulation and increased coordination of motor units. Neural muscular adaptations are not the only component which increases performance post training, hormonal changes are crucial for an athlete's development (Morton, 2008).

The major function of the neuro-endocrine system is that of metabolic, hormonal and ionic homeostasis. Aldosterone, which is released during high intensity exercise, is responsible for sodium and potassium balance. Cortisol is released in response to the stress induced by exercise, regulating fuel availability, energy and amino acids for tissue repair (Taylor, van Rosendal, Coombes, Gordon & Stowasser, 2010). Chronic exercise training can suppress testosterone secretion in males, which could result in under expression of male characteristics. Thyroid hormone is responsible for controlling the body's metabolic rate and during exercise thyroid stimulating hormone release is increased resulting in high levels of thyroid hormone (Acheson, Jéquier, Burger & Danforth Jr, 1984; Smallridge, Whorton, Burman & Ferguson, 1985).

D. RECOVERY

1. Overtraining / Overreaching

Overtraining is characterized by deterioration in performance and acute inflammation following periods of persistent training or training stressors. Over training has major health implications for the athletes, however currently, there is no single diagnostic marker for overtraining. Physical training causes mild tissue damage. During a recovery period, this tissue damage is repaired as an

adaptation to the stress, resulting in improved performance (Maurer, Burkhoff, Maybaum, Franco, Vittorio, Williams, White, Kamalakkannan, Myers & Mancini, 2009). The greatest increases in muscle size and strength are produced when two or three days of rest are allowed between training sessions for specific muscles. However when sufficient time for recovery is not allowed after high intensity and/or high volumes of exercise, this micro trauma could turn into severe tissue damage. Over training for an extended time period, is termed overtraining syndrome (OTS) and is characterised by decline in performance even when an extended recovery time is allowed. Chronic tissue damage prevents adaptation and the athlete develops physiological, biochemical, immunological and psychological symptoms (Margonis, Fatouros, Jamurtas, Nikolaidis, Douroudos, Chatzinikolaou, Mitrakou, Mastorakos, Papassotiriou, Taxildaris & Kouretas, 2007). Overtraining is thus associated with decrease in performance, altered hormone levels, loss of appetite, sleep disturbances, altered mood states, depression, increased anxiety, mental and physical fatigue, altered resting, exercise and recovery heart rate, muscular pains, swelling of tendons and ligaments and loss of muscle mass. It has been postulated that the mental changes are as a result of lowered serotonin levels in the brain, however a connection with serotonin and fatigue has not yet been found (Powers & Howley, 2007).

Inflammation is often viewed as a symptom that needs to be stopped and reduced so that training can resume. Inflammation is however an integral part of repair and regeneration of tissue which begins 24 to 48 hours after a training bout, with muscular discomfort peaking around 60 hours. Inflammation reduces range of motion, causes failure of contraction mechanisms and a reduction in muscular power, all of which limit the athlete's ability to train, thus preventing further injury (Close, Ashton, McArdle & MacLaren, 2005). To overcome this, athlete's often resort to using non-steroidal anti-inflammatory drugs (NSAIDS). NSAIDS have analgesic and anti-inflammatory effects resulting in decreased muscular pains, swelling and inflammation. Athletes feel better and often continue to train, despite the fact that the tissue damage has not been fully repaired, leaving the tissue vulnerable to further damage. Long duration NSAIDS use attenuates tissue repair and adaptive response to training, ultimately decreasing the athlete's training-induced performance gains (Ziltener, Leal & Fournier, 2010).

Over reaching is applying a maximal exercise stress to an athlete for a limited amount of time to cause maximal adaptation or super compensation. If the time period is too long, training intensity is

too high or training is repeated too frequently, the athlete's body can no longer adapt (Hug, Mullis, Vogt, Ventura & Hoppeler, 2003).

2. Post Exercise Recovery

Recovery is defined as the return of the body to its pre-exercise state following exercise (McMahon & Wenger, 1998). Returning the body to its' pre-exercise state is multifaceted and involves physiological, anatomical and psychological recovery. The process of recovery is assisted by an increased metabolic rate post-exercise, more specifically the aerobic system. During the first phase of recovery (fast phase), 70 percent of ATP and PCr energy stores are replenished within 30 seconds and phosphagen stores take a further four minutes to recover post-exercise. During the second phase (slow phase) the cardiac rate, respiratory rate and core temperature remains elevated for up to 24 hours post-exercise, depending on exercise duration and intensity. Lactate levels and pH take an hour or more to reach baseline values (Denadai & Higino, 2004). Fatigue, muscle pain, joint pain, head ache and anxiety are some of the symptoms that may persist for the full 24 hours. Signs and symptoms that last longer than 24 hours are due to physical injuries, caused by repeated movements required for endurance sports. Common repetitive strain injuries include: ligament strain, muscle strain, micro-muscle tears and joint injury (Callaghan, 2005).

Many methods have been suggested to accelerate this recovery process including, passive rest, relaxation techniques, stretching, massage, compression garments, hydrotherapy and light exercise. Hydrotherapy, namely hot and cold immersion, may have some merit in aiding recovery. It is suggested that the hot and cold temperature increase blood flow to the muscles, thus enhancing muscle metabolic rate and waste product removal. The higher blood flow rate facilitates lactate removal and supplies tissue with resources needed for repair (Cochrane, 2004).

Replenishing glycogen stores and ionic homeostasis are some examples of the physiological imbalances that need to be corrected before recovery is complete. Repairing damaged muscle fibres, tendons, ligaments and soft tissue are examples of anatomical recovery. Reducing sympathetic neural excitation, mental fatigue and mood recovery are psychological (Brooks, 1998; Sabapathy, Morris & Schneider, 2006).

3. Physiological Signs And Symptoms Of Exercise

Exercise places a high metabolic demand on the body, especially on the cardiovascular and respiratory system. According to one school of thought the metabolic increase could be as high as 2000 percent (Guyton & Hall, 1996). Muscles used during exercise, especially larger group muscles like the quadriceps used during cycling, have a very high metabolic rate, consequently placing a high demand on the body.

Enhancing athletes' performance has always been the goal of coaches, trainers, researchers and the athlete himself. Various drugs have been used to enhance performance such as erythropoietin to increase the number of red blood cells, anabolic steroids to increase muscle growth and amphetamines to stimulate the central nervous system. Although these and many other drugs can enhance performance, it is equally true that overuse of these drugs may lead to deterioration of performance and the body. However, by increasing the recovery rate, as well as the body's tolerance to the physiological stresses experienced during exercise, performance can greatly be improved (Brun et al., 2007).

Physiological changes during cycling in healthy male and female athletes include an increased heart rate, stroke volume, increase in volume of oxygen consumed each minute (vO_2), breathing rate, depth of breathing, increase sweating and general signs and symptoms of fatigue (Mary, 1984; Sullivan, Cobb & Higginbotham, 1991). Cycling uses large groups of muscles, which place a high physiological demand on the cardiovascular system. This increased demand is met by a steep rise in cardiac output as well as an increase in oxygen extraction. The increase in cardiac output, results from the increased heart rate levels and stroke volume during exercise (Fleg & Lakatta, 1984). Stroke volume increases during low levels of exercise via the Frank-Starling mechanism. During higher levels of exercise, stroke volume increases predominantly due to increased contractility of the left ventricle. At near maximum heart rates, there may even be a slight decline in stroke volume due to tachycardia and limited filling time (Bevilacqua, Savonitto, Bosisio, Chebat, Bertora, Sardina & Norbiato, 1989).

One of the major changes during exercise is the increased oxygen demand by the skeletal muscles used for the particular exercise. The respiratory and the cardiovascular system work together as a

unit “cardiopulmonary system” to match this increased demand (Lovering, Haverkamp & Eldridge, 2005).

The nervous and endocrine system is the two major components in regulating homeostasis within the body. Maintaining plasma glucose, electrolytes, pH and blood pressure, along with many other factors, within narrow limits, directly influences performance. Balancing fuel system utilization is also important (Powers & Howley, 2007).

Muscular fatigue is defined as a decline in maximal force production and a reduced ability to perform work. Prolonged exercise at sub-maximal intensity or short maximal intensity exercise, results in decreased muscle force of contraction, therefore fatigue. There are many factors that are responsible for fatigue, all of which are not fully understood. Fatigue varies according to the exercise and type of stimulation. During short maximal intensity exercise, accumulation of lactate, hydrogen ions, adenosine diphosphate (ADP), inorganic phosphate and free radicals within the active muscle fibres causes a disruption of homeostasis within the muscle cell. During prolonged exercise, accumulation of free radicals and depletion of glycogen stores causes an extra-cellular imbalance of homeostasis; Central nervous system (CNS) also plays a role in this type of fatigue. The role of the CNS in fatigue has been termed Central Fatigue; the subconscious brain reduces motor stimulation, resulting in reduced number of muscle cells activated. This decreased activity results in decreased force of contraction (Powers & Howley, 2007).

4. Psychological Signs And Symptoms Of Exercise

There is absence of measurable scientific data explaining the psychological effects of exercise, however many participants report a general feeling of well being, decreased levels of depression, anxiety and reduction in neuroticism. It has been reported that exercise is beneficial for all ages and sex groups. However these positive results of exercise can be reversed in people who exercise to an extreme degree, which include long durations, high intensities or high frequencies. It was found that overtraining is associated with mood disturbances (Alla, Sullivan, McCrory, Schneiders & Handcock, 2008). The converse also proved true where a decrease in exercise intensities resulted in an improved mood state and physical performance, thus highlighting the importance of appropriate training (Morgan & Goldston, 1987). Fatigued athletes often suffer from depression, lack of caring, sleep disorders, loss of self esteem, apathy, weariness, irritability, quarrelsomeness, mood swings,

emotional isolation and increased anxiety, of which depression is the most common manifestation. It was found that 80 percent of athletes studied over a ten year period showed clinical signs of depression (Morgan & O'Connor, 1988). Barron, Noakes, Levy, Smith & Miller (1985) found that repeated incidences of fatigue could lead to burnout, which could last for up to six months after onset. Burnout is a state of physical and psychological exhaustion that follows fatigue. The main difference between the two is that burnout is not a sudden occurrence, rather as a result of prolonged fatigue. Burnout is recognized by four stages; the first stage refers to depersonalisation, where the athlete emotionally withdraws from the sporting environment and shows a clear disinterest. The second stage follows with feelings of decreased self worth, dissatisfaction as well as the belief that they no longer contribute to their sport. In the third stage, the athlete isolates themselves from fellow teammates, often miss training sessions. The final stage is characteristic of psychological and physical exhaustion, with no desire or physical power to train or compete (Hug et al., 2003; Parfitt & Gledhill, 2004).

Some athletes are more prone to these effects of overtraining than others; however athletes that exhibit the following traits, such as perfectionism, image conscience, overly high self expectations are more susceptible. Different individuals may also be more affected either physically or psychologically (Hug et al., 2003).

E. HOMOEOPATHY

1. Introduction

Homoeopathy is a 200 year old system of medicine developed by the German physician Samuel Hahnemann and has persisted and grown in many countries throughout the world. Kaul (1996) cited estimates that over 500 million people worldwide receive homoeopathic treatment. Its persistence is especially striking because of intense scepticism and attacks that it has received since its inception (Bell, Baldwin, Schwartz & Russek, 1999).

2. Health And Disease In Homoeopathy

Homoeopathically disease is viewed as a manifestation of a larger disease; an imbalance of the body affecting it on multiple levels. Treatment of this imbalance should therefore not be limited to a single

body part or particular symptom, but rather selected on the totality of the clinical picture. To determine the total clinical picture, symptoms in the mental, physical and emotional realm are noted (Covinsky, Wu, Landefeld, Connors, Phillips, Tsevat, Dawson, Lynn & Fortinsky, 1999). The reason for this treatment is the belief that treating a symptom, single body part or an organ will only suppress that symptom causing the body to express its imbalance on another level. The new symptom, following suppression, is often more serious than the original symptom, the psyche is often most affected (O'Donovan, 1986). These new symptoms tend to move to a deeper level, e.g. from the skin to the lungs, and upward toward the head. Using this model progression during treatment should be in an opposite direction. Hering's Law of Cure states that proper treatment will lead to progressive changes, with symptoms resolving from within outward, from above downward, from the most to the least important organ systems and reversing their order of appearance over time (Saine, 1994). Research has confirmed this with investigations that found the link between eczema, asthma and depression, resulted in subjects with greater medical treatment having a higher incidence of somatic distress and mental disturbances (von Korff, Ormel, Katon & Lin, 1992; Saravay, Pollack, Steinberg, Weinschel & Habert, 1996; Unutzer, Patrick, Simon, Grembowski, Walker, Rutter & Katon, 1997; Covinsky et al., 1999).

However cycling, in combination with a homoeopathic substance, could cause an overreaction and aggravate the cyclist's condition. To minimise aggravation these medicinal substances are made less toxic by a pharmacotechnique called dynamisation (serial dilution with succussion), a system developed by Samuel Hahnemann. Furthermore dynamisation develops the substances latent dynamic power which is capable of stimulating the body's vital reaction force to overcome the current disease or stress (Teixeira, Guedes, Barreto & Martins, 2010).

Health, according to Vithoukas (1980: 3) is more than the absence of disease, rather as "freedom from pain in the physical body, having attained a state of well-being; freedom from passion on the emotional level, having as a result a dynamic state of serenity and calm; and freedom from selfishness in the mental sphere, having as a result total unification with Truth", expressing the importance of proper treatment and a fundamental prognostic tool.

3. Law Of Similars

Homoeopathic treatment relies on the selection of a single remedy that can cause a unique pattern of mental, emotional and physical symptoms in a healthy human being. The Law of Similars states that symptoms caused by a substance in a healthy person can be cured in a diseased person, with a similar symptom pattern. Homoeopaths spend a substantial amount of time, obtaining a comprehensive symptom pattern through detailed case taking, from the patient. The clinical focus is to find the core of the disturbance, often which is manifest in the form of a characteristic symptom, unique or distinguishing of the person and can not be explained due to the patho-physiology of the disease (Bellavite, Lussignoli, Semizzi, Ortolani & Signorini, 1997). These symptoms have been termed peculiar, queer, rare or strange (PQRS) symptoms and are the key to finding the curative substance similar to the disturbance “the similimum”. Curative substances found in *materia medica* (Remedies) have a symptom pattern or picture along with PQRS symptoms. This symptom picture is often present in the patient that has been successfully treated by that particular remedy and often the basis for prescription by the homoeopath (Bell et al., 1999).

Individuals may differ in their response to exercise, hence producing a varied symptom picture. *Phosphoricum acidum 200CH* may not fit every symptom picture, as individualization would result in a different indicated remedy. This could have a negative effect on the test results, posing a gap in the study. However, the researcher believes that *Phosphoricum acidum 200CH* will have a favourable result to a greater or lesser degree in all participants, as indicated through homoeopathic repertorisation.

4. Remedy Selection

The remedy was selected by repertorisation of signs and symptoms of overtraining and more specifically cycling overtraining. Symptoms included fatigue from perspiration loss, complaints of the calves, muscle pains, muscle twitching and cramping in the lower limbs, mental exertion, mental symptoms caused by injury and general symptoms from over exertion (Boericke, Boericke & Griffin, 1922 ; Phatak, 2005).

According to Ryan & Gregor (1992) most of the lower limb muscles are active during a cycling peddle stroke, namely the gluteus maximus, biceps femoris, vastus medialis, rectus femoris, soleus,

gastrocnemius, tibialis anterior, hamstrings and vastus lateralis. Muscle cramping, muscle fasciculations, discomfort and pain can be experienced during high intensity exercise (Caputo & Denadai, 2009). The rubrics regarding calf pain, muscle pain, twitching of the lower limb and muscle cramps were thus selected. According to Morgan & O'Connor (1988) cycling can induce mental stress, depression, physical injury and aggravation of symptoms due to continued exercise. Callaghan (2005) further demonstrated that common repetitive strain injuries include: ligament strain, muscle strain, micro-muscle tears and joint injury. The rubrics general weakness from perspiration loss, mental exertion, ailments from injury and general aggravation from exertion were thus selected.

Figure 2.5 Repertorisation

Homeopathic Day Clinic (31013)

untitled

This analysis contains 309 remedies and 8 symptoms.

Intensity is not considered

		arn.	calc.	cupr.	rhus-t.	sep.	ars.	phos.	sil.	ph-ac.	ambr.	graph.	kali-c.
	Sum of symptoms (sort:deg)	1	2	3	4	5	6	7	8	9	10	11	12
		7	6	6	6	6	6	6	6	6	6	6	6
		9	12	12	12	12	11	11	10	9	8	8	8
01. GENERALS - WEAKNESS - perspiration - from perspiration; weakr	1	1	2	2	2	3	2	3	2	2	1	1	1
02. EXTREMITIES - LEGS; complaints of - Calf	1	1	3	1	3	3	3	1	1	1	1	3	1
03. GENERALS - PAIN - sore - Muscles, in	2	1	-	-	-	-	-	-	-	-	-	-	1
04. EXTREMITIES - TWITCHING - Lower limbs	1	1	1	2	2	1	1	3	2	1	2	1	1
05. EXTREMITIES - CRAMPS - Lower limbs	1	-	2	3	-	1	1	1	1	1	1	1	-
06. MIND - AILMENTS FROM - mental exertion	1	1	1	3	1	1	1	1	2	2	1	1	2
07. MIND - AILMENTS FROM - injuries, accidents; mental symptoms fr	1	1	-	-	1	-	-	-	-	-	-	-	-
08. GENERALS - EXERTION; physical - agg.	4	3	3	1	3	3	3	2	2	2	2	1	2

As illustrated in *Figure 2.5*, *Arnica montana*, *Calcarea carbonica*, *Cuprum metallicum*, *Rhus toxicodendron*, *Sepia*, *Arsenicum album*, *Phosphorus*, *Silicea*, *Phosphoricum acidum*, *Ambra Grisea*, *Graphites* and *Kalium carbonicum* were highly ranked. Using the *Materia Medica* (Boericke et al., 1922 ; Phatak, 2005) the list of differentials was further narrowed down to five remedies for further investigation, namely *Arnica montana*, *Cuprum metallicum*, *Rhus toxicodendron*, *Phosphorus* and *Phosphoricum acidum*.

In previous studies *Arnica montana* was found to be effective in improving post exercise recovery. According to Phatak (2005: 78) *Arnica montana* is for the treatment of sore, painful, rigid, stiff muscles with a bruised sensation accompanied by involuntary muscle and tendon twitches. The symptoms are often triggered by exhaustion, overuse of an organ or due to repetitive strain. However there is little evidence that *Arnica montana* can improve performance, restore homeostasis or re-establish the mental state.

Cuprum metallicum has been successfully used to treat disorders involving involuntary nervous excitation e.g. epilepsy. This remedy is indicated for nervous exhaustion, muscle knots and cramping of muscles, ailments from bodily exhaustion (Phatak, 2005). However this state of exhaustion and weakness is on the mental plane and has little effect on the metabolic system.

Rhus toxicodendron has an affinity for the joints and ligaments, producing arthritic symptoms. The nerves and spinal cord are affected resulting in paralysis of affected parts. Further more, the intensity of *Rhus toxicodendron*'s symptoms are less and come on more slowly than those induced by intense cycling (Phatak, 2005).

Phosphorus has a great sensitivity to change, both of the internal and external environments. The action of high intensity cycling and physiologic compensation would result in an immense change. Legs feel heavy, as if glued to the floor, weakness and trembling of the limbs with exertion. There is imbalance of the circulatory system with easy bleeding, causing circulatory disturbances. However the main organs affected are the gastrointestinal tract, spinal cord and nerves, with very few symptoms related to the muscular system or the lower extremities, which are most important during cycling. Symptoms of *Phosphorus* come on slowly and insidiously, there is weakness of mind even when the body is strong (Phatak, 2005).

5. *Phosphoricum Acidum*

Phosphoricum acidum is one of homoeopathy's most effective remedy for exhaustion, listlessness, apathy, loss of appetite, craving for cold drinks and lethargy. This state is often caused by overwork, exhaustion, loss of fluid and stress. This remedy is considered an acute or sub-acute remedy. Mental symptoms include a peculiar emotional neutrality or numbness, grief and mental exhaustion. Patient feels like a ghost, drifting through an unreal life in which he performs almost automatically, with no motivation and no sense of satisfaction. Phosphoric acid's pathology is initially on the emotional level which then causes a decrease in physical performance (Cowperthwaite, 1927; Phatak, 2005).

The proposed mechanism of action is to stimulate the bodies buffering mechanism to prevent the accumulation of hydrogen ions and accumulation of inorganic phosphate. Mentally the participants will feel more focused, mentally alert and enabling an enhanced performance (Potma et al., 1995).

6. Phosphoric Acid Crude Substance (H_3PO_4)

Symptoms of a homoeopathic remedy often closely relate to the action, characteristics and toxicology of the crude substance. Phosphoric acid is the second most produced acid in the world, after H_2SO_4 . It was used as a raw material for the production of detergents, food products, toothpastes and fertilizers. H_3PO_4 is produced by reduction of phosphate rock, followed by oxidation, hydration and addition of heat (Thermal process) or by a reaction of phosphate rock with H_2SO_4 (Wet process). Wet processing yields an impure acid; impurities include fluoride, iron, copper and other metal ions, originally present in the phosphate rocks (El-Asmy, Serag, Mahdy & Amin, 2008).

Inorganic phosphoric acid can exist in two forms, anhydrous crystal or as clear syrup liquid. Pure anhydrous phosphoric acid is a white solid that melts at 42.35 degrees Celsius to a viscous liquid, ranging from clear, green, brown to blue-black, depending on its purity. Most of the acid is used in the production of agricultural fertilizers with the remainder being used for detergents, insecticides, cattle feed additives and water softener. The smallest portion, of the highest purity, is used in food and beverage industries as an acidulant and flavouring (E338), found particularly in carbonated cola drinks, beer, jams, jelly and cottage cheese (El-Asmy et al., 2008).

Phosphoric acid is a corrosive acid effecting the skin, mucous membranes and eyes, on contact with high concentrations. Historically it was used to treat lead poisoning. Symptoms of intoxication include diarrhoea, sour taste in mouth and coughing (Caravati, 1987).

7. Potency Selection

Many studies have used homoeopathic remedies on athletes in a 30CH often with negative or inconclusive result, resulting in another potency being indicated. The potency selected should match the energy of the disease but at the same time not be too strong for the patients vital force to handle. Adverse physiological effects of exercise have a high energy and the participants are healthy cyclists so a 200CH dosage is indicated, as it matches the energy of exertion. A 1M or 10M would not be indicated as the remedy will not be individually selected for each participant and could cause an aggravation (Vithoukias, 1985).

8. Posology

A homoeopathic remedy is typically administered once or twice over an extended period of time, with the object of aiding and assisting the individual's own healing process. Allopaths view drugs as physical molecular agents, only acting while present in the body by changing chemical reactions, such that higher doses produce bigger chemical response (Bell et al., 1999). A remedy acts as a activator to the body to heal itself, setting off a chain reaction, during this activation a change in the patient may be observed or experienced by the patient, it is recommended that treatment be suspended as soon as response is noted (Ledermann, 1969). One should wait for the remedy to act and not over stimulate the Vital force by repeated high doses. Repeating a remedy is only indicated when there is a relapse of symptoms or well indicated remedy has failed to act (Hacker, 1948).

9. Placebo Effect

It is widely accepted that psycho-social pressures can result in physiological changes, which can potentially initiate patho-physiological processes. It is therefore accepted that psychosomatic interference can have an effect on the body's physiology. However it is debatable whether psychological changes can curb or reverse established patho-physiological processes. Researchers have identified placebo effects on studies and have attempted to eliminate these, blinding and randomisation is often used. It has been noted, that an average of 60 percent of subjects experience some form of amelioration of symptoms following a placebo treatment (Kienle & Kiene, 1997).

To better understand the placebo effect an accurate definition is required. Historically the placebo effect was defined as 'any therapy or component of therapy that is used for its non-specific psychological or psychophysiological effect, or for it's presumed specific effect, but is without specific activity for the condition being treated' (Shapiro, Mike, Barten & Shapiro, 1973; Kienle & Kiene, 1997). However defining specific and non-specific effects proved problematic. Gotzsche (1994: 925) more accurately described it as 'The placebo effect is the difference in outcome between a placebo treated group and an untreated control group in an unbiased experiment'. However this definition is only functional when many participants are being tested and reflects the impossibility of defining the placebo effect in a single case as biases of various types can not be excluded. Often a false positive result in a study or in a clinical trial is due to placebo effects. However the placebo effect can be differentiated by its often characteristic pattern. This pattern includes a strong early improvement of

symptoms that fades back to original base values over time. This pattern is very different to the effect of homoeopathic treatment, which characteristically commences with an initial aggravation, followed by amelioration of symptoms that do not return to original baseline values (Peters, 2001).

F. LITERATURE STATEMENTS

1. Homoeopathy In Sport

Minimal literature was found with regards to homoeopathy and its ability to treat the stresses caused by exercise. Of the literature found, most of it pertained to remedies that can reduce the physiological effects of exercise after exercise, thus speeding up recovery (Vickers, Fisher, Smith, Wyllie & Lewith, 1997; Tveiten & Bruset, 2003). However, no literature could be found that tested changes in an athlete's economy and power output during performance. This study aims to investigate a remedy that is able to reduce the physiological effects of exercise during and after exercise, thus improving the athletes overall performance (Kayne, 1992).

Increasing performance homoeopathically assists the body systems already in place to improve the body's natural ability to deal with the stresses of exercise and speed up its recovery rate. This enhanced state of improved recovery, allows the body to repeatedly perform at its best with a shorter recovery period, therefore improving overall performance. This is especially important in sports that require repeated workloads, as cycling does. Unnatural ergonic aids, however, impose harmful stresses on the body by pushing it beyond its physiological capabilities (Thevis, Sigmund, Geyer & Schänzer, 2010).

Clinically, homoeopathic remedies assisted people to deal with many forms of stressors imposed by pathogens, lifestyle factors and environmental factors (Gutman, 1970). Generally, exercise is viewed as a healthy beneficial part of life. Signs and symptoms of over exercising are often ignored (Tveiten & Bruset, 2003; Plezbert & Burke, 2005), even though it causes damage to the body, resulting in the need for similar treatment.

La Grange (1999) performed a similar study utilizing *Sarcocollinum acidum* to investigate its effect on lactic acid accumulation and exercise fatigue. This study was, however, deficient and inaccurate due to limitations in technology accessible at the time. Deficiencies include heart rate and respiratory

gasses not being sampled and a stationary bike was mechanically braked using weights rather than electromagnetically, which is more accurate. The methodology for selecting his homoeopathic substance was also not in accordance with homoeopathic posology.

Vickers et al. (1997) conducted a study investigating the effect of Homoeopathy on delayed onset of muscle soreness (DOMS). This randomised double blind placebo controlled trials were conducted on 68 healthy volunteers. Participants undertook a ten minute period of bench stepping and were randomised to a homoeopathic medicine (a homoeopathic complex consisting of *Arnica montana* 30CH, *Rhus toxicodendron* 30CH and *Sarcolacticum acidum* 30CH) or placebo. The results were measured according to mean muscle soreness in the five day period after the exercise test. The results revealed the difference between group means was 0.17 in favour of placebo with 95 percent confidence intervals \pm 0.50. This study failed to prove any significant enhancement of the homoeopathic remedy to prevent muscle soreness. The researcher believes bench stepping was an inappropriate model to evaluate the effects of a homoeopathic treatment on DOMS because of wide variation between subject soreness scores.

Tveiten & Brusset (2003) also conducted a study to examine whether the homoeopathic medicine *Arnica montana* D30 had an effect on muscle soreness and cell damage after marathon running. Eighty two marathon runners were tested, while participating in the Oslo Marathon in 1990 and 1995. *Arnica montana* D30 or placebo was given morning and evening. Treatment started on the evening before the marathon and continued on day of the race and the three days following the race. The runners assessed muscular soreness on a visual analogue scale. Muscle enzymes, electrolytes and creatinine were measured before and after the marathon. Muscle soreness immediately after the marathon run was lower in the *Arnica montana* group than in the placebo group (P=0.04). Cell damage measured by enzymes was similar in the *Arnica montana* and the placebo group. These collective results suggest that *Arnica montana* D30 has a positive effect on muscle soreness after marathon running, but not on cell damage measured by enzymes. *Arnica montana* was a differential remedy on repertorisation, even though this study and *Materia Medica* knowledge proved that *Arnica montana* was a good recovery remedy, but did not enhance sport performance during the event.

2. Supplementation In Sport

It is generally accepted that diet, especially carbohydrate intake, plays a fundamental role in performance and recovery. This is especially true in the event of prolonged exercise (more than two hours). However, recent studies have shown that carbohydrate ingestion can also improve short duration exercise (less than an hour at greater than 75 percent $\text{vO}_{2\text{Max}}$) (El-Sayed, MacLaren & Rattu, 1997). The mechanism of improved performance is related to maintaining muscular fuel stores, bioavailability and maintenance of blood sugar levels. Carbohydrates ingested during exercise can be oxidised at a maximal rate of one gram per minute, even if large quantity are ingested. Combining more than one carbohydrate, e.g. glucose & fructose, use more than one transporter within the intestines and results in higher absorption and oxidation rate (Jeukendrup, 2004).

Healthcare organisations world wide, recommend that the general sedentary individuals to eat a balanced diet and regular exercise. The aim is to ensure a sense of well-being and health. It is well known that decreased intake of protein, fat and carbohydrates, have negative effects on performance and health. There is limited information on the effect of low intakes of vitamins and minerals on exercise and performance. Athletes however consume about the recommended amount for sedentary individuals. According to Lukaski (2004) severe deprivation of folate and vitamin B12 result in anaemia and reduced endurance work performance. Evidence of vitamin A and E deficiencies in athletes is lacking as the body stores large amounts of these vitamins. Iron deficiencies, with or without anaemia, causes muscular contraction and cardiovascular dysfunction. Magnesium deprivation increases oxygen demand which limits endurance capabilities. The converse is however not true, consuming increased levels of vitamins and minerals in supplements does not improve performance (Lukaski, 2004).

Buckley, Thomson, Coates, Howe, DeNichilo, & Rowney (2001) conducted a study to determine if whey protein can enhance skeletal muscle recovery, where 28 sedentary males were selected to participate in the study. Each participant completed two sets of 100 maximal eccentric contractions of their knee extensors. Muscle soreness, serum Creatine Kinase activity, plasma Tumour Necrosis Factor and Peak Isometric Torque were all used to plot muscle recovery. The initial test obtained baseline values and the subsequent test was used for comparison. Half the test subjects consumed a flavoured drink containing 25 grams whey protein and the other half consumed a flavoured placebo drink. It was concluded that the muscle recovery was better in the treatment (whey protein) group

than the placebo group. However whey protein had no effect on muscle damage as a result of eccentric contractions, as serum Creatine Kinase, a muscle damage marker, was the same in both groups.

Growth Hormone (GH) has been used by many athletes to enhance performance, however there is little evidence that it works. GH supplementation increases Lean Body Mass (LBM) in athletes however it is not certain whether this increase is due to an increase in muscle mass or an increase in Extra-Cellular Water (ECW). Protein anabolism in untrained individuals is increased fractionally however resistance training in untrained individuals has the same effect. During training, GH supplementation had no effect on protein metabolism. No studies could be found on GH's effect on elite athletes, as the adverse effects of such a study outweigh the possible efficacy. Side effects of GH supplementation range from mild symptoms including oedema, carpal tunnel syndrome, arthralgias, sweating, fatigue and dizziness and severe pathologies including diabetes, malignancy and myocarditis (Birzniece, Nelson & Ho, 2010).

3. Stellenbosch Mood Scale (STEMS) Questionnaire

Assessment of an athletes' mood is very important as it has a large effect on performance. A depressed mood is associated with confusion, fatigue and tension with reduced vigour and perceived readiness of exercise (Lane, 2001).

The Profile of Mood States (POMS) Questionnaire was developed in 1971 for people undergoing counselling or psychotherapy (McNair, Lorr & Droppelman, 1992). In 1975, the value of the questionnaire, which consists of 65 questions, was recognized by sportsmen and sportswomen and introduced into the sporting world. Since then, POMS has proved to be a valid assessment tool on performance status in athletes (Terry, Lane & Fogarty, 2003). The University of Stellenbosch developed the STEMS questionnaire which is an addition to the POMS questionnaire, modified to better assess athletes. Based on this, the questionnaire would prove a beneficial tool in assessing mood changes and aid in testing the hypothesis.

4. Numerical Rating Scale (NRS) Questionnaire

The measurement of the symptoms using a patient-rated 11 point (zero to ten) NRS was found to be both reliable and valid (Farrar, Troxel, Stott, Duncombe & Jensen, 2008). These values were a good indicator of changes in the participants' body between the two assessments (Appendix G). The reason a visual analogue scale (VAS) was not used is it would have to be converted by the researcher into a numerical value for statistical analysis. The NRS allowed this conversion to be done by the researcher. A VAS is problematic as the participant might draw the mark in the wrong place or a really large mark, making it difficult to analyse (Price, Patel, Robinson & Staud, 2008).

5. Velotron Ergometer

The Velotron Dynafit Pro is highly adjustable and thus caters for all different size cyclists. Adjustments cater for different leg length, arm length and preferred seat position in relation to the cranks and handle bars. The stainless steel construction provides a rigid frame which limits flexibility reducing energy dissipation.

The Velotron is computer controlled and its mechanical design provides the highest laboratory grade accuracy and repeatability. It has a wide load range and an authentic road feel that closely simulates cycling. The system uses an innovative patented, eddy-current brake, built around a heavy (25 kg), large diameter flywheel with an internal freewheel. It uses a fixed ratio, high efficiency chain-drive (Batz, 2005).

The Velotron is widely used in universities, sports science labs, and testing centres, often in conjunction with the optional Wingate Test software. Velotron's ultra-low starting load of five Watts is advantageous for rehabilitative cardiovascular, pulmonary and orthopaedic applications. On account of the exceptional accuracy of the Velotron, it is the only bicycle ergometer approved for use in the USA Cycling National Talent Search (Batz, 2005).

Figure 2.6 Velotron Ergometer (Batz, 2005)



Velotron creates loads from 5 to 2000 Watts which covers the entire range of human capability. Accuracy is ± 1.5 percent of reading across the entire load range with a repeatability of ± 0.2 percent. This level of accuracy and repeatability meets that of competitive laboratory bicycle ergometers (Baatz, 2005).

6. Continuous Graded Exercise Test

Graded exercise has been used extensively in many different situations including, testing response to exercise, cardiovascular respiratory function, rehabilitation and athlete training (White & Naish, 2001). It can either be performed on a treadmill or a cycle ergometer. Protocol can be adapted according to the needs of the researcher. Longer protocols are normally used on healthy individuals and are effective in demonstrating endurance. Shorter protocols can be used on sedentary individuals or to demonstrate peak power output. Protocols can either be submaximal or until voluntary exhaustion, with the latter only recommended for healthy individuals (Machado-Moreira et al., 2005). Increases in power can either be linear or stepped, most studies use stepped protocols as it allows for the body to reach a steady state. A steady state is achieved when the body reaches homeostasis and the participant is able to sustain this power for an extended period of time (Tucker, Bester, Lambert, Noakes, Vaughan & St Clair Gibson, 2006). Steady state is only possible below lactate threshold and is easier to demonstrate in fit individuals, as they are more resistant to fatigue (Laplaud, Talmud & Menier, 2005).

G. CONCLUSION

The popularity of cycling among all ages and ethnic groups has created a large demand for equipment, training programs, organisation of competitions, training routes, and more relevant to this study, treatment of symptoms caused by cycling and over training. Prolonged improper cycling can result in a wide array of signs and symptoms, and various strategies have been implemented to treat or prevent these symptoms. Homoeopathy has been proven to be effective in treating various sporting related injuries, whereas *Phosphoricum acidum* has been proven to be effective in many clinical situations.

CHAPTER THREE – RESEARCH METHODOLOGY

A. STUDY DESIGN

The study was a randomized controlled clinical trial, parallel group design. Participants were selected using convenience sampling of male road and mountain bike cyclists in the Western Cape. This study followed an explanatory Randomized Controlled Trial test, where the efficacy of the homoeopathic remedy (*Phosphoricum acidum 200CH*) was under investigation, participants were selected with great care and testing was completed under highly controlled conditions. Further more this study was a superiority trial, in which the homoeopathic intervention was hypothesized to be superior to the control group in a statistically significant way ($p < 0.05$).

B. EXPERIMENTAL INTERVENTION

1. Subjects

The sample group consisted of 30 well-trained male cyclists, recruited to participate in a randomised double-blinded placebo test. More specifically, the study consisted of two groups: one on *Phosphoricum acidum 200CH* and the other on an identical placebo. The researcher and the participants did not know which group the participants were in until the study was completed. The supervisor randomly assigned treatment or placebo to the participants.

Participants were recruited through advertisements on Stellenbosch notice boards, cycle shops, as well as from local cycling clubs (Appendix H). All participants were questioned and vital signs tested, to ensure they met the inclusion and exclusion criteria.

2. Inclusion And Exclusion Criteria

Individuals Were Included In The Study If:

1. They were male.
2. They were between 18 and 55 years of age, on the day of testing.

3. They had in the 3 months preceding the test cycled an average of two hours and completed a minimum of 60 km per week.
4. They had completed a 100km road race in three hours and 30 minutes or less in the past 12 months preceding the test.
5. They were willing to participate (Appendix D).

Individuals Were Excluded From The Study If:

1. Vitals on the test day were outside normal ranges (Bickley, Szilagyi, Bates & Prabhu, 2007)
 - Blood pressure of more than 140/110 mmHg (Hypotension) or less than 90/50 mmHg (Hypotension).
 - Core body temperature was less than 34 degrees Celsius (Hypothermia) or greater than 37.5 degrees Celsius (Fever)
 - Respiratory rate less than ten breaths per minute (Hypoventilation) or higher than 20 breaths per minute (Tachypnea)
 - Heart Rate less than 45 beats per minute (Bradycardia) or greater than 100 beats per minute (Tachycardia)
 - Weight / height² = BMI (kg/m²) less than 16.5 (anorexia) or greater than 35 (Obesity)
2. They had a cold or upper respiratory infection on the day of testing.
3. They had any medical condition that inhibited their exercise capacity or which may alter their physiological responses to exercise.
4. They were on drugs, ergogenic aids or other substances (medical drugs (acute or chronic) beta blocker, antibiotics, diuretics, thermogenics or recreational drugs)

3. Experimental Protocol

To test performance, one needs to adequately stimulate the body ensuring a fine balance between over and under stimulation. At low intensity work loads, small variations in performance can be difficult to observe and measure, whereas at moderate intensities a small change can easily be observed. However if the body is over stressed, the athlete may not be able to complete the test, resulting in no further data being able to be obtained. Constructing a testing method that ensured adequate stimulation to all the participants was accomplished by using average values obtained in previous studies (Morris, Myers, Froelicher, Kawaguchi, Ueshima & Hideg, 1993) see *Table 3.1*. The

stimulation was measured in Metabolic Equivalents (METS) at different workloads measured in Watts. The higher the METS the higher the biomechanical and physiological demand (Powers & Howley, 2007).

Table 3.1 Stationary Cycling

Metabolic Equivalents	Power (Watts)
5.5	100, Light effort
7.0	150, moderate effort
10.5	200, vigorous
12.5	250, very vigorous

The participants were instructed to abstain from exercise for 24 hours before testing. Eating and drinking was prohibited three hours before testing, participants were further instructed to refrain from stimulants (e.g. coffee, alcohol) four hours before testing. Drinking water was permitted before testing (Appendix C).

Each cyclist was, tested individually, remained seated during the test and pedalled between 80 and 100 rpm. The subjects performed two power interval tests on a stationary trainer, (*Excalibur Sport*®, Lode, Groningen, Netherlands) each power interval consisted of a warm up, a constant load, an ascending load until exhaustion and a passive recovery. The protocol was then repeated after administration of *Phosphoricum acidum* and a 30 minute recovery period as illustrated (*Figure 3.1* and *Figure 3.2*).

The interval test consisted of a ten minute warm-up at 100 Watts, followed by a five minute constant load at 150 Watts. Warming up before an exercise can improve performance and decrease injury rates, provided that the warm-up adequately elevates the body temperature and incorporates movements similar to exercise movement, duration of warm-up has little effect (Fradkin, Gabbe & Cameron, 2006). The purpose of the constant load was to test cycling economy. A five minute interval was used as blood lactate levels take three minutes to plateau and the further two minutes allowed time to perform the lactate test (Siciliano, Laura, Renna, Prontera, Mercuri & Murri, 2000). Thereafter the workload was increased to 200 W for 30 seconds and then the workload increased by 20 Watts every 30 seconds. A standardised incremental protocol could not be found in literature, as different ergometers have different capabilities and functions (Jammes, Steinberg, Brégeon & Delliaux, 2004;

Sabapathy, et al., 2006; Lenti, Sbriccoli, Scotto & Sacchetti, 2010). However many studies increased work load every 30 seconds until exhaustion. The test continued until the participant could not maintain the set repetitions per minute for that workload. The peak power output attained was recorded.

Following peak power output, $\dot{V}O_2$ and heart rate was monitored for a further three minutes during passive rest, during which, averages were calculated for the first 60 seconds and the last 60 seconds. The participants were instructed to climb off the bike and do five air squats with their feet shoulder width apart. A STEMS questionnaire was administered and lower extremity (leg & thigh) muscle pain, nausea, headache and dizziness was noted on a Numerical Rating Scale (NRS). This concluded the first power interval test, obtaining baseline values for each participant.

4. Passive Recovery

A 30 minute recovery followed, where participants were allowed to relax, re-hydrate using water and walking short distances (passive rest). According to Denadai & Higino (2004) a recovery of eight minutes or longer is sufficient for an athlete to recover between incremental exercise tests, the longer time was given to prepare equipment for second test, to complete questionnaires and for remedy to take action.

5. Place Of Study

All tests were conducted at the Sport Physiology Laboratory of the Department of Sport Science, Stellenbosch University by the researcher with the assistance of a Stellenbosch Sport Physiology Laboratory Technician. The room temperature during testing was maintained between 19 and 21 degrees Celsius.

6. Medicine Preparation

The 30 powders containing *Phosphoricum acidum 200CH* or the placebo were prepared by a research supervisor at the Durban University of Technology Homoeopathic day clinic. A medicating solution of *Phosphoric acidum 200CH* was used, purchased from Natura Laboratories which prepared the solution according to (Appendix B). The treatment powders were impregnated with one

drop of *Phosphoricum acidum 200CH*, and the placebo powders were impregnated with one drop 96 percent ethanol. Both sets of powders were packaged and labelled identically (Benyunes, 2005).

7. Dispensing Medication

At the beginning of the 30 minute recovery, one powder (*Phosphoricum acidum 200CH* or an identical placebo) was administered sublingually to each participant (according to the randomisation table Appendix A). The medication was dispensed by a qualified Homoeopath (Dr A. Haw), who acted as the clinician.

Figure 3.1 One Test Interval

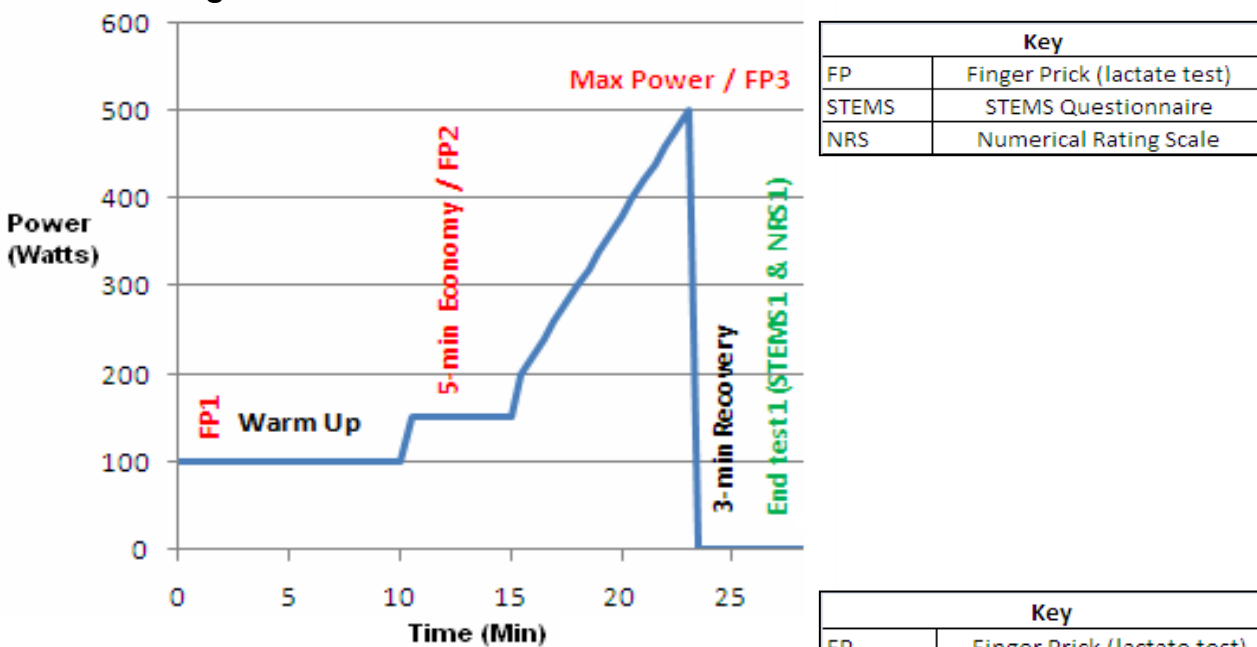
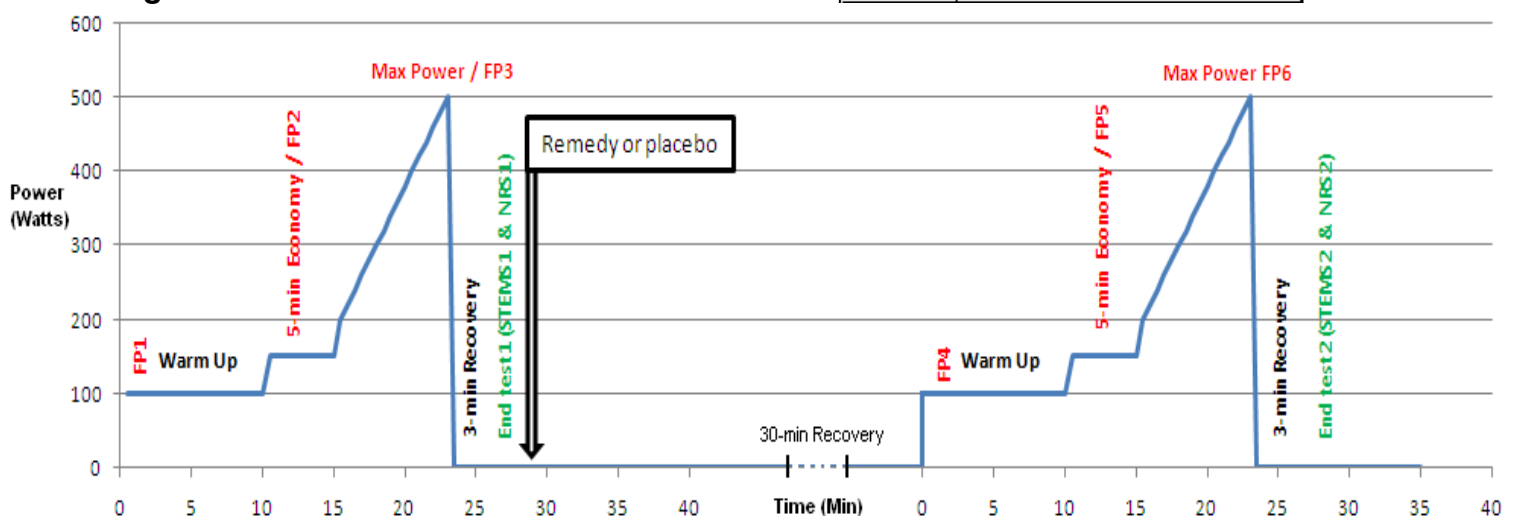


Figure 3.2 Test Protocol



C. DATA COLLECTION

1. Resting Measurements

Resting measurements were performed on each participant (Appendix E), after they were allowed to relax for two minutes. Participants lay prone, with the diagnostic bed at the 45 degree position. Resting heart rate was measured for 60 seconds using the radial pulse. Blood pressure was measured using a Blood pressure cuff (WelchAllyn®, New York, USA) and Stethoscope (Littmann®, Borken, Germany) ensuring the arm was aligned with the heart. Body core temperature was measured using an electronic thermometer (ThermoVal®, Hartmann, China).

2. Lactate Testing

A finger prick blood test was conducted by the researcher with the assistance of a Stellenbosch Sport Physiology Laboratory technician using a lancet device (*Accu-Chek*®, Roche, Mannheim, Germany) to determine blood lactate concentration, using a *Lactate Pro* device (Arkray Inc., Kyoto, Japan). A different finger on the participants' non-dominant hand was used for each sample. The finger was first cleaned using an alcohol swabs to reduce sample contamination and the finger, once pricked, was allowed to passively bleed and not compressed, as this could dilute the sample by allowing extra cellular fluid to mix with the sample. Blood samples were taken before, 15 minutes into the interval and directly after peak power output, for each participant.

3. Peak Power Output (PPO)

Peak power output (PPO) was calculated by the addition of the last completed work load and the proportion of the final work load completed, measured in seconds.

Formula 3.1 Peak Power Output (Bentley & McNaughton, 2003)

$$\text{PPO} = [\text{Completed work load}_{(\text{Watts})}] + [20_{(\text{Watts})} / 30_{(\text{seconds})} \times \text{seconds in final work load}]$$

4. Metabolic Testing

Throughout the test, breath-by-breath gases were continuously recorded. Expired gases, volumes and air flow were sampled through a flow meter and gas sampling line and heart rate was measured through telemetry (*Polar®*, Polar Electro, Oy, Finland) and analyzed by a cardio-pulmonary metabolic system (*Quark CPET®* Cosmed, Rome, Italy, 2009). Data recorded was filtered for values outside the normal ranges and averaged for every five seconds. Oxygen consumption ($\dot{V}O_2$ mL.min⁻¹) and heart rate (bpm) at different stages of each test interval were recorded. More specifically oxygen consumption and heart rates were averaged over the 5 minute section following the warm-up to determine cycling economy, directly after exhaustion for one minute and two minutes following exhaustion for another one minute period. Maximum attained heart rate was recorded for each interval test. Maximum oxygen consumption ($\dot{V}O_{2Max}$ Absolute) was calculated as the mean of the highest three values attained, this mean was then divided by the participants body mass to determine maximum aerobic capacity ($\dot{V}O_{2Max}$ Relative).

5. Numerical Rating Scale (NRS) Questionnaire

A NRS questionnaire was applied to five symptomatic questions, zero indicated no symptom and ten the most severe form of that symptom. Symptoms under investigation included (Appendix G):

- Lower limb muscle pain
- Lower limb stiffness,
- Nausea,
- Headache
- Dizziness

6. Stellenbosch Mood Scale (STEMS) Questionnaire

A Stellenbosch Mood Scale (STEMS) questionnaire (Appendix F) and was used to assess each participant's mental response to exercise before and after treatment.

Table 3.2 Stellenbosch Mood Scale

Category	Question	Question Number
Anger	annoyed, bitten, angry, bad-tempered	7, 11, 19, 22
Confusion	confused mixed-up, muddled, uncertain	3, 9, 17, 24
Depression	depressed, downhearted, unhappy, miserable	5, 6, 12, 16
Fatigue	worn-out, exhausted sleepy tired	4, 8, 10, 21
Tension	panicky, anxious, worried, nervous	1, 13, 14, 18
Vigour	lively, energetic, active, alert	2, 15, 20, 23

Table 3.3 Adult Athlete T-Scores

	Anger	Confusion	Depression	Fatigue	Tension	Vigour
0	45	42	45	40	37	29
1	52	46	52	44	40	32
2	58	50	58	47	43	34
3	65	54	64	51	46	37
4	71	58	70	54	49	39
5	78	62	77	58	52	42
6	84	66	83	61	55	44
7	91	70	89	65	58	47
8	98	74	95	68	61	49
9	104	77	102	72	64	52
10	111	82	108	75	67	55
11	117	86	114	79	70	57
12	124	90	120	82	72	60
13	130	94	127	86	75	62
14	137	98	133	89	78	65
15	143	102	139	93	81	67
16	150	106	145	96	84	70

D. DATA ANALYSIS

Two of the following three factors needed to be reached to ensure that all subjects fulfilled at least two of the following three criteria for $\dot{V}O_{2\text{Max}}$:

- A maximum heart rate of 90 percent of the age predicted maximal should be expected.
- A blood lactate value of greater than 8mmol is one of the factors that indicate a maximal intensity test.
- Respiratory exchange ratio (RER) greater than 1.1(Caputo, & Denadai, 2009).

Kidd (2010) advised that the level of significance was set at $P < 0.05$, effect size = 1, power $(1 - \beta) = 0.80$, for all analysis. The recommended sample size was 15 participants per group ($2 \times 15 = 30$), thus a total of 30 participants.

Statistical analysis was done with Statistica software[®] (version 8 STATSOFT, United States of America) and Microsoft Office Excel 2007. Descriptive data was reported as mean (\bar{x}) and Standard deviation (SD), unless otherwise specified. Differences between variables was assessed through a two-way ANOVA for repeated measures on all outcome variables (a multivariate analysis), to assess the effect of *Phosphoricum acidum 200CH* on cycling recovery and performance. The main group effect and time effect were two independent factors, with the interaction effect (group*time) as a dependent factor. Values from test one and test two for both treatment and control group were compared using a t-Test and Cohen's test. Percentage change (*Formula 3.3*) between test one and test two for both treatment and control group were compared using a t-Test (Two-Sample Assuming Equal Variances). Cohen's d was used to determine effect size (Speed & Andersen, 2000).

Formula 3.2 Cohen's d Effect Size (Speed & Andersen, 2000).

$$d = \frac{\bar{x}_t - \bar{x}_c}{\sqrt{\frac{(n_t - 1)s_t^2 + (n_c - 1)s_c^2}{n_t + n_c}}}$$

Formula 3.3 Percentage Change

$$\text{Percentage change} = (\text{Test2} - \text{Test1}) / \text{Test1} \times 100$$

E. CONCLUSION

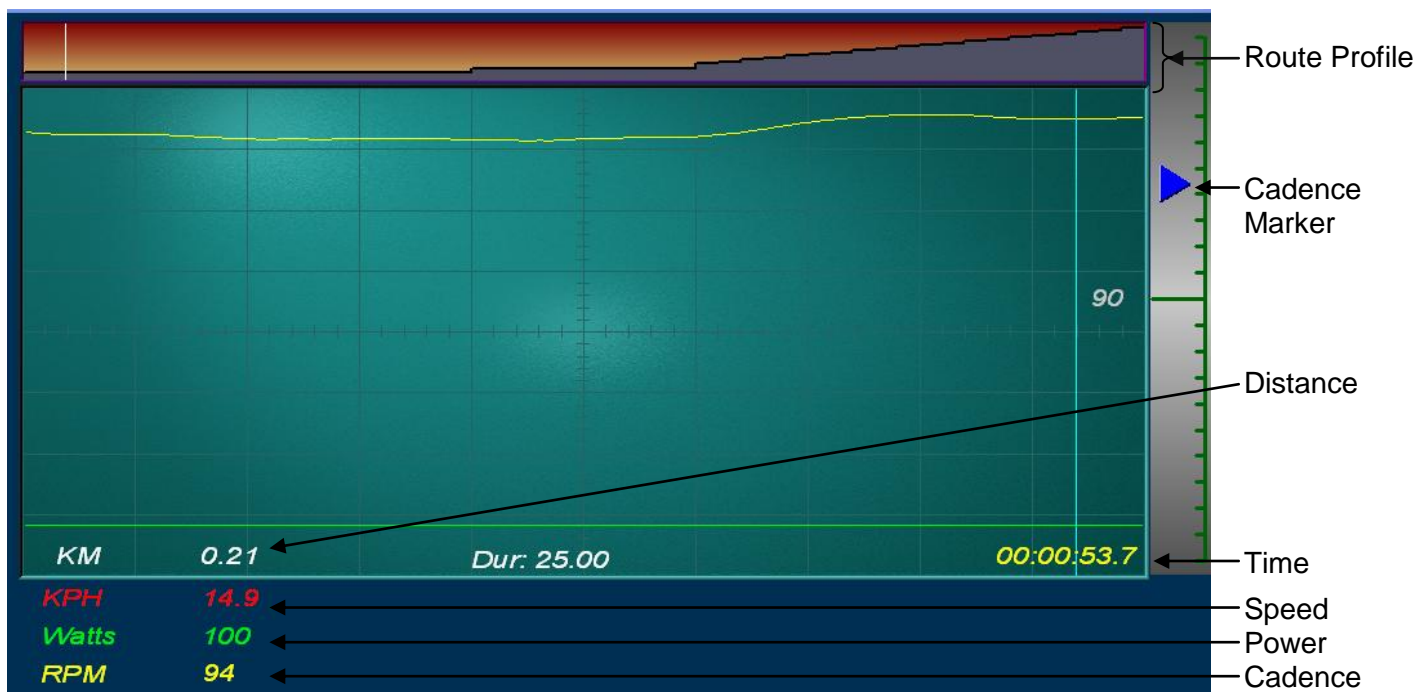
This study endeavoured to investigate the effects of *Phosphoricum acidum 200CH* on cyclists' performance and recovery, using a simple, cost-effective, reliable and time efficient testing protocol. The testing protocol ensured accurate, as well as adequate data of practical importance that could easily be analysed statistically. The testing protocol was short enough to reduce the chances of fuel store depletion, however sufficient in duration to adequately stimulate each participant to their maximum capabilities.

CHAPTER FOUR: RESULTS

A. INTRODUCTION

The study aimed to investigate the possible effect of *Phosphoricum acidum 200CH* on cycling performance and recovery in experienced cyclists during and after two incremental power to exhaustion tests. A double blinded placebo controlled study design was used, with each cyclist acting as his own control in the first incremental test. Selected variables included blood lactate concentration, heart rate, oxygen consumption and peak power output. Questionnaires were also used to track mental and physical symptoms.

Figure 4.1 Computer Display



During testing the participants were allowed to see their resting vital results (Bp, HR, core temperature, breathing rate, height, weight), lactate results (Appendix E) and all data on the computer screen (Figure 4.1). Computer data visible to participants during the testing included route profile, cadence, distance, test duration speed and power. The reason for allowing the participant to see computer data was that the participants needed to see their cadence to be able to maintain it between 80 and 100 rpm.

Aim:

- To test the effect of *Phosphoricum acidum 200CH* on the recovery rate of cyclists
- To test the effect of *Phosphoricum acidum 200CH* on the blood lactate concentration of cyclists
- To test the effect of *Phosphoricum acidum 200CH* on oxygen consumption of cyclists
- To test the effect of *Phosphoricum acidum 200CH* on peak power output of cyclists
- To test the effect of *Phosphoricum acidum 200CH* on the emotional status of cyclists

Hypothesis:

- The administration of *Phosphoricum acidum 200CH* would decrease blood lactate accumulation in the body
- The administration of *Phosphoricum acidum 200CH* would increase peak power output of cyclists
- The administration of *Phosphoricum acidum 200CH* would decrease submaximal oxygen consumption during performance and recovery
- The administration of *Phosphoricum acidum 200CH* would increase maximal oxygen consumption during performance
- The administration of *Phosphoricum acidum 200CH* would decrease heart rate during performance and recovery
- The administration of *Phosphoricum acidum 200CH* would decrease lower limb muscle pain, lower limb muscle stiffness, nausea, headache and dizziness symptom severity
- The administration of *Phosphoricum acidum 200CH* would decrease negative emotions (anger, confusion, depression, fatigue, tension) and increase positive emotions (vigour) induced by cycling.
- The administration of *Phosphoricum acidum 200CH* would improve the recovery rate of cyclists

B. DESCRIPTIVE CHARACTERISTICS

1. Subjects

The cyclists' physical and physiological characteristics measured before testing are presented in *Table 4.1*. The subjects were young to middle aged (between 19 and 47 years), cyclists competed at club level with a minimum cycling experience of two years, having completed a 100km cycle race in less than three hours and 30 minutes in the last 12 months, demonstrating an average speed greater than 29 kilometres per hour during the event. The average Body Mass Index (BMI) was in the upper limit of the normal range of 22 to 25.5 for sedentary individuals (24.4 ± 3.2); however endurance athletes normally have a BMI between 19 and 22. BMI is however, not very accurate for athletes, as it does not account for a high muscle and low fat indices, which is often seen in endurance athletes.

Table 4.1 Resting Characteristics of Participants ($n = 30$)

Variables	Mean (\bar{x})	\pm	SD
Age (years)	29.8	\pm	9.9
Height (cm)	175.4	\pm	9.2
Body mass (kg)	75.5	\pm	14.2
BMI ($\text{kg}\cdot\text{m}^{-2}$)	24.4	\pm	3.2
Resting HR (bpm)	60.8	\pm	11.3
Resting blood lactate (mmol)	2.4	\pm	0.9

BMI= body mass index; HR= heart rate

2. Maximum Cardiovascular Response

Table 4.2 summarizes the maximum physiological responses to the incremental baseline (first test) testing performed on a stationary trainer. The average maximum aerobic capacity ($\text{vO}_{2\text{Max}}$ $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) of the subjects falls within the “excellent” range for their age (Heyward, 1998).

Table 4.2 Maximum Exercise Capacity ($n = 30$)

Variables	Mean (\bar{x})	\pm	SD
vO_{2Max} (ml.kg⁻¹.min⁻¹)	55.8	\pm	7.0
vO_{2Max} (ml.min⁻¹)	4156	\pm	643
Maximum heart rate (bpm)	188	\pm	10.5
Age predicted maximum heart rate (%)	98.9%		
Maximum lactate (mmol)	13.9	\pm	2.4

vO_{2Max}, maximum aerobic capacity

3. Peak Power Output (PPO)

Peak power output variables are summarised in *Table 4.3*. According to the participants' average PPO and power to weight ratio (power/weight), the participants in the current study, ranked into the "Well Trained" cyclist category (Jeukendrup *et al.*, 2000).

Table 4.3 Peak Power Output ($n = 30$)

Test Results	\bar{x}	\pm	SD
Peak power output (W)	382	\pm	57
Power / weight (W.kg⁻¹)	5.1	\pm	0.6

C. TREATMENT EFFECT

1. Blood Lactate Concentration

Figure 4.2 illustrates resting blood lactate values which were significantly elevated above test one (baseline) concentrations in the second test, in both groups ($P < 0.001$). However, no significant interaction effect was observed between the groups for resting blood lactate ($P = 0.76$), with a negligible practical effect ($d=0.11$).

Figure 4.2 Resting Blood Lactate Concentrations ($n = 30$)

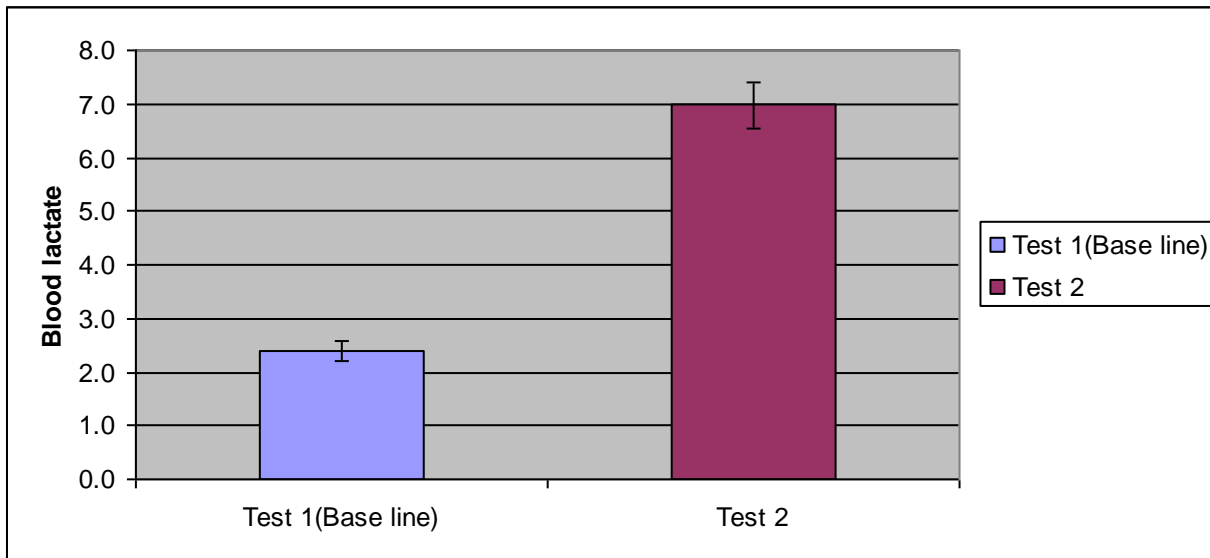
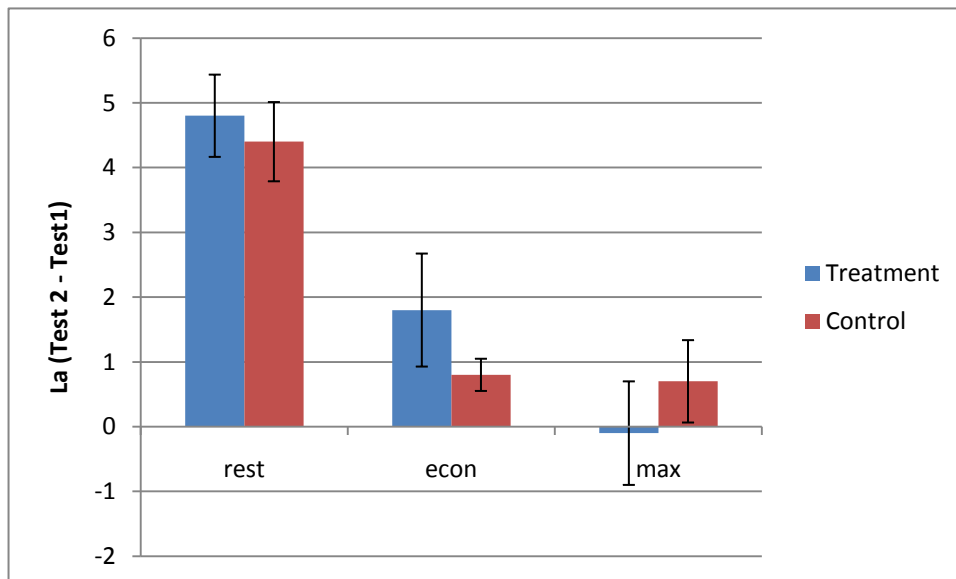


Figure 4.3 illustrates the difference in blood lactate concentration between the two tests, at different time points. At the economy time point, the treatment group had a greater change in blood lactate concentration compared to the control group and a smaller change at the final (maximum) time point.

Figure 4.3 Difference Between Test 1 And Test 2 Regarding Blood Lactate ($n=30$)



Rest=Resting; Econ= Economy; Max= Maximum; La= lactate

Table 4.4 demonstrates a medium practical effect on the economy lactate in favour of the control group and a small practical effect on maximum lactate in favour of the treatment group.

Table 4.4 Blood Lactate Results ($n = 30$)

Test Results		Treatment ($n=15$)	Control ($n=15$)	Effect Size	Effect size
		(mmol)	(mmol)	Test ^T Test ^C	% Δ ^T % Δ ^C
		$\bar{x} \pm SD$	$\bar{x} \pm SD$	d-value	d-value
Resting	Test 1	2.6 \pm 1.2	2.2 \pm 0.7	0.41 ^M	0.11 ^N
	Test 2	7.4 \pm 2.4	6.5 \pm 2.4	0.40 ^M	
Economy	Test 1	3.9 \pm 2.7	2.8 \pm 1.4	0.52 ^M	0.43 ^M
	Test 2	5.7 \pm 4.0	3.7 \pm 1.5	0.66 ^M	
Maximum	Test 1	13.6 \pm 2.9	14.3 \pm 1.8	0.30 ^S	0.2 ^S
	Test 2	13.6 \pm 3.5	15.0 \pm 2.6	0.47 ^M	

% Δ percentage change; ^Ttreatment; ^Ccontrol; *Effect size*: ^Nnegligible; ^Ssmall; ^Mmedium

As illustrated in *Table 4.5* the major observable effect on blood lactate in this study was seen in percentage changes between economy blood lactate concentration and maximum lactate concentration in the second test ($p=0.01$), with a very large practical effect. This change was only significantly different following treatment, as there was no difference between the two groups ($p=0.49$) in the first test (baseline) with a small practical effect. A medium effect was observed in test two, when comparing lactate changes between resting and maximum lactate ($p=0.06$), with no difference observed in the first test ($p=0.58$). There was a medium effect during the 30 minute recovery in favour of the control group.

Table 4.5 Percentage Blood Lactate Change Between Time Points ($n=30$)

	Blood Lactate	Treatment ($n=15$)	Control ($n=15$)	T-test p -value	Effect size d -value
Test one	Resting	2.6	2.2		
	Economy	3.9	2.8	0.58	0.21 ^S
	Maximum	13.6	14.3	0.49	0.27 ^S
30 minute Recovery					
Test two	Resting	7.4	6.5		
	Economy	5.7	3.7	0.06	0.75 ^L
	Maximum	13.6	15.0	0.01*	1.14 ^{VL}

* Significant difference between Treatment and Control group ($p < 0.05$). Effect size: ^Nnegligible; ^Ssmall; ^Mmedium; ^Llarge; ^{VL}very large

It was hypothesised that the administration of *Phosphoricum acidum 200CH* would decrease blood lactate accumulation in the body. From these results it is thus evident that the hypothesis is accepted as having a small to very large practical effect on maximum lactate accumulation.

2. Peak Power Output (PPO)

There was no time ($p=0.86$), group effect ($p=0.88$) or practical ($d=0.06$) significant effect between the two groups. *Figure 4.4* demonstrates that the control group improved by 3.5 Watts and the treatment group improved by two Watts, with a medium practical effect, however there was no practical difference the two groups. *Table 4.6* illustrated that the control group achieved a higher peak power

output in the first (baseline) and second test with a medium practical advantage over the treatment group in both tests. *Figure 4.5* illustrates that both groups achieved a higher peak power output in the second test.

Figure 4.4 Peak Power Output ($n = 30$)

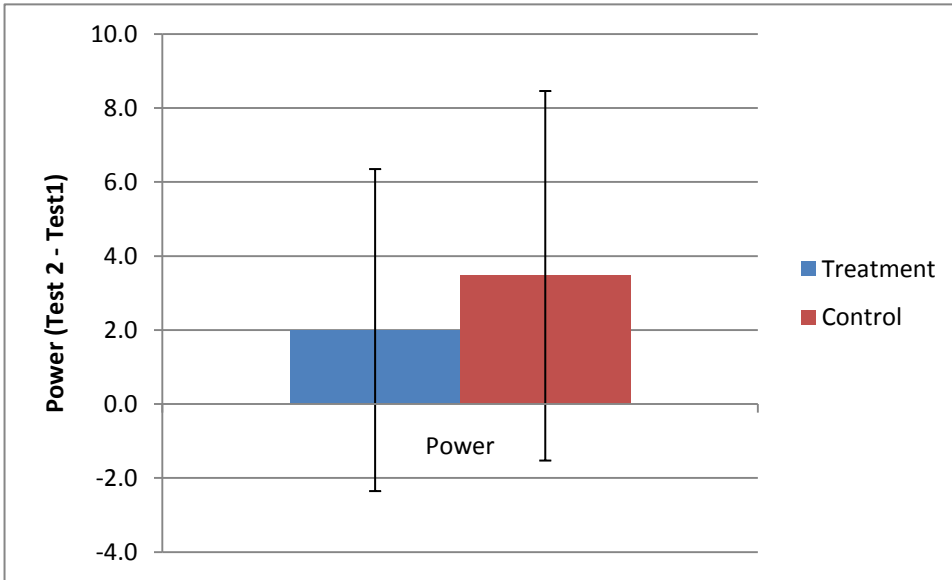


Figure 4.5 Peak Power Output ($n = 30$)

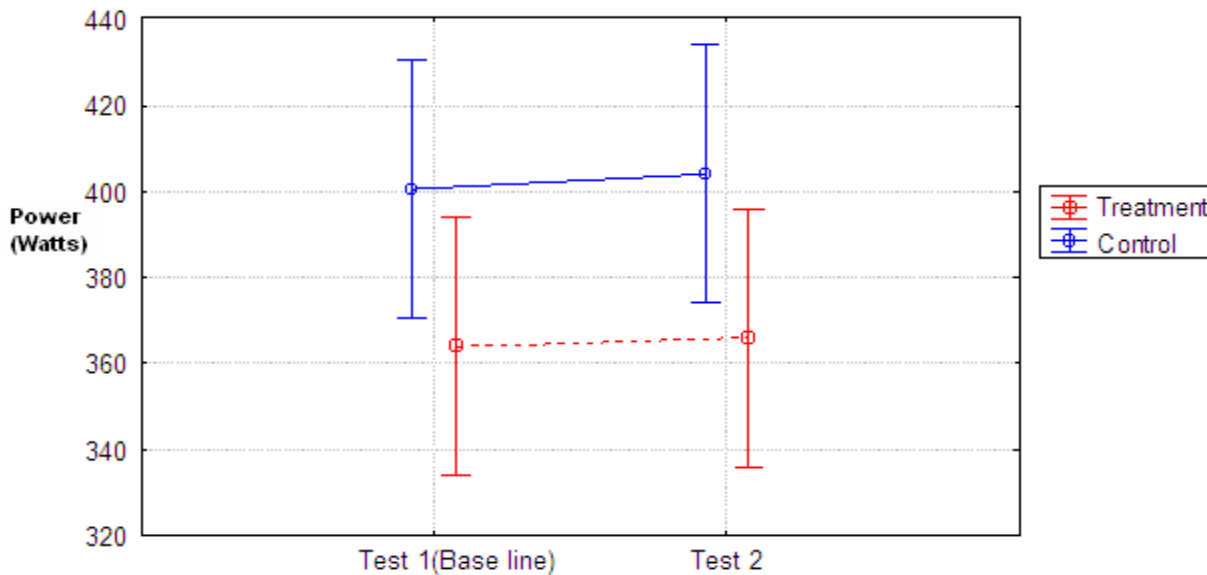


Table 4.6 Peak Power Output ($n = 30$)

Test Results	Treatment	Control	Effect Size	
	($n=15$)	($n=15$)	Test ^T	Test ^C
	(Watts)	(Watts)	% Δ^T	% Δ^C
	$\bar{x} \pm SD$	$\bar{x} \pm SD$	<i>d</i> -value	<i>d</i> -value
Test 1	364 \pm 54	401 \pm 56	0.71 ^M	
Test 2	366 \pm 56	404 \pm 61	0.70 ^M	0.06 ^N

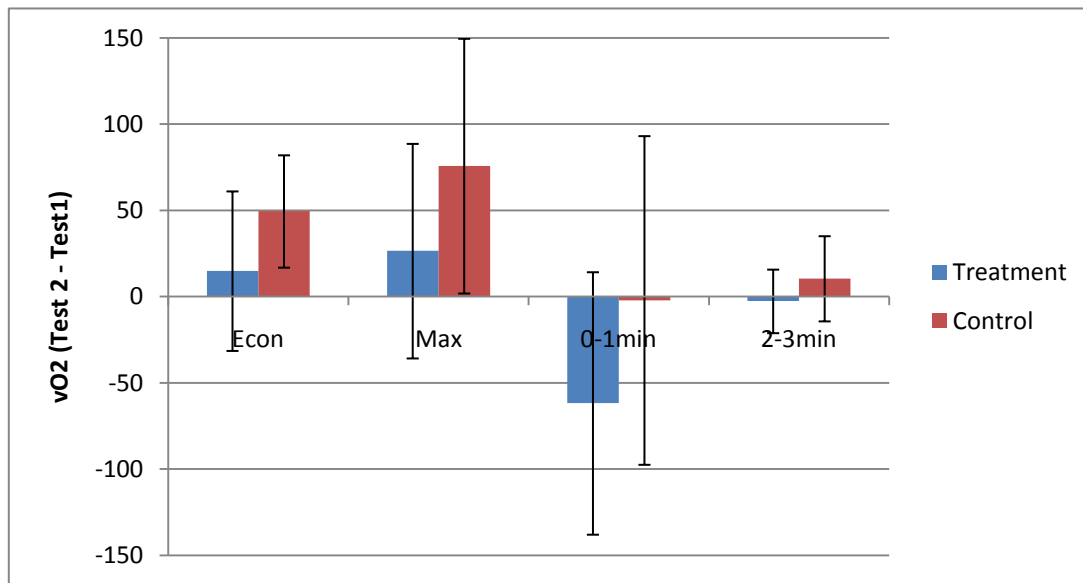
% Δ percentage change; ^Ttreatment; ^Ccontrol; *Effect size*: ^Nnegligible; ^Mmedium

It was hypothesised that the administration of *Phosphoricum acidum 200CH* would increase the peak power output of cyclists. From these results it is thus evident that the hypothesis is rejected.

3. Oxygen Consumption

Figure 4.6 illustrates that the treatment group had a smaller relative change in oxygen consumption at all time points, with a negligible to small practical difference (*Table 4.7*). *Table 4.7* illustrates that the control group consumed less oxygen during the economy time point for both tests with a negligible to small practical difference. However the control group showed a greater deterioration of oxygen economy in the second test than the treatment group. The control group showed a higher peak aerobic capacity (medium practical advantage), demonstrating a greater talent. Even though the control group demonstrated a greater talent, the control group performed worse during the recovery periods, with small to large practical differences.

Figure 4.6 Differences Between Test 1 And Test 2 Regarding Oxygen Consumption ($n= 30$)



vO_2 = Oxygen Consumption ($ml.min^{-1}$)

Table 4.7 Oxygen Consumption ($n = 30$)

Test Results		Treatment ($n=15$)	Control ($n=15$)	Effect Size	Effect Size
		($ml.min^{-1}$)	($ml.min^{-1}$)	Test ^T	Test ^C
		$\bar{x} \pm SD$	$\bar{x} \pm SD$	d-value	d-value
Econ	Test 1	2357 ± 235	2318 ± 205	0.19 ^S	0.2 ^S
	Test 2	2372 ± 213	2368 ± 218	0.02 ^N	
Max	Test 1	3981 ± 644	4332 ± 612	0.59 ^M	0.1 ^N
	Test 2	4007 ± 546	4407 ± 655	0.72 ^M	
0-1 min	Test 1	2598 ± 524	2949 ± 590	0.68 ^M	0.24 ^S
	Test 2	2536 ± 475	2947 ± 454	0.94 ^L *	
2-3 min	Test 1	926 ± 199	990 ± 213	0.33 ^S	0.14 ^N
	Test 2	923 ± 202	1000 ± 214	0.40 ^M	

* Significant difference between Treatment and Control, ($p=0.02$); % Δ percentage change; ^Ttreatment; ^Ccontrol; Econ= Economy; Max= Maximum; 0-1 min= average one minute post exhaustion; 2-3 min= average two to three minutes post exhaustion; Effect size: ^Nnegligible; ^Ssmall; ^Mmedium; ^Llarge

It was hypothesised that the administration of *Phosphoricum acidum 200CH* would decrease submaximal oxygen consumption during performance and recovery. From these results it is thus evident that the hypothesis is accepted having a small practical effect during economy and recovery.

It was hypothesised that the administration of *Phosphoricum acidum 200CH* would increase maximal oxygen consumption during performance. From these results it is thus evident that the hypothesis is rejected having a negligible practical effect.

4. Heart Rate

Heart rate is an indicator of effort level with regards to performance, with lower levels indicating lower levels of exertion. *Figure 4.7* illustrates that the treatment group showed a smaller change in maximum heart rate and average heart rate for one minute following peak power output, with a medium practical significance (*Table 4.8*). The average heart rate, two and three minute after peak power output time point, showed a small practical significant change in favour of the control group (*Table 4.8*).

Figure 4.7 Heart Rate ($n = 30$)

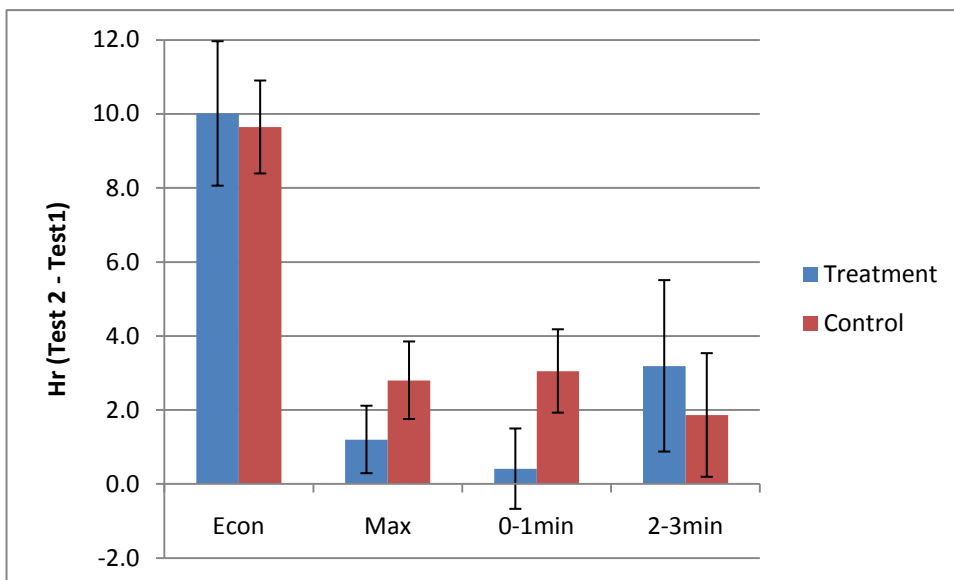


Table 4.8 Heart Rate ($n = 30$)

Test Results		Treatment ($n=15$)	Control ($n=15$)	Effect Size	Effect Size
		(bpm)	(bpm)	Test ^T Test ^C	% Δ^T % Δ^C
		$\bar{x} \pm SD$	$\bar{x} \pm SD$	d-value	d-value
Econ	Test 1	138 \pm 15.0	132 \pm 14.8	0.43 ^M	0 ^N
	Test 2	148 \pm 14.5	142 \pm 14.5	0.44 ^M	
Max	Test 1	188 \pm 12.4	187 \pm 8.6	0.1 ^N	0.48 ^M
	Test 2	189 \pm 13.0	190 \pm 7.5	0.1 ^N	
0-1 min	Test 1	173 \pm 13.4	175 \pm 8.0	0.48 ^M	0.66 ^M
	Test 2	173 \pm 13.8	178 \pm 7.1	0.46 ^M	
2-3 min	Test 1	119 \pm 14.2	126 \pm 10.4	0.22 ^S	0.2 ^S
	Test 2	122 \pm 14.4	127 \pm 9.7	0.0 ^N	

% Δ percentage change; ^Ttreatment; ^Ccontrol; Econ=Economy; Max=Maximum; 0-1 min= average one minute post exhaustion; 2-3 min= average two to three minutes post exhaustion; *Effect size*: ^Nnegligible; ^Ssmall; ^Mmedium

It was hypothesised that the administration of *Phosphoricum acidum 200CH* would decrease heart rate during performance and recovery. From these results it is thus evident that the hypothesis is accepted having a medium practical effect on maximum heart rate and average heart rate for one minute following peak power output.

5. Numerical Rating Scale (NRS) Questionnaire

The numerical rating scale, regarding lower limb muscle pain, lower limb stiffness, nausea, headache and dizziness, administered following each test interval showed no significant time or group effect ($p > 0.05$). The treatment group displayed higher means in both tests, at every time point, with a medium to huge practical difference (*Table 4.9*). There was a small practical group effect for lower limb muscle pain and headache, in favour of the control group (*Table 4.9*). It was observed that nausea, headache and dizziness symptoms were not experienced to a high degree by the participants in both groups, with means of two or lower, ranking into mild category. Lower limb pain and stiffness was experience as 4.4 or less, ranking into mild to moderate category.

Table 4.9 Numerical Rating Scale Symptom Results ($n = 30$)

Test Results		Treatment	Control	Effect Size		
		($n=15$)	($n=15$)	Test ^T	Test ^C	% Δ ^T % Δ ^C
		$\bar{x} \pm SD$	$\bar{x} \pm SD$	d -value	d -value	
Lower Limb	Test 1	3.7 \pm 0.61	2.4 \pm 0.61	2.27 ^H	0.36 ^S	
Muscle Pain	Test 2	4.4 \pm 0.77	3.1 \pm 0.68	1.18 ^H		
Lower Limb	Test 1	3.5 \pm 0.64	3.2 \pm 0.68	0.49 ^M	0.12 ^N	
Muscle Stiffness	Test 2	4.1 \pm 0.72	3.7 \pm 0.77	0.58 ^M		
Nausea	Test 1	1.9 \pm 0.76	0.7 \pm 0.53	1.89 ^H	0.11 ^N	
	Test 2	1.3 \pm 0.55	0.5 \pm 0.35	1.77 ^H		
Headache	Test 1	0.7 \pm 0.42	0.5 \pm 0.40	0.52 ^M	0.39 ^S	
	Test 2	0.8 \pm 0.39	0.3 \pm 0.15	1.68 ^H		
Dizziness	Test 1	2.0 \pm 0.78	1.4 \pm 0.57	0.91 ^L	0.06 ^N	
	Test 2	1.7 \pm 0.63	1.3 \pm 0.43	0.76 ^L		

% Δ percentage change; ^Ttreatment; ^Ccontrol; *Effect size*: ^Nnegligible; ^Ssmall; ^Mmedium; ^Llarge; ^Hhuge

It was hypothesised that the administration of *Phosphoricum acidum 200CH* would decrease lower limb muscle pain, lower limb muscle stiffness, nausea, headache and dizziness symptom severity. From these results it is thus evident that the hypothesis is rejected.

5. Stellenbosch Mood Scale (STEMS) Questionnaire

The numerical rating scale, regarding anger, confusion, depression, fatigue, tension and vigour categories, administered following each test showed no significant time or group effect ($p > 0.05$). As illustrated in *Table 4.10* there was however a medium practical effect on anger and depression and a small practical effect on confusion and tension. All these practical effects were in favour of the control group.

Table 4.10 Stellenbosch Mood Scale ($n = 30$)

Test Results		Treatment	Control	Effect Size	
		($n=15$)	($n=15$)	Test ^T	Test ^C
		$\bar{x} \pm SD$	$\bar{x} \pm SD$	d -value	
Anger	Test 1	51 \pm 8.4	50 \pm 9.4	0.12 ^N	0.58 ^M
	Test 2	52 \pm 13.1	46 \pm 2.5	0.62 ^M	
Confusion	Test 1	53 \pm 9.1	48 \pm 8.7	0.60 ^M	0.32 ^S
	Test 2	53 \pm 13.4	46 \pm 6.9	0.66 ^M	
Depression	Test 1	56 \pm 13.1	52 \pm 13.5	0.32 ^S	0.45 ^M
	Test 2	57 \pm 21.4	46 \pm 3.8	0.70 ^M	
Fatigue	Test 1	60 \pm 9.9	56 \pm 11.7	0.40 ^M	0.11 ^N
	Test 2	60 \pm 10.7	56 \pm 11.1	0.39 ^S	
Tension	Test 1	45 \pm 6.1	41 \pm 6.7	0.67 ^M	0.32 ^S
	Test 2	44 \pm 8.4	39 \pm 3.5	0.77 ^L	
Vigour	Test 1	52 \pm 7.8	53 \pm 8.5	0.13 ^N	0.09 ^N
	Test 2	50 \pm 8.3	51 \pm 10.8	0.11 ^N	

% Δ percentage change; ^Ttreatment; ^Ccontrol; *Effect size*: ^Nnegligible; ^Ssmall; ^Mmedium; ^Llarge

It was hypothesised that the administration of *Phosphoricum acidum 200CH* would decrease negative emotions (anger, confusion, depression, fatigue, tension) and increase positive emotions (vigour) induced by cycling. From these results it is thus evident that the hypothesis is rejected.

D. CONCLUSION

The treatment with homoeopathic remedies, namely *Phosphoricum acidum 200CH* has proved to be effective in many clinical situations; however confirmation of its use within the sporting industry is limited. From the results above it is clearly apparent that the control group proved to be of a higher calibre when comparing performance variables of the two groups namely:

- Higher peak power output
- Higher vO_{2Max}
- Lower economy lactate

- Lower resting lactate

Despite the treatment group being the weaker of the two groups, they showed improvement in performance after administration of the remedy. This improvement was manifest through physiological alteration in the second test. More exclusively is the acceptance of the hypothesis concerning heart rate and oxygen consumption, whereby results showed that the administration of *Phosphoricum acidum 200CH* decreased heart rate and submaximal oxygen consumption rates during performance and recovery. There was no observable psychological effect during this study. The results suggest that *Phosphoricum acidum 200CH* primarily demonstrated physiological effects on the cyclists. The researcher believes that this is due to insufficient time given for psychological alterations.

CHAPTER FIVE: DISCUSSION

A. INTRODUCTION

The data recorded in this study was evenly distributed and parametric testing was applied to it. The analysis was aimed at investigating the effect that *Phosphoricum acidum 200CH* had on the performance and recovery of cyclists during and after repeated incremental ergometer tests.

B. DATA IN PERSPECTIVE

Results indicated that there were no statistical differences between the two groups' ($p > 0.05$) performance variables, however there were practical significant differences, which were determined using Cohen's d test, where the percentage difference between test one (base line) and test two were the major components in tracking performance and recovery changes. Each test was divided, according to time points, into four phases as illustrated in *Table 5.1*. To have a large practical effect on performance, it is important that positive effects of treatment are manifest on more than one parameter for a certain time point for there to be a true overall positive effect on performance during that phase. However if one of the systems are coping more efficiently with the stress of exercise, this will in turn lead to a reduction in overall stress, allowing the body to focus resources and energy on the systems still under stress.

Table 5.1 Effect Size At Different Time Points ($n=30$)

Phase	Description	Effect Size <i>d</i> -value	Positive Effect Group
Phase One (Resting)	Blood lactate concentration	0.11 ^N	N
Phase Two (Cycling Economy)	Blood lactate concentration	0.43 ^M	C
	Heart rate (average 10-15 min into test)	0.0 ^N	N
	Oxygen Consumption ($\dot{V}O_2$) (10-15 min into test)	0.2 ^S	T
Phase Three (maximum)	Blood lactate concentration	0.2 ^S	T
	Heart rate (maximum)	0.48 ^M	T
	Oxygen Consumption (average highest three)	0.1 ^N	N
	Peak Power Output	0.06 ^N	N
Phase Four (Recovery)	Heart rate (one minute after exhaustion)	0.66 ^M	T
	Oxygen Consumption (one min after exhaustion)	0.24 ^S	T
	Heart rate (2-3 minutes after exhaustion)	0.2 ^S	C
	Oxygen Consumption (2-3 min after exhaustion)	0.14 ^N	N

T=treatment; C=control; N=None; *Effect size*: ^Nnegligible; ^Ssmall; ^Mmedium

Table 5.1 illustrated that the treatment had a negligible effect on Phase One lactate ($d=0.11$) with a slight increase in the treatment group. Phase Two compared cycling economy changes between the two tests, lactate in the treatment group showed an increase of medium practical significance ($d=0.43$) and oxygen consumption ($\dot{V}O_2$) was largely decreased ($d=0.2$). Economy lactate changes were in favour of the control group, however oxygen consumption was in favour of the treatment group. The decreased oxygen consumption indicates better cycling economy, however higher lactate expressed a worse cycling economy. Phase Three compared maximum values between the two tests; heart rate maximum was largely decreased with a medium effect ($d=0.48$) and maximum blood lactate decreased with small practical effect, both in favour of the treatment group. Phase Four compared recovery values between the two tests. Heart rate and oxygen consumption one-minute after peak power output showed a medium effect ($d=0.66$) and small effect ($d=0.24$) respectively,

both in favour of the treatment. This lower heart rate maximum indicates a more rapid heart rate recovery. There was a further small effect ($d=0.2$) on heart rate recovery two to three minutes following peak power output in favour of the control.

C. PERFORMANCE AND RECOVERY

1. Blood Lactate Concentration

The cyclists resting blood lactate concentrations were higher in both groups than would be expected for their fitness levels (2.4 ± 0.9 mmol) with athletes normally ranging between 0.8 mmol up to two mmol, with world class endurance athletes having values closer to 0.8 mmol. There are many possible reasons for this increased resting blood lactate concentrations, emotional distress, recent ingesting of caffeine, improper endurance training, overtraining or over reaching in the week before the testing. Participants were advised to refrain from ingesting caffeine four hours before the testing to eliminate the effect on blood lactate concentrations. Further more considering the participants superior performances, improper training, over training or over reaching were not probable causes of increased levels. However many participants reported various emotional stressors before and during testing, emotions including:

- Anticipation anxiety
- Performance anxiety
- Fear of being unable to complete the test
- Fear of the new environment (e.g. different bike, breathing mask, testing machines)
- Fear of being observed
- Fear of blood lactate testing (fear of needles or seeing blood)
- Personal anxiety (e.g. stressful day)

All these emotional factors could possibly explain the increased resting blood lactate concentration. High resting lactate concentrations were evident in eight of the 30 participants, where their economy lactate levels were lower than their resting lactate levels. A further explanation for these lower lactate levels may be as a result of active recovery or decreased emotional stress (Denadai & Higino, 2004). Active recovery has proved to be beneficial in reducing blood lactate concentrations. Active recovery occurs at low intensity exercise, during which the excess blood lactate can be removed. The first 15

minutes of the test was at low intensity which allowed for active recovery and giving the participants time to relax and become accustomed to the new environment.

Blood lactate concentrations above lactate threshold, need to return below this level to enable continued performance. At levels above four mmol pH and homeostasis within the muscles can no longer be maintained (Yun-tao, 2011).

Blood lactate concentrations directly after exhaustion should be higher than eight mmol. The lowest final lactate for all participants was 9.6 mmol with a mean of (13.9 ± 2.4) . Type two muscle fibres are able to produce lactate at higher rates, long term training increases the proportion of type two muscle fibres, therefore highly trained individuals have a higher lactate level after a maximal test.

The desired effect in the second test is for lactate levels, relative to the first test, to be lower in the treatment than in the control group, showing a decreased lactate production and an enhanced rate of removal.

2. Peak Power Output (PPO)

The peak power output in the second test should be equal to, or higher than the first (baseline) test, which would demonstrate complete recovery between the two tests. A lower PPO in the second test would indicate incomplete recovery, lack of mental stamina, or the negative after effects as a result of the first (baseline) test. Negative effects may include,

- Muscle damage
- Depletion of energy stores
- Physical exhaustion
- Mental exhaustion

It was observed that both groups showed an increased PPO in the second test, which may be due to the baseline test acting as a warm-up and thus allowing true peak performance in the second test or due to psychological factors. Psychological factors include participants better preparing themselves for the second test and experience gained in the first test. It was observed that both groups showed a small practical increase in peak power output between test one and test two, however there was no

significant group interaction. Increasing peak power output often takes weeks of training, which allows time for physiological and anatomical adaptations, the time between the two tests was too short to allow for these adaptations, resulting in no observable effect of treatment. It can therefore be concluded that further long term studies need to be completed to test the long term effects of *Phosphoricum acidum 200CH* on cyclists' peak power output, muscle and physiological adaptations.

3. Oxygen Consumption

It was observed that both groups were less economical and consumed more oxygen 10 to 15 minutes into the second test compared to the first test during same time period. However, this decline in economy was experienced to a lesser degree within the treatment group with a small practical effect ($d= 0.2$). Both groups showed increased maximum oxygen consumption in the second test, this may be due to the baseline test acting as a warm-up and thus allowing true peak performance in the second test. Oxygen consumption in the treatment group was lower in the second test during the recovery period. Lower oxygen consumption per minute during performance is desirable as it indicates superior cycling economy. However, higher maximum oxygen consumption is desirable at peak exertion as this allows higher rates of aerobic energy production. *Phosphoricum acidum* is effective in treating respiratory symptoms including (Phatak, 2005):

- Difficulty breathing
- Dyspnoea
- Tachpnea
- Feeling of pressure behind the sternum
- Twisting pain behind the sternum

The treatment thus had the desired effect on the cyclists causing increased cycling economy and an enhanced rate of recovery one minute after exhaustion.

4. Heart Rate

Participants' maximum heart rates were very close to their age predicted heart rates ($98.7\% \pm 5.0$). Age predicted heart rate was calculated according to *Formula 5.1* (Brawner, Ehrman, Schairer, Cao & Keteyian, 2004). For sedentary individuals maximum heart rate is calculated according to *Formula*

5.2 (Tanaka, Monahan & Seals, 2001). Calculating maximum heart rate is beneficial to determine exercise intensity and training zones. The formula works on the assumption that, age causes a decrease in elasticity of the vascular system and skeletal muscle resulting in a lower maximum heart rate. On average, maximum heart rate decreases at a rate of one beat per year. This rate of decrease can be reduced by regular moderate intensity cardiovascular exercise, resulting in the need for a more accurate formula for calculating an athlete's maximum heart rate.

Formula 5.1 Age predicted heart rate (athlete)

$$\text{Heart rate maximum} = 210 - (\text{age} \times 0.65)$$

Formula 5.2 Age predicted heart rate (sedentary)

$$\text{Heart rate maximum} = 220 - \text{age}$$

It was observed that average heart rate over the selected time intervals were higher at all time points for both groups. However the increase was smaller in the treatment group. *Phosphoricum acidum* is effective in treating cardiovascular symptoms including:

- Palpitations
- Irregular heart rate
- Intermittent heart rate

The treatment thus had the desired effect on the cyclists causing decreased maximum heart rate and recovery heart rate one minute after exhaustion.

5. Numerical Rating Scale (NRS) Questionnaire

The desired effect of the treatment on the lower limb muscle pain, lower limb muscle stiffness, nausea, headache and dizziness is that symptom severity should be lower or equal to the first test. It was observed that nausea, headache and dizziness symptoms were mild in both groups, with means of two or lower. Lower limb pain and stiffness was experience as mild to moderate NRS rating. A possible reason for no observable treatment effect may be due to low ratings of all symptoms in the first test (baseline), which made it more difficult to observe a change. It can be suggested that alternate symptoms be selected in future studies or that the participants be asked to describe their

symptoms qualitatively. Performing the questionnaires during the exercise could yield better results as highly trained cyclists' recovered rapidly, resulting in low symptom scores in this study. It can be concluded that the treatment had no effect on these five symptoms directly after the ergometer tests. *Phosphoricum acidum* according to Phatak (2005) is effective in treating muscle weakness and cramping of the arms and hands, having little effect on the lower limb or on muscle pain. *Phosphoricum acidum* does not experience nausea but rather loss of appetite with cravings for refreshing, juicy food and drinks. *Phosphoricum acidum* is effective in treating headaches however these are as a result of eye strain or after coition, not as a result of over exertion. *Phosphoricum acidum* is effective in treating vertigo and dizziness, however this vertigo most often experienced while supine, not during activity.

Patients requiring *Phosphoricum acidum* are extremely fatigued often as a result of fluid loss. It may have been more beneficial if the tests were performed in a controlled warm humid environment which would have resulted in greater fluid loss through sweating, which was not evident in the temperature controlled environment where the testing was conducted (Boericke et al., 1922 ; Phatak, 2005).

6. Stellenbosch Mood Scale (STEMS) Questionnaire

Patients requiring *Phosphoricum acidum* answer slowly, suffer from brain fog, poor memory, confusion, hopelessness, mental fatigue and loss of vigour. The remedy has little effect on anger, depression or tension. Homoeopathic remedies are only effective in treating symptoms already present; there is little evidence of homoeopathic remedies preventative mechanisms. It was observed that there were few symptoms present after the first test, for this reason there was no observable treatment effect.

D. CONCLUSION

The *Phosphoricum acidum 200CH* has proven to be effective in enhancing cycling economy, reducing maximum heart rate and enhancing recovery to a large degree for the first minute following exhaustion. These positive effects are of great importance as the treatment group was the weaker of the two groups, resulting in the possibility of even larger results being observable in repeated studies where both groups have similar performance abilities.

CHAPTER SIX: CONCLUSION AND RECOMMENDATIONS

A. CONCLUSION

Insufficient research has been performed on the effects of *Phosphoricum acidum 200CH* on cyclists' performance and recovery rate. The results of this study showed that cyclists taking *Phosphoricum acidum 200CH* demonstrated enhanced cycling economy, reduction in maximum heart rate and enhanced recovery to a large degree for the first minute following exhaustion. The positive effect of enhanced recovery one minute after exhaustion is of immense importance to cyclists as it allows for rapid recovery following accelerations, repeated breakaways or climbing of steep inclines.

This study did, however, not demonstrate an effect on peak power output. The researcher believes that more time was needed for physiological and anatomical adaptation to demonstrate possible effects. The positive effects of this study are of great significance as the treatment intervention was only administered in a single dose and only 30 minutes was allowed for its action. The greatest shortcoming in this study was the lack of observable psychological effects, resulting in the need for more precise methods of measuring emotional responses to the treatment.

B. RECOMMENDATIONS

As illustrated in *Table 6.1*, younger athletes in this study had higher resting blood lactate concentrations than older athletes. Three of the cyclists who participated in this study had a resting blood lactate concentrations above four mmol in the first test. All three of these participants were younger than 25 years of age resulting in the need for further studies to investigate the effect of age on resting blood lactate concentrations.

Table 6.1 Resting Blood Lactate Test 1 ($n=30$)

Age Range Years	(n)	Blood Lactate			
		\bar{x}	\pm	SD	Max
<25	14	2.8	\pm	1.2	5.1
26-35	5	2.0	\pm	0.8	3.1
36-45	9	1.9	\pm	0.6	3.3
>46	2	1.7	\pm	0.4	2.0

Max=Maximum

It was noted that participants reacted differently to both tests, with some performing better in the second test and others worse. Participants commented that they would have performed better if they were allowed time to become accustomed to the equipment and the testing protocol. Participants' reactions to the two test intervals varied greatly, this could be due to a variety of reasons namely:

- Fitness level
- Skeletal muscle fibre type proportions
- Metabolic status
- Fuel availability
- Emotional status
- Individual constitution

A further method for eliminating different reactions of the participants to the treatment and testing protocol is to do a crossover study with a long enough washout period between the two tests thus making sure that the remedy has stopped working. This will allow all the participants to do the test once with treatment and once with the placebo.

Individual constitutional remedy prescription according to homoeopathic posology could yield even better results as some participants would not respond fully to *Phosphoricum acidum*. A larger sample size could make the results both practically and statistically significant. Because homoeopathic remedies have systemic effects, further testing needs to be performed to identify them. The researcher suggested testing for muscle damage and blood pH.

Participants in this study had full access to power readings, cadence, time completed in the test, as well as lactate readings. Concealing the monitor during both tests is something that could be implemented as some participants had given up before they actually reached their maximum ability because they had reached the goals they had set for themselves.

Appendix A

Randomisation Table

Randomly draw an 'X' in either the Placebo or the Treatment Group for each participant. This table will correspond to the dispensing of either *Phosphoricum acidum 200CH (Phos ac.)* (lactose powder with one drop *Phosphoricum acidum 200CH*) or an identical Placebo (lactose powder with one drop alcohol 96%). Powders will be labelled from "1" to "30". The researcher will only be given access to this table once all testing and data capturing has been completed.

Participant	Group	
	Placebo	Phos ac.
1	X	
2	X	
3		X
4	X	
5	X	
6		X
7	X	
8		X
9		X
10		X
11	X	
12		X
13	X	
14		X
15		X
16		X
17	X	
18		X
19	X	
20		X
21	X	
22		X
23		X
24		X
25	X	
26	X	
27	X	
28	X	
29	X	
30		X

Appendix B

Remedy Preparation

Aims: Trituration of phosphoric acid to the third potency

To convert trituration into liquid potency *Phosphoricum acidum*

To prepare and impregnate lactose powders with *Phosphoricum acidum 200CH*

Description of crude substance:

Phosphoric acid is a colourless, odourless liquid of syrup consistency and is miscible in water and alcohol. A solution should contain no less than 85% and no more than 90% w/w of H_3PO_4 . Phosphoric acid is made by combining, calcium phosphate (which occurs naturally as Apatite) with sulphuric acid, which is then dissolved in alcohol to form the mother tincture (Benyunes, 2005: 67).

Succussion

Each succussion should be equivalent to the force exerted by an average healthy man when striking a hand-held phial forcefully against a firm surface (Benyunes, 2005).

Apparatus:

Labels, Elastic bands

1X 25ml; 196 X 5ml amber glass bottle

200 X glass dropper pipettes

Rubber/neoprene dropper units “teats”

30 X Pre-folded Lactose powders

Mass balance with spatula

Mortar and pester

Alcohol (60% 96%), Distilled water

Method: according to Benyunes (2005) method 6, 7 and 8.

1. Clean all apparatus
2. Weight out 0.1g of crude substance (phosphoric acid) or previous potency
3. Weigh out 9.9 g of lactose powder divide into three equal parts (3.3 g each)
4. Combine the 0.1g crude substance and 3.3g lactose in the mortar
5. Process: 3 stages per potency
 - 6 minutes trituration, 3 minutes scraping, 1 minute mixing
 - Repeat twice before adding another 3.3g lactose powder
 - Repeat for each of the three parts
6. Repeat the procedure for 2CH and 3CH
7. Label

Phosphoricum acidum (Potency)

Manufacture date, expiry date

Manufacturer name and location

Batch number

8. Weigh out 0.1g *Phosphoricum acidum* 3CH and add to 25ml Amber glass bottle
9. Add 5ml distilled water and allow to dissolve
10. Add an additional 5ml alcohol (60%), resulting in a 30% alcohol concentration
11. Cap, seal and succuss 100X, resulting in a 4CH potency.
12. Measure 1/100 of 3ml = 0.3ml (4CH)

99/100 of 3ml = 2.7ml alcohol (96%)

Cap, seal and succuss 100X, resulting in 5CH.

13. Repeat the above procedure until the 200CH potency level.
14. Unfold 15 lactose paper powders, place one drop of *Phosphoricum acidum* 200CH on each one. Each pre-folded paper contains 500mg lactose powder.
15. Fold each paper closed, place an elastic band around each, insert into an envelope and label each envelope.

Phosphoricum acidum 200CH or Placebo (1 powder)

Participant number [number powder according to randomization table (Appendix A)]

Dispense according to the randomization table

Dissolve powder under your tongue after completing first cycling interval

Manufacture date, expiry date

Manufacturer name and location

Batch number

16. Place one drop of 96% alcohol on each of the other 15 powders

17. Fold each paper closed, place an elastic band around each, insert into an envelope and label each envelope.

Phosphoricum acidum 200CH or Placebo (1 powder)

Participant number [number powder according to randomization table (Appendix A)]

Dispense according to the randomization table

Dissolve powder under your tongue after completing first cycling interval

Manufacture date, expiry date

Manufacturer name and location

Batch number

Appendix C

Information Letter

Title of the Research Study: The effect of *Phosphoricum acidum 200CH* on the adverse physiological effects induced by exercise in cyclists.

Principle Investigator/s: Giovanni Pantalone

Co-Investigator/s: Izel Botha, Elmarie Terblanche

Brief Introduction and Purpose of the Study: You have volunteered to participate in a study to investigate the effects of a homoeopathic remedy on the human body during exercise.

All participants will be divided into two groups; half will receive the active substance and half the placebo. You will be required to carry out two cycling intervals on a stationary trainer in the testing laboratory at Stellenbosch University. A 30 minute recovery will be given between the two intervals. You will be tested individually and will have to remain seated during the test. A heart rate monitor will be attached to your chest and an air flow mask over your mouth and nose to check your breathing.

Outline of the Procedures:

BEFORE THE TEST

You would be required to, for 24 hours before the test, not do any hard exercise, and not to drink any coffee or alcohol for at least four hours before the test. Eating and drinking should stop three hours before the test however drinking water is permitted. You are also required not to be on any heart or blood pressure medication and you need to be in a healthy state to exercise.

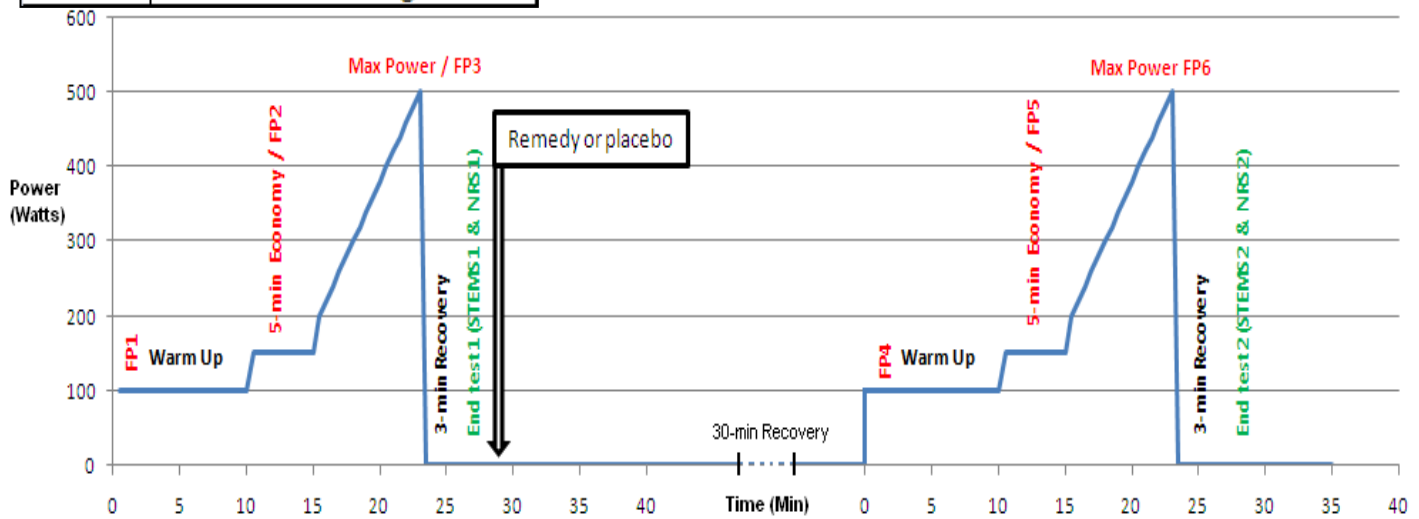
DURING THE TEST

A finger blood prick sample will be taken before you start (one drop of blood is required). The cycling interval will consist of a ten minute warm-up at a 100 Watts (feels like riding on a level road), following a five minute constant load at 150 Watts (feels like a slight up hill). A second finger blood prick sample will then be taken. Then a final interval of 200 Watts will be conducted, increasing the Wattage by 20 every 30 seconds until you can't peddle between 80 and 100 rpm (feels like a moderate hill that gets steeper progressively, this interval will last for about seven minutes) A third finger prick blood sample will then be taken. Heart Rate will be sampled for three minutes after the test. You will be asked to do five squats and answer a questionnaire. This concludes the first test.

One powder of the Homoeopathic remedy or placebo will be given to you to take dissolved under your tongue (the powder is pleasant tasting, similar to icing sugar). You will be allowed a 30 minute recovery period before the test is repeated.

DIAGRAM OF TESTS

Key	
FP	Finger Prick (lactate test)
STEMS	STEMS Questionnaire
NRS	Numerical Rating Scale



Risks or Discomforts: High levels of exercise can cause symptoms including; light-headedness, dizziness, fainting, chest, jaw, neck or back pain or pressure, severe shortness of breath, wheezing, coughing or difficulty breathing, nausea, cramps or severe pain or muscle ache and severe prolonged fatigue or exhaustion, since this is a maximal test to exhaustion.

Benefits: You will receive a free performance test to determine your vO_{2Max} , maximum power output and cycling efficiency. Your effort can add to sport and medical science in finding safe ways to increase performance and recovery, the results of the study would be made available to you.

Reason/s why the Subject May Be Withdrawn from the Study: If you are unable to complete the tests or you choose to withdraw from the study.

Costs of the Study: There is no cost to you, the participant.

Confidentiality: You will be assigned a reference number and will not be identified personally.

Research-related Injury: You may experience some discomfort, dizziness or faint spells during the finger pricks for lactate. A few participants may experience local tenderness of fingertips for one or two days after the blood sample are taken. Side effects from the medication may include fatigue, mental dullness, headache, loss of appetite and fever, lasting no more than 12 hours.

Persons to Contact in the Event of Any Problems or Queries:

Researcher: Giovanni 03pantag@webmail.co.za
 Supervisor: Dr Izel Botha Izelb@dut.ac.za
 Co-supervisor: Prof Elmarie Terblanche et2@sun.ac.za

Appendix D

Informed Consent

Statement of Agreement to Participate in the Research Study:

(I,.....subject's full name,
ID number....., have read this document in its entirety and understand its contents. Where I have had any questions or queries, these have been explained to me by **Giovanni Pantalone** to my satisfaction. Furthermore, I fully understand that I may withdraw from this study at any stage without any adverse consequences and my future health care will not be compromised. I, therefore, voluntarily agree to participate in this study.

Subject's name (print)

Subject's signature:.....

Date:.....

Researcher's name (print): Giovanni Pantalone

Researcher's signature:.....

Date:.....

Witness name (print):

Witness signature:

Date:.....

Supervisor's name (print):.....

Supervisor's signature:

Date:.....

Appendix E

Participant Details And Vitals

Report						
Date of Birth						
Full Name						
Age						
Height (cm)				Weight (kg)		
BMI (kg/m ²) >16.5 ; <35						
Blood Pressure >90/50 <140/110				Resting Heart Rate (BPM) >35 ; <100		
Temperature >34 ; <37.5				Breathing Rate (BPM) >10 ; <20		
Any medical conditions, or on any medication:						
First Test (Base line)				Second Test		
Lactate Testing						
	Lactate	HR	VO	Lactate	HR	VO
Before						
15:00						
Final						
Final + 1 min						
Final + 3min						
Power Testing (Watts)						
Completed Work Load						
Seconds In Final						
Peak Power Output						

Appendix F

Numerical Rating Scale Questionnaire

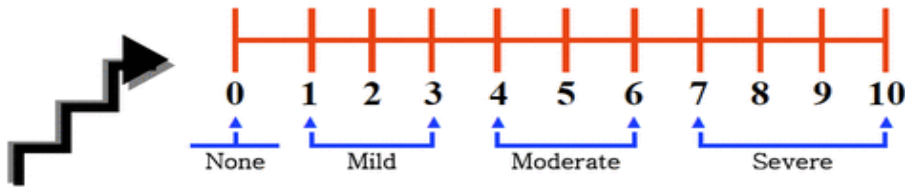
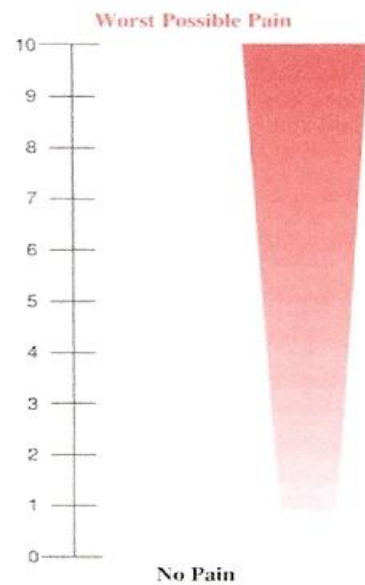
NB complete five air squats before completing this questionnaire

Rate each of the following symptoms from zero to ten (11 point).

Zero being none and Ten being the worst discomfort ever.

Write down the number of how you feeling right now.

- A. Lower Limb Muscle Pain [/10]
- B. Lower Limb Muscle Stiffness [/10]
- C. Nausea [/10]
- D. Headache [/10]
- E. Dizziness [/10]



Appendix G

Stellenbosch Mood Scale (STEMS) Questionnaire

Below is a list of words that describe feelings people have. Read each one carefully. Then circle the answer that describes **how you feel right now**.

Hieronder is 'n lys van woorde wat die gevoelens van mense beskryf. Lees elkeen noukeurig. Omsirkel daarna die antwoord wat die beste beskryf hoe jy op hierdie oomblik voel.

	<i>Not at all Glad nie</i>	<i>A little Effens</i>	<i>Moderately Taamlik</i>	<i>Quite a bit Baie</i>	<i>Extremely Uiters</i>	
Panicky	0	1	2	3	4	Paniekerig
Lively	0	1	2	3	4	Lewendig
Confused	0	1	2	3	4	Verward
Worn out	0	1	2	3	4	Vermoeid
Depressed	0	1	2	3	4	Neerslagtig
Competent	0	1	2	3	4	Bekwaam
Downhearted	0	1	2	3	4	Mismoedig
Annoyed	0	1	2	3	4	Vererg
Exhausted	0	1	2	3	4	Uitgeput
Mixed-up	0	1	2	3	4	Deurmekaar
Sleepy	0	1	2	3	4	Vaak
Bitter	0	1	2	3	4	Verbitterd
Proud	0	1	2	3	4	Trots
Unhappy	0	1	2	3	4	Ongelukkig
Anxious	0	1	2	3	4	Angstig
Worried	0	1	2	3	4	Bekommerd
Energetic	0	1	2	3	4	Energiek
Miserable	0	1	2	3	4	Ellendig
Muddled	0	1	2	3	4	Ontwrig
Nervous	0	1	2	3	4	Senuweeagtig
Confident	0	1	2	3	4	Vol vertroue
Angry	0	1	2	3	4	Kwaad
Active	0	1	2	3	4	Aktief
Tired	0	1	2	3	4	Moeg
Bad tempered	0	1	2	3	4	Humeurig
Alert	0	1	2	3	4	Op en wakker
Satisfied	0	1	2	3	4	Tevrede
Uncertain	0	1	2	3	4	Onseker



Are you a cyclist interested in



your performance???

- ✓ Get your **cycling economy, peak power output and VO_{2Max}** tested for **Free!**
- ✓ Be part of a **Masters research!**
- ✓ All participants should be **Male**, Aged **18–55yrs**, train at least 2 hours per week and not on Blood pressure/heart medication.
- ✓ **High Fitness** level required! (sub 3:30 for 100km or MTB equivalent)
- ✓ Test done at **Stellenbosch University Sports Physiology lab**
- ✓ Test will only take **1.5hours (once off)**

For more information contact: **Giovanni** (Limited space available)

03pantag@webmail.co.za

REFERENCES

Acheson, K, Jéquier, E, Burger, A & Danforth Jr, E. 1984. Thyroid hormones and thermogenesis: The metabolic cost of food and exercise. *Metabolism*, 33 (3): 262-265.

Alla, S, Sullivan, SJ, McCrory, P, Schneiders, AG & Handcock, P. 2008. Does exercise evoke neurological symptoms in healthy subjects?, *Journal of Science and Medicine in Sport*, 13 (1): 24-26.

Atchley Jr, AE & Douglas, PS. 2007. Left Ventricular Hypertrophy in Athletes: Morphologic Features and Clinical Correlates. *Cardiology Clinics*, 25 (3): 371-82.

Baatz, W. 2005. *The Brains behind RacerMate* [online]. Available WWW: racermateinc.com (30 November 2010).

Baldwin, KM. & Haddad, F. 2001. Effects of different activity and inactivity paradigms on myosin heavy chain gene expression in striated muscle, *Journal of Applied Physiology*, 90 (1): 345–357.

Barron, JL. Noakes, TD. Levy, W. Smith, C & Millar, RP. 1985. Hypothalamic dysfunction in over trained athletes. *Endocrinology and Metabolism*, 60: 803-806.

Bassett, DR & Howley, ET. 2000. Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Medicine & Science in Sports & Exercise*, 32 (1): 70.

Bell, IR, Baldwin, CM, Schwartz, GER & Russek, LGS. 1999. Integrating Belief Systems and Therapies in Medicine: Application of the Eight World Hypotheses to Classical Homeopathy. *Integrative Medicine*, 1 (3): 95-105.

Bellavite, P, Lussignoli, S, Semizzi, ML, Ortolani, R & Signorini, A. 1997. The similia principle: From cellular models to regulation of homeostasis. *British Homoeopathic journal*, 86 (2): 73-85.

Bentley, DJ & McNaughton, LR. 2003. Comparison of Wpeak, VO₂peak and the ventilation threshold from two different incremental exercise tests: Relationship to endurance performance. *Journal of Science and Medicine in Sport*, 6 (4): 422-435.

Benyunes, S. 2005. *German Homoeopathic Pharmacopoeia*. First Supplement ed. Stuttgart: medpharm GmbH Scientific Publishers.

Bevilacqua, M. Savonitto, S. Bosisio, E. Chebat, E. Bertora, PL. Sardina, M. & Norbiato, G. 1989. Role of the Frank-Starling mechanism in maintaining cardiac output during increasing levels of treadmill exercise in beta-blocked normal men. *The American Journal of Cardiology*, 63 (12): 853-7.

Bickley, L.S., Szilagyi, P.G., Bates, B. & Prabhu, F.R. 2007, *Bates' guide to physical examination and history taking* Lynn S. Bickley, Peter G. Szilagyi, 9th edn, Lippincott Williams & Wilkins, Philadelphia.

Birzniece, V, Nelson, AE & Ho, KKY. 2010. Growth Hormone Administration: Is It Safe and Effective for Athletic Performance. *Endocrinology & Metabolism Clinics of North America*, 39 (1): 11-23.

Boericke, W. Boericke, OE. & Griffin, HR. 1922 *Pocket Manual of Homoeopathic Materia Medica: Comprising the Characteristic and Guiding Symptoms of all Remedies (Clinical and Pathogenetic)*, 8th, rev a enl, with the addition of a repertory by Oscar E Boericke -- ed. New York: Boericke & Runyon, pp. 1128.

Bompa, T.O. & Bompa, T.O. 1999, *Periodization : theory and methodology of training*, 4th edn, Human Kinetics, Champaign, IL.

Brawner, CA, Ehrman, JK, Schairer, JR, Cao, JJ & Keteyian, SJ. 2004. Predicting maximum heart rate among patients with coronary heart disease receiving [beta]-adrenergic blockade therapy. *American Heart Journal*, 148 (5): 910-914.

Brooks, GA. 1998. Mammalian fuel utilization during sustained exercise. *Comparative Biochemistry and Physiology Part B: Biochemistry and Molecular Biology*, 120 (1): 89-107.

Brun, JF, Connes, P. & Varlet-Marie, E. 2007. Alterations of blood rheology during and after exercise are both consequences and modifiers of body's adaptation to muscular activity. *Science & Sports*, 22 (6): 251-66.

Bruton, A. 2002. Muscle Plasticity: Response to training and detraining. *Physiotherapy*, 88 (7): 398-408.

Buckley, JD, Thomson, RL, Coates, AM, Howe, PRC, DeNichilo, MO & Rowney, MK. 2001. Supplementation with a whey protein hydrolysate enhances recovery of muscle force-generating capacity following eccentric exercise. *Journal of Science and Medicine in Sport*, 13 (1): 178-81.

Callaghan, MJ. 2005. Lower body problems and injury in cycling. *Journal of Bodywork and Movement Therapies*, 9 (3): 226-236.

Caputo, F & Denadai, BS. 2009. Does 75% of the difference between vO_2 at lactate threshold and vO_2 max lie at the severe-intensity domain in well-trained cyclists? *Science & Sports*, 24 (5): 257-61.

Caravati, EM. 1987. Metabolic abnormalities associated with phosphoric acid ingestion. *Annals of Emergency Medicine*, 16 (8): 904-906.

Chapman, AR, Vicenzino, B, Blanch, P & Hodges, PW. 2008. Patterns of leg muscle recruitment vary between novice and highly trained cyclists. *Journal of Electromyography and Kinesiology*, 18 (3): 359-71.

Close, GL, Ashton, T, McArdle, A & MacLaren, DPM. 2005. The emerging role of free radicals in delayed onset muscle soreness and contraction-induced muscle injury. *Comparative Biochemistry and Physiology - Part A: Molecular & Integrative Physiology*, 142 (3): 257-266.

Cochrane, DJ. 2004. Alternating hot and cold water immersion for athlete recovery: a review. *Physical Therapy in Sport*, 5 (1): 26-32.

Correia, LCL, Lakatta, EG, O'Connor, FC, Becker, LC, Clulow, J, Townsend, S, Gerstenblith, G & Fleg, JL. 2002. Attenuated cardiovascular reserve during prolonged submaximal cycle exercise in healthy older subjects. *Journal of the American College of Cardiology*, 40 (7): 1290-1297.

Covinsky, KE, Wu, AW, Landefeld, CS, Connors, AF, Jr, Phillips, RS, Tsevat, J, Dawson, NV, Lynn, J & Fortinsky, RH. 1999. Health status versus quality of life in older patients: does the distinction matter? *The American Journal of Medicine*, 106 (4): 435-440.

Cowperthwaite, AC. 1927, A text-book of materia medica and therapeutics : characteristic, analytical, and comparative, 13th edn, Boericke & Tafel, Philadelphia.

Denadai, BS & Higino, WP. 2004. Effect of the passive recovery period on the lactate minimum speed in sprinters and endurance runners. *Journal of Science and Medicine in Sport*, 7 (4): 488-496.

Dunn, M, Thomas, JO, Swift, W, Burns, L & Mattick, RP. 2010. Drug testing in sport: The attitudes and experiences of elite athletes. *International Journal of Drug Policy*, 21 (4): 330-332.

El-Asmy, AA, Serag, HM, Mahdy, MA & Amin, MI. 2008. Purification of phosphoric acid by minimizing iron, copper, cadmium and fluoride. *Separation and Purification Technology*, 61 (3): 287-292.

El-Sayed, MS, MacLaren, D & Rattu, AJ. 1997. Exogenous carbohydrate utilisation: Effects on metabolism and exercise performance. *Comparative Biochemistry and Physiology Part A: Physiology*, 118 (3): 789-803.

Farrar, JT, Troxel, AB, Stott, C, Duncombe, P. & Jensen, MP. 2008. Validity, reliability, and clinical importance of change in a 0--10 numeric rating scale measure of spasticity: a post hoc analysis of a randomized, double-blind, placebo-controlled trial. *Clinical Therapeutics*, 30 (5): 974-85.

Fleg, JL. & Lakatta, EG. 1984. Prevalence and prognosis of exercise-induced nonsustained ventricular tachycardia in apparently healthy volunteers. *The American Journal of Cardiology*, 54 (7): 762-4.

- Fradkin, AJ. Gabbe, BJ. & Cameron, PA. 2006. Does warming up prevent injury in sport?: The evidence from randomised controlled trials?, *Journal of Science and Medicine in Sport*, 9 (3): 214-20.
- Gardner, FL & Moore, ZE. 2004. A mindfulness-acceptance-commitment-based approach to athletic performance enhancement: Theoretical considerations. *Behavior Therapy*, 35 (4): 707-723.
- Giada, F, Bertaglia, E, De Piccoli, B, Franceschi, M, Sartori, F, Raviele, A & Pascotto, P. 1998. Cardiovascular adaptations to endurance training and detraining in young and older athletes. *International Journal of Cardiology*, 65 (2): 149-155.
- Gonzalez, H & Hull, ML. 1989. Multivariable optimization of cycling biomechanics. *Journal of Biomechanics*, 22 (11-12): 1151-1161.
- Gotzsche, PC. 1994. Is there logic in the placebo? *The Lancet*, 344 (8927): 925-926.
- Gutman, W. 1970. Preventive treatment of influenza and influenzosis. *British Homoeopathic journal*, 59 (4): 211-3
- Guyton, AC & Hall, JE. 1996. Sports Physiology. Human Physiology and Mechanisms of Disease. 6th ed. United States of America: Saunders. 687
- Hacker, CF. 1948. Vital Force and homoeopathy. *British Homoeopathic journal*, 38 (1): 32-45.
- Heyward, V. 1998. Assessing cardiorespiratory fitness. *In Advance fitness assessment and exercise prescription*. In S. Wikgren & E. Mustain 3rd edn. 48.
- Hug, M, Mullis, PE, Vogt, M, Ventura, N & Hoppeler, H. 2003. Training modalities: over-reaching and over-training in athletes, including a study of the role of hormones. *Best Practice & Research Clinical Endocrinology & Metabolism*, 17 (2): 191-209.
- Inch, K. 2001. Homoeopathy for Sports, Exercise and Dance. *Physiotherapy*, 87 (7): 386-386.

Jammes, Y. Steinberg, JG. Brégeon, F. & Delliaux, S. 2004. The oxidative stress in response to routine incremental cycling exercise in healthy sedentary subjects, *Respiratory Physiology & Neurobiology*, 144 (1): 81-90.

Jeukendrup, AE. 2004. Carbohydrate intake during exercise and performance. *Nutrition*, 20 (7-8): 669-77.

Jeukendrup, AE, Craig, NP & Hawley, JA. 2000. The bioenergetics of world class cycling. *Journal of Science and Medicine in Sport*, 3 (4): 414-33.

Kaul, PN. 1996. Alternative therapeutic modalities. *Alternative medicine: Prog Drug Res* (47): 251–277.

Kawabata, T, Suzuki, T & Miyagawa, T. 2004. Effect of blood volume on plasma volume shift during exercise. *Journal of Thermal Biology*, 29 (7-8): 775-778.

Kayne, S. 1992. Homoeopathy in sports medicine. *British Homoeopathic journal*, 81 (3): 142-147.

Kelley, G. 1996. Mechanical overload and skeletal muscle fibre hyperplasia: A meta-analysis, *Journal of Applied Physiology*, 81: 1584–1588

Kidd, M. 2010. Pantalone, G. Stellenbosch at the centre for statistical consultation, 11 February

Kienle, GS & Kiene, H. 1997. The Powerful Placebo Effect: Fact or Fiction? *Journal of Clinical Epidemiology*, 50 (12): 1311-1318.

Kisner, C. & Colby, LA. 1990. *Therapeutic Exercise: Foundations and techniques*, F A Davis, Philadelphia.

Komi, PV. & Hakkinen, K. 1991. Strength and power, *The Olympic Book of Sports Medicine*, Blackwell Scientific Publications, Oxford :181–193.

- Kronfeld, DS. 2001. Body fluids and exercise: Physiological responses (part I). *Journal of Equine Veterinary Science*, 21 (7): 312-22.
- La Grange, CD. 1999. *A Study of the Efficacy of Homeopathic Treatment in Controlling Lactic Acid Accumulation and Exercise Fatigue*. Dissertation (M.Tech) Technicon Natal.
- Lane, A. 2001. Relationships between perceptions of performance expectations and mood among distance runners: The moderating effect of depressed mood. *Journal of Science and Medicine in Sport*, 4 (1): 116-28.
- Laplaud, D, Talmud, J & Menier, R. 2005. Do graded exercise tests conceal accurate aerobic endurance and physiological overwork markers? *Science & Sports*, 20 (3): 147-149.
- Ledermann, EK. 1969. The vital force in Homoeopathy and in general medical science. *British Homoeopathic journal*, 58 (3): 148-60.
- Lenti, M. De Vito, G. Sbriccoli, P. Scotto di Palumbo, A. & Sacchetti, M. 2010. Muscle fibre conduction velocity and cardiorespiratory response during incremental cycling exercise in young and older individuals with different training status, *Journal of Electromyography and Kinesiology*, 20 (4): 566-571.
- Lovering, AT, Haverkamp, HC & Eldridge, MW. 2005. Responses and Limitations of the Respiratory System to Exercise. *Clinics in Chest Medicine*, 26 (3): 439-457.
- Lukaski, HC. 2004. Vitamin and mineral status: effects on physical performance. *Nutrition*, 20 (7-8): 632-644.
- MacDougall, JD. 1986. Morphological changes in human skeletal muscle following strength training and immobilisation. *Human Muscle Power* : 270–285
- Machado-Moreira, CA, de Castro Magalhães, F, Vimieiro-Gomes, AC, Lima, NRV & Rodrigues, LOC. 2005. Effects of heat acclimation on sweating during graded exercise until exhaustion. *Journal of Thermal Biology*, 30 (6): 437-442.

Margonis, K, Fatouros, IG, Jamurtas, AZ, Nikolaidis, MG, Douroudos, I, Chatzinikolaou, A, Mitrakou, A, Mastorakos, G, Papassotiriou, I, Taxildaris, K & Kouretas, D. 2007. Oxidative stress biomarkers responses to physical overtraining: Implications for diagnosis. *Free Radical Biology and Medicine*, 43 (6): 901-910.

Mary, DASG. 1984. Exercise Physiology: Energy, Nutrition, and Human Performance: Editors: W.D. McArdle, F.I. Katch and V.I. Katch Lea & Febiger, Philadelphia; 1981; 518 pp.; \$25.25. *International Journal of Cardiology*, 5 (4): 559-559.

Mathie, RT. 2003. The research evidence base for homeopathy: a fresh assessment of the literature. *Homeopathy*, 92 (2): 84-91.

Maurer, MM. Burkhoff, D. Maybaum, S. Franco, V. Vittorio, TJ. Williams, P. White, L. Kamalakkannan, G. Myers, J. & Mancini, DM. 2009. A Multicenter Study of Noninvasive Cardiac Output by Bioreactance During Symptom-limited Exercise. *Journal of Cardiac Failure*, 15 (8): 689-99.

McArdle, W, Katch, FI, Katch, VL. 2001. *Exercise Physiology Energy, Nutrition and Human Performance*. United States of America: Lippincott Williams & Wilkins.

McKenna, MJ. 1995. Effects of training on potassium homeostasis during exercise. *Journal of Molecular and Cellular Cardiology*, 27 (4): 941-949.

McMahon, S & Wenger, HA. 1998. The Relationship Between Aerobic Fitness and Both Power Output and Subsequent Recovery During Maximal intermittent Exercise. *Journal of Science and Medicine in Sport*, 1 (4): 219-27.

McNair, DM. Lorr, M. and Droppleman, L.F., 1992. *Revised manual for the Profile of Mood States*, Educational and Industrial Testing Services, San Diego, CA.

Miki, T, Yokota, Y, Seo, T & Yokoyama, M. 1994. Echocardiographic findings in 104 professional cyclists with follow-up study. *American Heart Journal*, 127 (4, Part 1): 898-905.

- Milgrom, LR. 2007. Conspicuous by its absence: the Memory of Water, macro-entanglement, and the possibility of homeopathy. *Homeopathy*, 96 (3): 209-219.
- Morgan, WP. & Goldston, SE. (eds) 1987. Exercise and mental health, Washington, DC:Hemisphere.
- Morgan, WP. & O'Connor, PJ. 1988. Exercise adherence: Its impact on public health, *Exercise and mental health*, :91-121.
- Morris, CK, Myers, J, Froelicher, VF, Kawaguchi, T, Ueshima, K & Hideg, A. 1993. Nomogram based on metabolic equivalents and age for assessing aerobic exercise capacity in men. *Journal of the American College of Cardiology*, 22 (1): 175-182.
- Morton, AR. 2008. Exercise Physiology. In MT Lynn, Md, IL Louis, Ao, D Litt & Fracp (eds). *Pediatric Respiratory Medicine (Second Edition)*. Philadelphia: Mosby. 89-99.
- Moudon, AV, Lee, C, Cheadle, AD, Collier, CW, Johnson, D, Schmid, TL & Weather, RD. 2005. Cycling and the built environment, a US perspective. *Transportation Research Part D: Transport and Environment*, 10 (3): 245-61.
- Nielsen, B. Hales, B. Strange, JRS. Christensen, S. Warberg, NJ & Saltin, B. 1993. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment, *J. Physiol.* 460: 467-485
- O'Donovan, JB. 1986. Homoeopathy and holism. *British Homoeopathic journal*, 75 (2): 91-95.
- Owen, JM. 2003. Homeopathy for sports, exercise and dance. *Journal of Manipulative and Physiological Therapeutics*, 26 (3): 212.
- Parfitt, G & Gledhill, C. 2004. The effect of choice of exercise mode on psychological responses. *Psychology of Sport and Exercise*, 5 (2): 111-117.
- Peters, D. 2001. *Understanding the Placebo Effect in Complementary medicine: Theory, Practice and Research* London: Churchill Livingstone.

Phatak, S. 2005. *Materia Medica Of Homoeopathic Medicines*. Second Edition Revised And Enlarged ed. New Delhi: B. Jain Publishers (P) Ltd.

Pisani, DF, Leclerc, L, Jarretou, G, Marini, J-F & Dechesne, CA. 2005. SMHS1 is involved in oxidative/glycolytic-energy metabolism balance of muscle fibers. *Biochemical and Biophysical Research Communications*, 326 (4): 788-793.

Plezbert, JA. & Burke, JR. 2005. Effects of the homeopathic remedy *Arnica* on attenuating symptoms of exercise-induced muscle soreness. *Journal of Chiropractic Medicine*, 4 (3): 152-61.

Potma, EJ, van Graas, IA & Stienen, GJ. 1995. Influence of inorganic phosphate and pH on ATP utilization in fast and slow skeletal muscle fibers. *Biophysical Journal*, 69 (6): 2580-2589.

Powers, SK & Howley, ET. 2007. *Exercise physiology : theory and application to fitness and performance*. 6th ed. Boston: McGraw-Hill.

Price, DD, Patel, R, Robinson, ME & Staud, R. 2008. Characteristics of electronic visual analogue and numerical scales for ratings of experimental pain in healthy subjects and fibromyalgia patients. *Pain*, 140 (1): 158-166.

Ryan, MM & Gregor, RJ. 1992. EMG profiles of lower extremity muscles during cycling at constant workload and cadence. *Journal of Electromyography and Kinesiology*, 2 (2): 69-80.

Sabapathy, S, Morris, NR & Schneider, DA. 2006. Ventilatory and gas-exchange responses to incremental exercise performed with reduced muscle glycogen content. *Journal of Science and Medicine in Sport*, 9 (3): 267-273.

Saine, A. 1994. Hering revisited. *British Homoeopathic journal*, 83 (4): 231.

Salazar Vázquez, BY, Wettstein, R, Cabrales, P, Tsai, AG & Intaglietta, M. 2008. Microvascular experimental evidence on the relative significance of restoring oxygen carrying capacity vs. blood viscosity in shock resuscitation. *Biochimica et Biophysica Acta (BBA) - Proteins & Proteomics*, 1784 (10): 1421-1427.

- Sale, DG. 1986. Neural adaptations in strength and power training, *Human Muscle Power*, Human Kinetics Publishers, Ontario pp. 289–305.
- Saravay, SM, Pollack, S, Steinberg, MD, Weinschel, B & Habert, M. 1996. Four-year follow-up of the influence of psychological comorbidity on medical rehospitalization. *Am J Psychiatry*, 153: 397-403.
- Schiaffino, S & Serrano, A. 2002. Calcineurin signaling and neural control of skeletal muscle fiber type and size. *Trends in Pharmacological Sciences*, 23 (12): 569-575.
- Scott, W. Stevens, J. & Binder-Macleod, SA. 2001. Human skeletal muscle fibre type classifications, *Physical Therapy*, 81 (1): 1810–1816.
- Shapiro, AK, Mike, V, Barten, H & Shapiro, E. 1973. Study of the placebo effect with a self-administered placebo test. *Comprehensive Psychiatry*, 14 (6): 535-548.
- Shave, R & Franco, A. 2006. The physiology of endurance training. In W Gregory & BSPMP FACSM (eds). *The Physiology of Training*. Edinburgh: Churchill Livingstone. 61-84.
- Siciliano, G. Laura Manca, M. Renna, M. Prontera, C. Mercuri, A. & Murri, L. 2000. Effects of aerobic training on lactate and catecholaminergic exercise responses in mitochondrial myopathies, *Neuromuscular Disorders*, 10 (1): 40-5.
- Smallridge, RC, Whorton, NE, Burman, KD & Ferguson, EW. 1985. Effects of exercise and physical fitness on the pituitary-thyroid axis and on prolactin secretion in male runners. *Metabolism*, 34 (10): 949-954.
- So, RCH, Ng, JKF & Ng, GYF. 2005. Muscle recruitment pattern in cycling: a review. *Physical Therapy in Sport*, 6 (2): 89-96.
- Speed, HD & Andersen, MB. 2000. What exercise and sport scientists don't understand. *Journal of Science and Medicine in Sport*, 3 (1): 84-92.

- Sullivan, MJ, Cobb, FR. & Higginbotham, MB. 1991. Stroke volume increases by similar mechanisms during upright exercise in normal men and women. *The American Journal of Cardiology*, 67 (16): 1405-12.
- Sverdlova, NS & Witzel, U. 2010. Principles of determination and verification of muscle forces in the human musculoskeletal system: Muscle forces to minimise bending stress. *Journal of Biomechanics*, 43 (3): 387-396.
- Talmadge, RJ. 2000. Myosin heavy chain isoform expression following reduced neuromuscular activity: Potential regulatory mechanisms, *Muscle and Nerve*, 23 : 661–679.
- Tanaka, H, Monahan, KD & Seals, DR. 2001. Age-predicted maximal heart rate revisited. *Journal of the American College of Cardiology*, 37 (1): 153-156.
- Taylor, PJ, van Rosendal, SP, Coombes, JS, Gordon, RD & Stowasser, M. 2010. Simultaneous measurement of aldosterone and cortisol by high-performance liquid chromatography-tandem mass spectrometry: Application to dehydration-rehydration studies. *Journal of Chromatography B*, 878 (15-16): 1195-1198.
- Teixeira, MZ, Guedes, CHFF, Barreto, PV & Martins, MA. 2010. The placebo effect and homeopathy. *Homeopathy*, 99 (2): 119-29.
- Terry, PC, Lane, AM. & Fogarty, GJ. 2003. Construct validity of the Profile of Mood States -- Adolescents for use with adults. *Psychology of Sport and Exercise*, 4 (2): 125-39.
- Thevis, M, Sigmund, G, Geyer, H & Schänzer, W. 2010. Stimulants and Doping in Sport. *Endocrinology & Metabolism Clinics of North America*, 39 (1): 89-105.
- Tonkonogi, M, Walsh, B, Svensson M. & Sahlin, K. 2000. Mitochondrial function and antioxidative defence in human muscle: Effects of endurance training and oxidative stress, *Journal of Physiology* 528: 379–388.

Tucker, R. Bester, A. Lambert, EV. Noakes, TD. Vaughan, CL. & St Clair Gibson, A. 2006. Non - random fluctuations in power output during self - paced exercise, *British Journal of Sports Medicine*, 40 (11): 912–917

Tveiten, D. & Brusset, S. 2003. Effect of *Arnica* D30 in marathon runners. Pooled results from two double-blind placebo controlled studies. *Homeopathy*. 92 (4): 187-189.

Unutzer, J, Patrick, DL, Simon, G, Grembowski, D, Walker, E, Rutter, C & Katon, W. 1997. Depressive symptoms and the cost of health services in HMO patients aged 65 years and older: A 4-year prospective study. *JAMA*, 277: 1618-1623.

Valberg, SJ. 2008. Skeletal Muscle Function. In *Clinical Biochemistry of Domestic Animals (Sixth Edition)*. San Diego: Academic Press. 459-484.

Van Sickle Jr, JR & Hull, ML. 2007. Is economy of competitive cyclists affected by the anterior-posterior foot position on the pedal? *Journal of Biomechanics*, 40 (6): 1262-1267.

Vickers, AJ. Fisher, P. Smith, C. Wyllie, SE. & Lewith, GT. 1997. Homoeopathy for delayed onset muscle soreness: a randomised double blind placebo controlled trial, *British Journal of Sports Medicine*, 31 (4): 304–307.

Vithoukas, G. 1980. *The Science of Homeopathy*. New York: Grove Weiden field.

Vithoukas, G. 1985. *Homoeopathy : medicine of the new man*, Thorsons, Wellingborough.

von Korff, M, Ormel, J, Katon, W & Lin, EH. 1992. Disability and depression among high utilizers of health care. *Arch Gen Psychiatry*, 49: 91-100.

Wadley, G & Le Rossignol, P. 1998. The relationship between repeated sprint ability and the aerobic and anaerobic energy systems. *Journal of Science and Medicine in Sport*, 1 (2): 100-110.

Walach, H. 1997. The pillar of homoeopathy: Homoeopathic drug provings in a scientific framework. *British Homoeopathic journal*, 86 (4): 219-224.

Wells, GD & Norris, SR. 2009. Assessment of physiological capacities of elite athletes & respiratory limitations to exercise performance. *Paediatric Respiratory Reviews*, 10 (3): 91-98.

White, PD & Naish, VAB. 2001. Graded Exercise Therapy for Chronic Fatigue Syndrome: An Audit. *Physiotherapy*, 87 (6): 285-288.

Yun-tao, MA. 2011. Homeostasis and Stress in Sports and Exercise. *In Acupuncture for Sports and Trauma Rehabilitation*. Saint Louis: Churchill Livingstone. 6-19

Ziltener, JL, Leal, S & Fournier, PE. 2010. Non-steroidal anti-inflammatory drugs for athletes: An update. *Annals of Physical and Rehabilitation Medicine*, 53 (4): 278-288.