GUEST EDITORIAL

It is indeed a privilege to be guest editor of this edition of the South African Journal of Sports Medicine which is devoted to Drugs in Sport. I have been involved in drug testing for 9 years and have been campaigning for at least 6 of those for the establishment of a government funded central drug control agency. It is therefore a great pleasure to confirm that such an agency is now in place. Similarly,it is a pleasure to note that Dr Pieter van der Merwe's Analytical Laboratory in the Pharmacology Department of the University of the Orange Free State has officially been accredited by the International Olympic Committee. It is the first in Africa and the second in the southern hemisphere.

Dr van der Merwe's statistical analysis of the results of drug testing for banned substances over a 9 year period makes fascinating reading. The main aim of drug testing is not to catch the culprits, but to act as a deterrent. Table I shows this clearly when one notices that the average percentage of positive tests was between 5 and 6% for the first 6 years. Then over the past 3 years, in spite of more tests being conducted, the percentage dropped significantly to only 1,1% of positives. The most important aspect of the control of drugs in sport is an education programme. With the recent establishment of the Central Drug Agency, this will soon become a reality. Together, an effective education campaign and out-of-competition testing on a regular basis, will have a powerful effect on controlling the abuse of drugs in sport.

Most readers will be quite familiar with the inherent risks and dangers to athletes taking anabolic-androgen steroids (A.A.S), so it is refreshing to read Dr Lambert's scientific analysis of the proposed mechanisms of action of AAS on increasing muscle mass and strength. Whereas the mechanism of increased protein synthesis is generally known, the possibly more important anti-catabolic effect of AAS is less known.

The problem of harmful and ergolytic drugs in sport is comprehensively covered by Dr Constantinou. He gives numerous examples of the potentially harmful side effects of ordinary and acceptable drugs. So, for the elite sportsperson, one has to prescribe with even greater caution and not only avoid banned substances.

Blood testing as a adjunct to urine testing was introduced for the first time at the Winter Olympics in Lillehamer, Norway in 1994. Since then, Canada has also elected to do limited blood testing in selected events. There are many potential pitfalls, especially legal and ethical issues, but also several practical ones. Once these have been overcome we are likely to see blood testing (sampling) being used more widely, especially in sport where blood doping and use of erythropoeiten is suspected.

The publication of this quarterly journal happens to co-incide with the Rugby World Cup 1995 competition. So far, there have not been any positive drug tests. The editorial board of this journal therefore wish the organisers and players a drug and scandal free tournament!

Dr Joe Skowno

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Blood doping and blood testing

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Introduction

There is an ever increasing interest in the sport community about the use of blood sampling and testing for doping control purposes. In conjunction with the 1994 Commonwealth Games in Victoria, Canada, the Canadian centre for Drug free Sport held a workshop to discuss blood sampling for doping control and the issues it raises for drug free sport. As one of the South African team doctors I was invited to the workshop and much of the information in this article was obtained from this workshop.

Definition

The International Olympic Committee banned list defines **blood doping** as *"... the administration of blood or related red blood products to an athlete other than for legitimate medical treatment."*

Blood doping is based on the principle that the amount of oxygen available to body tissues is limited by the number of red cells in the blood. In theory, increasing the red blood cells increases the amount of oxygen to the tissues which in turn allows the athlete to exercise more vigorously or for longer.

An explanation of how this could be achieved is that an athlete could have blood removed, the red cells (erythrocytes) separated from the plasma, and the red blood cells freeze preserved. Meanwhile the athlete's own red blood cells would gradually return to normal in about two months. Shortly before a competition, the red blood cells could be thawed, reconstituted with normal fluid, and then reintroduced into the athlete's blood stream. Consequently, the athlete would have the benefit of an abnormal number of red blood cells, a condition known as erythrocythemia. The preservation of blood requires a sophisticated process and considerable laboratory assistance. However, there is an easier method, that of transfusing someone else's blood into the athlete (non-autologous or homologous blood doping).

Benefits of blood doping

Blood doping would be of benefit to athletes in events taking place over a long time. For example, long distance running, cross country skiing and cycling.

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Risks of blood doping

- Infections, including HIV infection.
- Potentially lethal reactions from incompatible blood.
- Allergies
- High blood pressure

Blood sampling

The use of blood sampling (as opposed to urine testing) has advantages and disadvantages.

Advantages of using blood samples

- Relatively fast procedure to collect sample
- Allows more reliable quantitative analysis for some substances
- Identity of samples indisputable
- Required to detect homologous (non-autologous) blood doping
- Provides best matrix to detect autologous blood doping
- Probenecid (masking agent) is now easily detected
- Corticosteroids are more easily detected
- Blood may be useful to confirm testosterone doping although urine may give much greater concentrations
- Plasma usually has greater concentration of peptide hormones
- There are population-based reference ranges for clinical haematological tests

Disadvantages for using blood samples

- Legal/Ethical issues
- Sampling by medically trained person; small sample size (20ml); preparation of sample
- Reference ranges for peptide hormones and their secondary factors are unknown
- Analysis methodology is not fully established to do a complete profile, therefore urine analysis is still required
- Urine is a better matrix for measuring Beta-2 agonist (eg Clenbuterol)
- Because of quick half-life of peptide hormones (6-24 hours) and steroids (72 hours) in blood, they may be hard to detect
- Haematological parameters may be altered by altitude and hard training
- Concentration of substances with low molecular weights are lower than in urine by a factor of 100-1000

Although progress is being made on the detection of blood doping, there is at present no foolproof method to determine that a high red blood cell count is the result of blood doping.

Dr Norman Gledhill, an exercise physiologist and former president of the Sports Medicine Council of Canada, met with IOC Medical Com-

mission representative Dr Arnold Beckett in July 1978 and presented him with the evidence that blood doping definitely enhances performance. Dr Gledhill suggested that blood doping be banned specifically. He was of the view that blood doping need not be detectable to be on the banned list. Athletes would know that blood doping was considered cheating and they would have to make the personal decision whether to cheat or not. Dr Beckett however, took the position that blood doping should not be banned until it could be detected. After the Los Angeles Olympic in 1984, members of the US Cycling team admitted having competed with the aid of blood doping. They had used blood from donors, not their own blood. Of seven athletes involved, three had become ill. Shortly thereafter, blood doping was banned by the IOC.

Blood sampling not blood testing

From a legal point of view, the analysis or actual testing itself is only one and perhaps the least problematic step in the use of blood samples for doping control. A preferable term is blood sampling because it is this "front end" of the process that is legally the most sensitive.

Blood sampling in doping control: some practical considerations.

Sampling procedure:

1. Blood sampling can be quicker than urine sampling. There is no need to wait for dehydrated athletes to produce a sample.
2. While physically invasive, blood sampling is far less a disruption to personal dignity. Female athletes in particular, may be more comfortable giving a blood sample than being scrutinised while giving a urine sample.

Analytical:

Non-autologous (homologous) blood can be tested.

Autologous blood at present cannot be detected.

Possibilities for the detection of autologous blood transfusions.

1. Repeated testing of erythropoeitin ratio to haemoglobin and haematocrit to establish normal values?
2. Parameters to detect haemolysis after the transfusion of stored blood?
3. Markers for senescent erythrocytes?

Legal and ethical aspects

These aspects are potentially very complicated because of the overlap and possible conflict between:

1. International laws such as the European Charter of Human Rights.
2. National laws such as domestic constitutional documents guaranteeing certain individual rights or protections.
3. State or provincial laws in nations with federal systems of government.
4. The rules of international sport governing bodies and the rules of their national affiliates.
5. The requirements of international games organisers, the International Olympic Committee and the Commonwealth Games Federation being obvious examples.

One area where conflict already manifests itself is dispute resolution, particularly the role, if any, of domestic courts in intervening in otherwise private sporting matters.

Particular issues

1. Consent:

Without informed consent the taking of a blood sample, being physically invasive, is considered legally to be an act of assault and battery with criminal and/or civil liability. This requires an additional and specific consent form, above and beyond consent to participate in doping control which is a condition of membership for all national sporting bodies and teams.

2. Use of blood sample:

It must be clearly stated on the consent form that the blood samples will be used for the testing for banned substances only. Under no circumstances may the sample be used for research or any other purposes unless clearly stated and endorsed and understood by the athlete concerned.

Conclusions

In view of the continuing problems associated with the relatively simple process of requiring, collecting, identifying and securing urine samples, the practical difficulties of a successful blood sampling programme should not be underestimated. If urine sampling can still problematic, due to human failure if nothing else, blood sampling will give rise to no fewer errors.

At the Winter Olympic in Lillehamer, Norway in 1994, limited blood sampling was done for the first time. This was discussed at the Workshop held in Victoria, and as a result two countries, Canada and Norway made the decision to use blood sampling as part of their doping control measures. Other countries will be monitoring their progress with interest and once many of the more serious problems have been ironed out it is likely that blood sampling will become an additional tool in doping control internationally.

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Anabolic-androgenic steroids: effects on muscle size and strength

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Introduction

Androgens are a class of steroid hormones which are secreted mainly by the testes, but also by the adrenal gland and ovaries. Testosterone is the main androgen secreted by the testes. Testosterone regulates differentiation and secretory function of the male sex organs and stimulates protein synthesis in muscle, bone and kidneys.[1]

Testosterone was isolated in 1935 and by 1937 pharmaceutical companies were already performing clinical trials with testosterone to determine it's potential muscle building properties and uses in medicine.[2] However, treatment with testosterone was not successful, because it was difficult to sustain high circulating plasma levels of testosterone after testosterone treatment. Orally ingested testosterone is rapidly absorbed into the portal blood and degraded by the liver, and injected testosterone is rapidly absorbed from the injected medium and degraded.[3]

However, from the early 1940's synthetic modified versions of testosterone were developed. These drugs were more resistant to degradation and were used clinically to promote muscle growth after chronic illness, as a hormone replacement therapy for hypogonadal males, and as treatment for depression. These synthetic forms of testosterone have become known as anabolic-androgenic steroids (AAS). Given the results of the early tests which showed positive effects on muscle growth, it was not surprising that sports participants started experimenting with AAS with the aim of trying to improve their physical performance.[9] By 1976 AAS's reputation among athletes and officials had grown to the point that the International Olympic Committee banned their use. However, that has not reduced their use and currently in South Africa they are being used across the spectrum from schoolchildren[4,5] to elite bodybuilders[6] in an attempt to improve muscle size or strength.

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There are many issues about drug use in sport, ranging from ethical considerations, the concept of cheating, and the problem of side-effects. It is beyond the scope of this review to discuss all these issues. The following discussion will therefore focus on the ergogenic and myogenic properties of AAS, with the aim of developing an explanation for their mode of action based on well controlled mechanistic and observational studies. Readers interested in the side-effects of AAS are referred to Hickson et al (1989) for a detailed discussion.[7]

Do AAS promote an increase in muscle mass and strength?

In a recent study 97% of all subjects who had used AAS believed they had become stronger as a result of AAS.[6] The perceived average increase in strength as a result of AAS use was around 30%.[6] However, this overwhelming belief in the ergogenic properties of AAS is not shared with the same enthusiasm by many scientists, probably because of the lack of sound scientific evidence. This can be attributed to the inherent problems in conducting well-controlled studies on the effects of AAS which has resulted in an unclear understanding of the mechanisms of action of AAS administered in supraphamalogical doses.

Inherent problems in AAS experimentation

(i) Blind experiments

It is impossible to do a blinded experiment with subjects either using supraphamacological doses of AAS or placebos, because the subjects in the AAS group will know they are using AAS within a few days from the overt symptoms which they develop. This may affect the outcome of the experiment because it has been shown that athletes get stronger if they believe they are ingesting AAS, even if they are in fact ingesting placebo.[8] In addition, other factors which may increase strength (training intensity, motivation, nutrition) are all affected by AAS.[9] Therefore, many studies have been confounded by poor control over these factors.

(ii) Ethics

It is illegal to provide AAS to subjects in the actual doses and combinations used by sports participants. The doses used by sports participants

sometimes exceed the maximum clinical doses by 18 fold,[6] with potentially serious side-effects.[7] Therefore, the results from studies in which clinical doses of AAS have been used, cannot be generalised to AAS use among sports participants.[10]

Concensus studies

The experimental constraints have resulted in a paucity of scientific literature on the ergogenic and myogenic properties of megadoses of AAS. In a position statement in 1977 the American College of Sports Medicine concluded that *"... there is no conclusive scientific evidence that extremely large doses of anabolic steroids will aid or hinder athletic performance".*[11] However, the studies used in developing this position statement were later criticised because many used inexperienced, untrained subjects, lacked dietary controls, used low intensity training and used non-specific forms of testing muscle strength. Therefore, in 1984 the ACSM revised the position statement to conclude: *"anabolic steroids in the presence of an adequate diet can contribute to increases in body weight, often in the lean mass compartment ... and the gains in muscle strength achieved through high intensity exercise and proper diet can be increased by use of anabolic steroids in some individuals".*[12] Subsequently, other reviews of the literature have concluded from carefully selected studies that *"anabolic steroids have the most pronounced affect in those athletes who have trained to the point that they are in a chronic catabolic state",*[13] and *"anabolic steroids may slightly enhance muscle strength in previously strength trained athletes".*[10]

Therefore, it may be concluded from the anecdotal and scientific evidence that AAS increase muscle mass and strength providing the athlete is (i) highly trained, (ii) training hard while using the drugs, and (iii) eating a high energy, high protein diet. The proposed mechanisms of action will now be discussed in more detail.

Mechanism of action of AAS

(i) Circulating androgens

The testes secrete about 5-10mg testosterone per day,[3] which maintains a circulating plasma concentration of 0.5-0.6µg/100ml.[1] About 40% of circulating testosterone is bound to albumin and 58% to sex hormone binding globulin. The remaining testosterone is free and reflects the testosterone available for interaction with target cells.[1] This concentration of circulating free testosterone remains fairly constant until the age of 50 years, after which it starts to decrease.

(ii) Androgen receptors

Free testosterone binds to androgen receptors located in the cytoplasm and nuclei of cells. These receptors are protein molecules and occur in most tissues.[14] Androgen sensitive tissue, such as the prostate glands, may have 25 times the

number of androgen receptors compared to skeletal muscle.[15] AAS and endogenous androgens bind with equal affinity to the receptors.[16] The androgen receptors in most tissue are saturated in men with normal circulating testosterone concentrations,[17] therefore, the number of androgen receptors may be a limiting step in AAS action.[18]

An increase in circulating testosterone concentrations, for example after AAS treatment, down-regulates these receptors[19] and a decrease in circulating testosterone concentration, for example after castration, up-regulates the receptor concentrations.[20] Exercise training also increases the receptor concentrations in skeletal muscle. Inoue et al., (1993) showed that the androgen receptor concentration in rat muscle increased by 25% three days after starting an exercise programme. However, by 4 weeks the muscles had not stopped growing, even though the androgen receptor concentration had by this time plateaued.[21] The authors concluded that an increased receptor concentration is an important trigger for initiating muscle hypertrophy, but thereafter other factors are necessarily for the maintenance of the hypertrophic response in skeletal muscle.

(iii) Metabolic action of androgens

The generally accepted model explaining androgen-receptor action is that the testosterone molecule binds to the androgen receptor in the cytoplasm and induces a conformational change to the receptor. The androgen-receptor complex is translocated into the nucleus and subsequently binds to chromatin acceptor sites where it initiates the transcription process, resulting in new messenger RNA formation.[18] After translation on ribosomes, new protein is synthesized.[22] The nature of the protein synthesized is dependent on the target tissue.

This theoretical model shows that AAS exert their ergogenic and myogenic effects by stimulating protein synthesis. However, as described earlier, there is a limited capacity for anabolic steroid binding to androgen receptors since the receptor number is a limiting factor and may be down-regulated after AAS treatment. Although protein synthesis may increase slightly after AAS treatment,[23] this increase does not account for the total increase in muscle mass. Therefore, further explanation is required.

(iv) Anti-catabolic effects

Another mechanism for the action of AAS is that they exert their effects through the glucocorticoid receptor. Glucocorticoids are a class of hormones secreted mainly from the adrenal cortex during a bout of exercise or in response to any stressful stimuli. These hormones, of which cortisol predominates, promote amino acid efflux from muscle and increase protein degradation.[24] In essence, androgens have an anabolic, whereas glucocorticoides have a catabolic action. These catabolic processes, as a result of glucocorticoid action, seem to be exacerbated following high intensity training. The exposure to excessive training loads for prolonged periods causes the overtraining syndrome which results in elevated circulating cortisol concentrations at rest.[25] This causes a catabolic state in the athlete.[26] There are separate receptors for androgens and glucocorticoids in skeletal muscle[27,28] with 20 to 100 times more glucocorticoid receptors compared to androgen receptors in muscle.[27,28,29] The results of several experiments have shown that androgens can compete with glucocorticoids for glucocorticoid receptor binding in skeletal muscle.[24,29] This antagonism can block the action of glucocorticoids, and therefore, through this process androgens have an anti-catabolic effect. The net effect of this anti-catabolism will be to increase muscle mass.

Therefore, there is a strong argument that AAS exert their myogenic effects, not through increasing protein synthesis alone, but rather though reducing muscle breakdown. In addition, the anti-catabolic effect of AAS may allow the subject to train harder without developing the overtraining syndrome. Additional training alone may also stimulate muscle growth and strength.

Summary

There is both anecdotal and scientific evidence that suprapharmacological doses of AAS increases muscle mass and strength in previously trained athletes undergoing intensive training and eating a diet sufficient in protein and energy. Although an increase in protein synthesis contributes to the increase in muscle mass and strength, it is likely that the more significant mode of action of AAS is through reducing the catabolic processes which occur following intensive training.

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"Ergolytic and harmful drugs in sport"

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Drugs in sport evokes certain dark images in one's mind, with behind the scenes, secretive use of tablets and injections to improve on optimum preparation; and beat an opponent. Cheating in this way may involve so-called ergogenic aids. "Ergo" is derived from the Greek word ergon[1] and "gen" from the Greek "to be produced"[1] and has come to mean any means used to enhance energy utilization (performance).[2]

"Lytic" is an adjective for "dissolution",[3] and ergolytic therefore defines means of impairing performance. This of course is something that no athlete would deliberately or intentionally pursue. Even small ergolytic effects may have vastly significant effects on performance, particularly significant in elite athletes.

Doping control is covered elsewhere in this journal, and is necessary for several reasons. One such reason relates to the potential health risks.[4] This paper highlights this aspect in relation to potential ergolytic effects and also classes of drugs that may not be banned, but may be harmful to sportspeople. Those drugs that have a blanket banning by the International Olympic Committee (IOC), and sporting codes that follow those guidelines, will not be alluded to here, and will be discussed elsewhere.

There are however some that are banned only in some sports, or have special conditions that may be applied: and include caffeine, alcohol, marijuana, corticosteroids and beta-blockers. These will be discussed, as they have relevance to the emphasis of this paper.

It should be remembered that all drugs, by virtue of having pharmacological properties, may have side effects. It is beyond the scope of this paper to cover the vast array of these and various drug interactions, and should be referred to as necessary.

Some drugs may be necessary to modify diseases, sustain or improve quality of life. These can be used for acute or chronic conditions, and many of these populations may be partaking in exercise. Some are used only for recreational purposes.

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The moral and ethical issues of the use of ergogenic aids has been greatly deliberated,[4,5] and to extend these arguments to include ergolytic and potentially harmful drugs complicates matters exponentially.

Therapeutic drugs will now be discussed in alphabetic order of class.

Analgesics: *(see also anti-inflammatories)*
Paracetamol is widely used as an analgesic due to the safety profile, and has no known ergolytic effect. High doses may lead to hepatic toxicity.[6] It is often found in combination analgesics, which may contain prohibited substances.

Codeine is no longer banned by the IOC, and may be an effective analgesic. This too is often found in combination analgesics and remedies for the relief of common colds. High doses may lead to narcotic side effects such as nausea, vomiting, dizziness, respiratory depression, and may have addictive properties.[7,8]

Antiarrhythmics: *(see also beta-blockers, antihypertensives)*
Often patients requiring these may be restricted form exercise for medical reasons, but others may be encouraged to exercise. These may have arrhythmias as side-effects and may impair cardiac output with exercise and lead to hypotension and syncope.[8,9]

Antibiotics:
All classes of antibiotics have side effects that may be severe enough in some individuals to affect performance. These vary from headache, gastro-intestinal (GIT) disturbances, nausea, vomiting, diarrhoea and idiosynchratic reactions.[6] Often the reason antibiotics were prescribed for will limit one's participation in exercise and sport, and antibiotics per se are not contra-indications to participation.

Anticoagulants:
Warfarin and heparin type anticoagulants may have significant drug interactions with non-steroidal anti-inflammatories drugs (NSAID) and anabolic-androgenic steroids, which sportspeople may be taking. Their side effects of nausea, vomiting, skin reactions and fat necrosis[6] may affect performance. Their real dangers are that haemorrhage may occur. This may be secondary to acute injuries, and are therefore relative contra-indications in collision and contact sports.

Haemorrhage may also occur in an occult form and lead to GIT, cystic or renal blood loss, with subsequent anaemia and impaired work capacity.

Antidiarrhoeals:
In endurance athletes and particularly in runners there is an increased incidence of GIT symptoms including diarrhoea.[8,10] The management may include antidiarrhoeals such as loperamide,[8,10] H_2-antagonists[8] with side effects of nausea, dizziness, fatigue and allergic reactions.[6] Anticholinergics may be included in some preparations and cause dry mouth, blurred vision, tachycardia and urinary retention.[6]

Antiemetics:
Most drugs in this class have sedation as side effects, and some have extrapyramidal neurological impairment, both which may significantly affect performance.

Antiepileptics:
Most anti-epileptic drugs lead to poor concentration, co-ordination and cognitive function.[6,8] They may also cause nystagmus, ataxia and drowsiness, and therefore have special reference for participation in contact sports, those requiring fine dexterity skills and scuba diving.

Antihistamines:
Oral and injectable preparations of the older types cause drowsiness, muscle weakness, dizziness, headache, visual disturbances, dry mouth, thirst, feeling of heaviness in upper limbs, incoordination, tight chest, and may precipitate convulsions.[6] Their use will certainly lead to ergolytic effects. The newer generation long-acting forms are more selective, and are said to lack significant sedative effects, but may still cause nausea, fatigue, dizziness, myalgia and arthralgia, and so may still have undesirable effects on performance. Topical creams may cause local irritation with minimal systemic effects, but nasal preparations may cause somnolence, headache, tiredness and epistaxis.

Antihypertensives:
Diuretics are prohibited by the IOC[11] and may have side effects that may impair performance and be harmful to health. Some of the older, nonspecific, calcium channel blockers may impair cardiac output with exercise.[6,8]

Beta-blockers are banned in some sports, but may be used therapeutically for hypertension, certain arrhythmias, angina, anxiety and migraine prophylaxis.[6,9] They may aid in fine coordination sports such as archery and pistol shooting by blunting anxiety and fine tremors.[4,8]

However, reduced aerobic capacity[4] and lethargy[6] may impair performance in endurance activities. Other side effects may be risks to health and include bronchospasm, masking of hypoglycaemic symptoms and adversely affecting lipid profiles,[6,12]

Alpha-blockers may cause syncope, hypotension, acute tachycardia.[6,9] Angiotensin Converting Enzyme inhibitors may cause chronic cough, hypotension, headache, dizziness, fatigue, dyspepsia, nausea and interact with NSAIDs.[6,9]

Anti-hypercholesterolaemics *(and dyslipidaemic modifying drugs)*
There are various classes of such drugs, and have important implications as people taking these are (or should) be participating in controlled or supervised exercise programs. The following side effects may affect performance.

Fibrates – GIT disturbances, sweating, nausea, arrhythmias, blurred vision, fluid retention, headache, dizziness.[6,8]

Nicotinic acid – Abdominal pain, hyperglycaemia, hyperuricaemia (?gout), blurred vision, hypotension.

HMGCoA reductase inhibitors – nausea, constipation, headaches, insomnia, myalgia, muscle stiffness, postural hypotension and peripheral neuropathy.

Probucol – diarrhoea, abdominal pain, arrhythmias, hyperuricaemia.

Anti-inflammatories:
Non-steroidal anti-inflammatories are used extensively by sportspeople in prescription and over the counter formulations. They function by inhibiting prostaglandin production[4,6,8,13] and are used mostly for acute injuries, some believing in their use for fractures and chronic injuries too.[4] Among the side effects that are significant and dangerous are GIT blood loss,[7,8,14] peptic ulceration (often asymptomatic)[8] and renal damage with acute tubular necrosis and renal failure.[7,8,13] This is particularly of risk in the presence of dehydration. Other side effects include bronchospasm, tinnitus, allergic reactions, headaches and hepatic damage.[7,8] Some have strong analgesic properties and partaking in exercise or sport under their influence of analgesia (or combined with analgesics) may worsen injuries and increase the risks for other injuries. Under these exercising conditions the risks for GIT haemmorrhage, diarrhoea and renal damage are particularly high. Aspirin may have all the above problems and in addition has strong anti-platelet adhering properties, which may lead to increased bleeding in acute injuries.[6,8] Use of aspirin in children has the risk of Reye's syndrome, which may be fatal.[3,4] Some of the drugs and formulations report fewer side effects, but are all still at risk of the above.

Topical NSAIDs (gels, creams, ointments, transcutaneous patches, sprays) can lead to systemic effects if used in high doses, frequently or for prolonged periods of time.

Corticosteroids have potent anti-inflammatory properties with little data supporting their benefits in most acute or chronic sports injuries, but

have significant side-effects.[4,6] Inhaled corticosteroids and topical preparations are permitted by the IOC (with written medical support) and the small doses have few side effects.[4,11] Oral and injectable forms interfere with collagen synthesis and may lead to poor tissue healing, with increased risks of further injury. The systemic catabolic effects may impair performance and the side effects include myopathies, fluid retention, immunosuppression, electrolyte disturbances, inhibition of growth, GIT disturbances and bleeding.[4,6,7,8]

Antimalarials:
These are often taken by sportspeople travelling to compete. Most antimalarial drugs may cause GIT side effects with abdominal pain, nausea, vomiting and diarrhoea; which may affect performance.[6] These are often transient and in general are not a problem. Mefloquine has neurological and neuropsychiatric disturbances that makes it the only anti-malarial drug contra-indicated in scuba diving.

Anxiolytics:
These may affect psychomotor skills, reaction time, cognitive function, level of wakefulness and thermoregulation[6,8] and can therefore be ergolytic and increase risks of injury. These and other psychoactive drugs are definitely contra-indicated with scuba-diving, and can be life-threatening.[15]

Haematinics *(and iron supplements):*
These can lead to GIT disturbances, nausea, vomiting, abdominal pain and constipation.[6] They will however tend to have an improving performance effect as the anaemia is corrected.

Hypoglycaemics:
As with hypertension and dyslipidaemias, diabetics often include exercise as part of their management protocol. Oral hypoglycaemics may cause GIT disturbances, headaches and dizziness.[6] These and insulin may cause hypoglycaemia, and exercise prescription in diabetics should address the issues of nutrition, timing of taking insulin/medication, intensity and volume of exercise. As a rule insulin dependent diabetics should not partake in scuba diving.

Recreational habits may include the use of ergolytic substances or harmful drugs. Here reference is made in alphabetic order of substance.

Alcohol:
The IOC bans alcohol in riflery[18,19] for the obvious dangers, and has special considerations for other sports. Several sports ban alcohol, and locally Basketball South Africa has recently banned its use (personal communication). There has been no evidence of ergogenic effects, but there may well be ergolytic effects with alcohol intake. Certainly there are health risks with abuse. Alcohol may cause hypoglycaemia by reducing glucose release from the liver,[7] cause diuresis by reducing vasopressin secretion[20] and impairing psychomotor ability, memory and decision making skills, up to 48 hours after ingestion,[2,7] with obvious consequences. Alcohol may also have addictive properties.[4,7,17,21]

Caffeine:
Although a stimulant and banned in certain quantities by the IOC,[4,7,8,11] it is included here because it is often taken recreationally in coffee and colas. No studies have shown significant improved physiological performance in smaller doses,[2] but the side effects may in fact impair performance, and include irritability, anxiety, insomnia, palpitations, headache, diarrhoea, hypertension and diuresis.[8,16,17] In high doses there have been reports of seizure, coma and even death.[18]

Tobacco:
Smoking tobacco may have deleterious effects on physical performance.[7] This common habit affects mucous membrane cilia function and mucous production with increased incidence of dyspnoea and coughing with exertion.[7] Carbon monoxide has a 300 X affinity for oxygen over haemoglobin (Hb) and smokers of 15-25 cigarettes per day can have 6-7% carbon monoxide combined to Hb. This may significantly affect physical exertion and this together with mucous plugging and air trapping may lead to increased risks of gas embolism in scuba divers.

Nicotine is readily absorbed and although undergoes hepatic metabolism and excretion, can lead to increased adrenaline and noradrenaline with tachycardia and hypertension for several hours after smoking.[7,20] For this reason alone smoking should be discouraged for 24 hours before competition. Nicotine also has addictive properties.[7] Tar is also a respiratory irritant and carcinogen. The risks of passive smoking are

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becoming more recognised and may have significance in team sports. Chewing tobacco excludes the inhalation problems of carbon monoxide and tar, but there are local irritant and potential carcinogenic effects.

Nicotine is still absorbed, causing its effects. Smoking is also a well recognised risk factor for coronary heart disease and will therefore increase the risks of sudden death while exercising.[7,8,22]

Marijuana:
This is legally prohibited in South Africa and can lead to criminal charges if found to have this in one's possession. The IOC has special considerations,[4,11] and is found to be widely used throughout Africa,[23] Use of marijuana may affect judgement and performance.[4,8]The addictive properties[7] and disinhibited motivation effects[8] can affect an athletes performance and indeed career. The issue of passively inhaling marijuana has been raised and has interesting implications.

As a general rule any drug has the potential to affect performance, and seemingly innocuous drugs may in some individuals or conditions have severely undesirable effects. A case in point is scuba diving, where relatively harmless drugs may have unpredictable effects under hyperbaric conditions.

Just as athletes, coaches and health care professionals should as a rule ensure any medication taken has no banned substances, so they should ensure there are no ergolytic or potentially hazardous consequences.

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Verbode Middels in Sport: Resultate van toetsing vir die tydperk 1986-1994

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Abstract
Screening procedures were performed on 5 714 urine specimens collected from competitors in 43 different sports during the period 1986-1994. Samples were tested by gas chromatography and gas chromatography/mass spectrometry for the presence of stimulants, narcotics, anabolic steroids, diuretics and ß-blockers.

Drugs classified as forbidden substances by the International Olympic Committee were detected in 196 (4.17%) of the samples. Anabolic steroids were detected in 86 (43.9%) of the samples and stimulants in 82 (41.8%). The anabolic steroid most frequently detected was 19-nortestosterone while the stimulant was fencamfamine. The ephedrine's as a group accounted for 52 (61.5%) of the positive stimulant samples. This high incidence of the use of banned substances indicates that the dope testing programme to curb use of banned substances by competitors in sport should continue and be expanded to all sport.

Inleiding
In 1983 is die eerst toetse vir die gebruik van verbode middels in sport in Suid-Afrika gedoen toe die Departement Farmakologie aan die Universiteit van die Oranje Vrystaat urienmonsters geanaliseer het vir 'n beperkte aantal stimulante.[1]

Sederdien is die analitiese metodes sodanig ontwikkel om vir al die groepe middels wat deur die Internasionale Olimpiese Komitee (IOK) as verbode geklassifiseer is, te kan toets. Hierdie groepe middels sluit in stimulante, anaboliese steroiede, narkotiese analgetika, diuretika en ß-blokkeerders. Tans voldoen hierdie laboratorium aan internasionale standaarde en is 'n ten volle geakkrediteerde laboratorium van die IOK- die enigste in Afrika en, benewens Australië, die tweede in die Suidelike Halfrond.

Hierdie verslag handel oor die resultate van die afgelope 9 jaar van toetsing in Suid Afrika.

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Metode
Urienmonsters is ontvang vanaf 43 verskillende sportsoorte in Suid Afrika. Die urienmonsters is versamel deur die betrokke sportliggame in die Envopak houers en daarna versend na die laboratorium. Elke urienmonster is in duplikaat versamel, 'n A-monster en 'n B-monster. Die A-monster is geanaliseer vir die teenwoordigheid van verbode middels[2-4] terwyl die B-monsters gevries is by -20°C vir moontlike latere analise.

Indien die resultate van die siftingsprosedures aangedui het dat 'n verbode middel teenwoordig mag wees is bevestigingsanalise op die A-monster uitgevoer volgens 'n internasionale aanvaarde protokol.[5] Sodoende is onteenseglike bewys gelewer vir die teenwoordigheid van die betrokke middel.

Resultate en bespreking
In die tydperk 1986-1994 is altesaam 5 714 urienmonsters geanaliseer, die resultate word aangetoon in tabel 1. Daar was 'n afname in die aantal positiewe monsters en in 1994 was die persentasie positiewe monsters feitlik gelyk aan die van die IOK geakkrediteerde laboratoriums vir 1992. Tabel 2 gee 'n ontleding van die verbode middels in hulle onderskeie groepe. 'n Totaal van 204 verbode middels is geïdentifiseer in 196 urienmonsters wat daarop dui dat sommige urienmonsters meer as een groep verbode middels bevat het. Soms is meer as een middel binne 'n groep in een urienmonster aangetoon. Tot vier anaboliese steroiede is in sekere urienmonsters geïdentifiseer. Van die 196 positiewe monsters het 86 (43.9%) anaboliese steroiede bevat en 82 (41.8%) stimulante. Dit is interessant om hierdie resultaat te vergelyk met die van die een-en-twintig IOK geakkrediteerde laboratoriums. Hulle syfers wys dat 552 (68.9%) van die 805 positiewe monsters anaboliese steroiede bevat het en 221 (27.5%) stimulante.

Uit tabel 2 blyk dit dat die afname in die aantal positiewe monsters oor die laaste 2 jaar hoofsaaklik toegeskryf kan word aan die vermindering in die aantal urienmonsters wat anaboliese

steroïede bevat het. Die afleiding kan gemaak word dat die streng optrede teen deelnemers wat hul skuldig maak aan die gebruik van verbode middels (tot 4 jaar skorsing uit enige kompeterende sport vir 'n steroied positief) die nodige uitwerking het. Dit sou egter verkeerd wees om hierdie afname slegs aan dié rede toe te skryf aangesien sommige sportsoorte waar die deelnemers beskou word as potensiële hoë gebruikers van anaboliese steroïede nie die afgelope 2 jaar toetse laat doen het nie. Dit word ook verder vermoed dat die werklike gebruik van anaboliese steroïede veel hoër mag wees as aangedui in tabel 2. Die vinnig-werkende steroïede, veral die orale preparate, word gouer uit die liggaam uitgeskei en sal dus nie meer tydens kompetisie in die liggaam teenwoordig wees nie en sal dus nie deur die toetse aangetoon kan word nie. Dit moet in gedagte gehou word dat anaboliese steroïede as "oefeningsmiddels" beskou word wat hoofsaaklik in die voorbereiding seisoen gebruik word en dus nie meer noodwendig tydens kompetisie in die liggaam teenwoordig is nie. Dit beklemtoon die belangrikheid van buite-kompetisie toetsing wat internasionaal op groot skaal toegepas word in 'n poging om die misbruik van anaboliese steroïede aan bande te lê. Meer as 40% van die aantal toetse wat deur die IOK geakkrediteerde laboratoriums in 1992 gedoen was, was buite-kompetisie toetse. Hierdie soort toetsing word nog nie ernstig in Suid Afrika toegepas nie.

Tabel 3 toon dat nandroloon die anaboliese steroïed is wat die meeste in die urienmonsters voorgekom het. Dit moet toegeskryf word aan die langwerkendheid van hierdie preparaat. In tabel 3 word ook 'n vergelyk gemaak tussen Suid Afrika en Internasionaal ten opsigte van die aantal steroïede as 'n persentasie van die totale aantal steroïede wat in die urienmonsters voorgekom het.

Tabel 4 toon dat fenkamfamien, wat in die skedule 5 tonikum Reactivan® voorkom, die stimulant is wat die meeste aantal kere in die urienmonsters voorgekom het. Dit wys op die gewildheid van hierdie tonikum onder deelnemers aan kompeterende sport. Hierdie middel word egter nie internasionaal veel as 'n positief gevind nie. Die efedriene as 'n groep nl. efedrien, pseudoefedrien, fenielpropanolamien en norpseudoefedrien was verantwoordelik vir 52 (61.5%) van die positiewe stimulant monsters. Hierdie syfer vergelyk redelik goed met die 46.2% wat internasionaal gevind word. Tabel 5 toon dat die efedriene in al 9 jaar 'n groot bydrae gemaak het tot die

Tabel 1

RESULTATE VAN DIE TOETSING VIR VERBODE MIDDELS IN SPORT IN SUID AFRIKA IN DIE TYDPERK 1986-1994

Jaar	Aantal byeenkomste	Aantal monsters	Totale positiewe monsters	
			Aantal	%
1986	16	152	8	5.3
1987	17	161	6	3.7
1988	19	230	12	5.2
1989	33	330	18	5.5
1990	41	482	27	5.6
1991	55	711	42	5.9
1992	129	1 472	52	3.5
1993	104	1 254	21	1.7
1994	103	922	10	1.1
Totaal		5 714	196	4.17
1992*		84 088	805	0.96

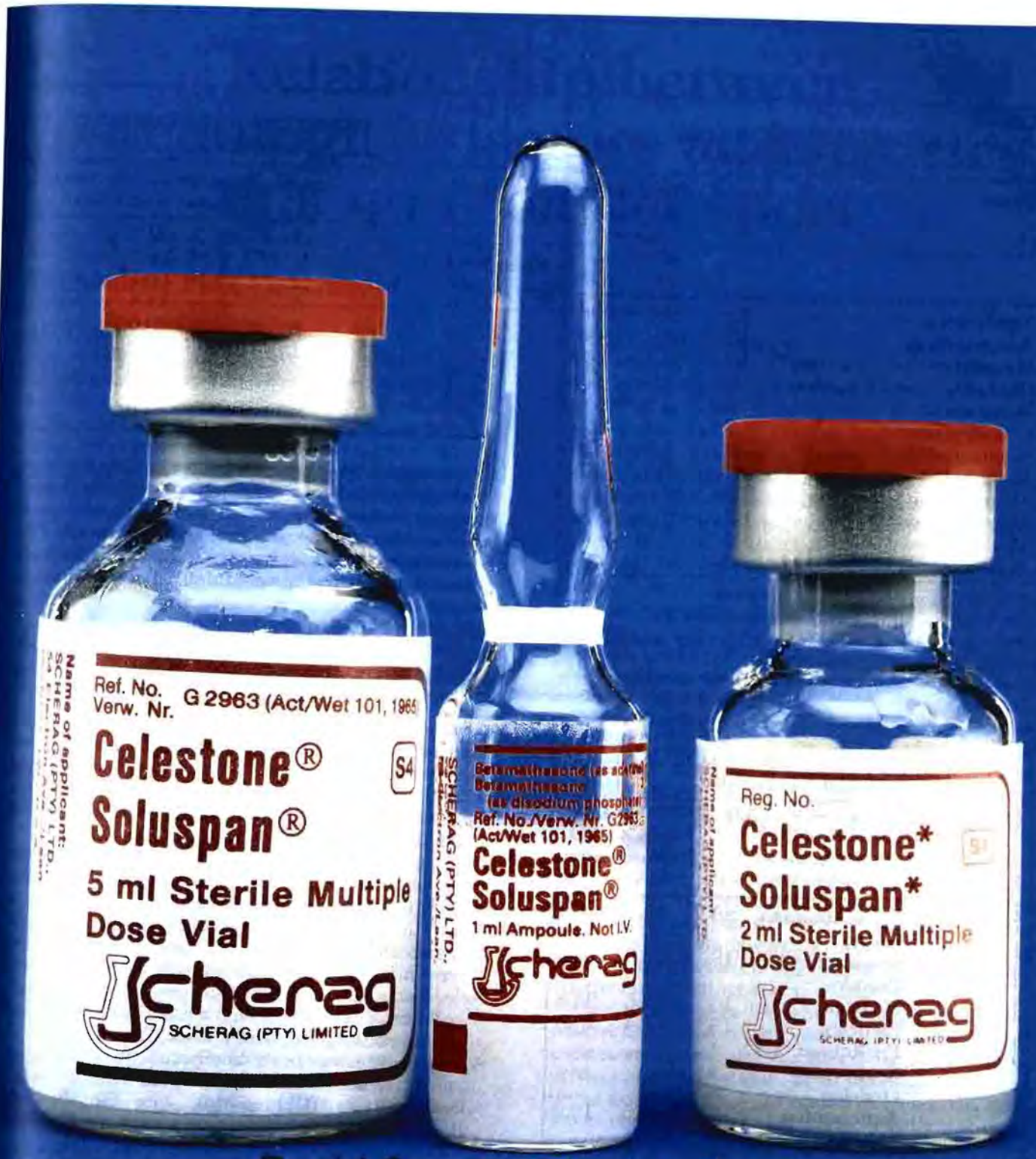
** IOK geakkrediteerde laboratoriums (n=21)*

Tabel 2

VERDELING VAN DIE AANTAL MONSTERS WAT VERBODE MIDDELS BEVAT IN DIE VERSKILLENDE GROEPE

Jaar	Stimulante	Narkotiese analgetika	Anaboliese steroïede	Diuretika	ß-Blokkers	Ander	Totaal
1986	7	0	1	0	0	0	8
1987	4	0	2	0	0	0	6
1988	4	3	7	0	0	0	12*
1989	10	2	6	0	0	0	18
1990	14	1	13	0	0	0	27*
1991	18	3	18	0	0	5**	42*
1992	12	6	29	0	5	0	52
1993	9	2	8	2	3	0	21*
1994	4	1	2	3	0	0	10
Totaal	82	18	86	5	8	5	196
1992***	221	72	552	47	10	1**	805*

* *Sommige urienmonster het meer as een verbode middel bevat*
** *Probenesied as maskeermiddel*
*** *IOK geakkrediteerde laboratoriums (n = 21)*



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Tabel 3
ANABOLIESE STEROÏEDE GEÏDENTIFISEER

Naam	Aantal	Presentasie van totale aantal steroïede	
		SA	IOK
Nandroloon	72	62.0	29.9
Testosteroon	12	10.2	33.9
Metenoloon (Primobolan®)	10	8.5	9.4
Oksimetoloon (Anapolon®)	8	6.8	1.1
Boldenoon*	7		
Oxandroloon**	3		
Stanozolol**	2	1.7	9.4
Dianabol**	1	0.8	8.0
Mesteroloon (Proviron®)	1		
Drostanoloon**	1		
Metieltestosteroon	1		
Probenesied***	5		

* Slegs vir veterinêre gebruik
** Nie in Suid Afrika geregistreer nie
*** Gebruik as maskeermiddel vir anaboliese steroïede

Tabel 4
ANDER VERBODE MIDDELS GEÏDENTIFISEER

Groep	Naam	Aantal	Presentasie van aantal stimulante	
			SA	IOK
Stimulante	Fenkamfamien	26	30.2	1.4
	Feniel-propanolamien	18	20.9	10.4
	Pseudo-efedrien	16	18.6	25.8
	Efedrien	15	17.4	10.0
	Prolintaan	4		
	Norpseudo-efedrien	3		
	Amfetamien	3	3.5	12.2
	Metielfenidaat	1		
Narkotiese Analgetika	Kodeïen*	16		
	Propoksifeen	2		
Diuretika	Hidro-chlorotiasied	3		
	Furosemied	2		
ß-blokkers	Propranolol	5		
	Sotalol	1		
	Oxprenolol	1		
	Atenolol	1		

* Word sedert 1994 nie meer as 'n verbode middel geklassifiseer nie.

Tabel 5
AANTAL MONSTERS WAT EFEDRIENE BEVAT

Jaar	Aantal	Presentasie van totale aantal positiewes
1986	5	62.5
1987	1	16.7
1988	2	16.7
1989	8	44.4
1990	8	29.6
1991	11	26.2
1992	10	19.2
1993	4	19.0
1994	3	30.0

totale aantal positiewe monsters. Hierdie verbindings word dikwels aangetref in vele verkoue, griep en hoes medisynes en kan sonder 'n voorskrif van 'n medikus in apteke gekoop word. Dit veroorsaak dat sommige deelnemers medisynes gebruik sonder om te besef dat dit verbode middels bevat. **Die onus rus egter op die deelnemers self om toe te sien dat hulle nie enige medisyne kort voor 'n kompetisie gebruik wat verbode middels bevat nie. Onkunde word nie as 'n verskoning aanvaar as 'n urienmonster positief toets nie.**

Gevolgtrekking

Hierdie resultate toon dat:

1. sportliggame in Suid Afrika moet voortgaan met 'n verbode middel toetsingsprogram en dat so 'n program vir alle sportsoorte in Suid Afrika moet geld.
2. inligtings- en opvoedingsprogramme ingestel moet word om deelnemers, afrigters en sport administrateurs in te lig oor verbode middels en die gevare aan die gebruik daarvan.
3. geneeshere bewus moet wees daarvan dat sommige griep-, verkoue en hoespreparate verbode middels bevat en dus nie deur deelnemers gebruik mag word kort voor of gedurende sportbyeenkomste nie.
4. 'n program van buite-kompetisie toetsing deur alle sportliggame in Suid Afrika ingestel moet word.

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Relationship between psychological factors and injuries in a non-contact sport

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Abstract
The incidence and perceived impact of injuries sustained by forty-four athletes were monitored during a competitive season of road running in order to ascertain relationships between illness/injury and selected psychological factors. No significant relationships were found between illness/injury and the psychological variables of locus of control, extroversion, neuroticism and life events. Modest, but significant relationships were present between competitive anxiety, daily hassles and illness/injury. These results underline the desirability for athletes to acquire coping skills.

Key words: *Sports injuries, Road running, Stress.*

Introduction
In addition to obvious physical reasons, psychological factors may also play a role in the occurrence of sports injuries. For example, the relationship between personality and injury has been the subject of earlier research. However, results have been inconsistent. (Brown, 1971; Irvin, 1975; Jackson, Jarret, Barley, Kausch, Swanson & Powell, 1978; 1982; Passer & Seese, 1983; Valliant, 1981).

The relationship between stressful life events and sports injuries became the focus of more recent research. In 1970 Holmes reported that football players scoring high on the Social Readjustment Rating Scale (SRRS) were more likely to get injured than their low-scoring team-mates (Andersen & Williams, 1988). Bramwell, Masuda, Wagner and Holmes (1975) adapted the SRRS for use in a sports setting and found similar results to Holmes among football players. In more recent studies, Coddington and Troxell (1980) reported an increase in injuries among high-school football players as a result of factors such as stressful family events. Cryan and Alles (1983) also found a higher rate of injury among high-stress football players when compared to low-stress players. Hardy, Richman and Rosenfeld (1991) concluded that injury rate increased with an increase in life events and a decrease in social support. Passer and Seese (1983) made a distinction between positive and negative life events and found a relationship between negative life events and football injuries. This relationship did not apply to positive life events.

Although the life events/stress-injury relationship has been established in football, it is not so clear-cut whether this also holds for non-contact sports (Hardy et al., 1991). Kerr and Minden (1988), in one of the few studies dealing with non-contact sport (gymnastics), found that timing of injury was related to impending competition with an increase in injury rate as the competition drew nearer. They suggested that this pattern could be attributed to heightened stress associated with the impending competition. The study of Kerr and Minden (1991) is significant, but the results do not concur with those of Williams, Tonyman and Wadsworth (1986), who found no relationship between life stress and injury in another non-contact sport, namely volleyball.

One of the possible shortcomings of earlier research is the focus on major life events, to the exclusion of the stress caused by minor daily problems or irritations. It is hypothesized that these everyday stressful events decrease the athlete's coping ability and consequently increase the incidence of injury (Miller, Vaugh & Miller, 1990).

The study of the relationship of stress and injury would be incomplete without considering personality variables (Williams & Roepke, 1993). The purpose of this study was to ascertain the relationship between illness/injury and selected moderator personality factors and life stressors in a non-contact sport by monitoring athletes throughout a competitive season. This method of investigation differs from most previous studies, where factors such as life stressors were investigated post hoc only after the occurrence of the injury.

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Method

Sample

A group of 37 male and 7 female competitive road runners (Mean age = 24.4 yr) was used in this study. The sample consisted of experienced runners, ranging from provincial, national athletes to an Olympic silver medalist, who competed in a four month Winter league.

Instruments

The following questionnaires were mailed to the runners:

Sport Competition Anxiety Test (SCAT) (Martens, Vealey & Burton, 1990)

This 15 item questionnaire assesses differences in competitive trait anxiety, or the tendency to perceive competitive situations as threatening.

The Internal-External Locus of Control Scale (Rotter, 1966)

Locus of control is a personality trait dealing with the degree to which individuals view their lives and surroundings as under their personal control. Individuals who function on an internal locus of control believe that they can control their destiny on the bases of relatively permanent personal abilities and efforts. The I-E scale comprises 29 pairs of items. The score is the total number of external choices made by the respondent.

Eysenck Personality Inventory (Eysenck & Eysenck, 1964)

The EPI measures personality on two dimensions, namely introversion-extroversion and neuroticism-stability. Extroverts tend to be outgoing, impulsive and uninhibited and like to take part in group activities. Introverts are usually quiet and introspective. Persons scoring high on the Neuroticism scale are emotionally unstable and tend to overreact to everyday events. They frequently complain of vague somatic problems and tend to worry excessively.

The Social Readjustment Scale

This scale, developed by Holmes and Rahe (1967), ranks the magnitude of various life change events. A preset numerical weighting is given to each event on the presumed degree of the adaptation required by a typical individual. Respondents indicated the frequency of each event during the past three months. Because some events are of a personal and confidential nature, the values awarded to life events were relatively reduced to simplify calculating the final score. Respondents were requested to complete the questionnaire, calculate their score themselves and return only the final score. They were instructed to destroy the completed questionnaire.

Daily Hassles Scale (Kanner, Coyne, Schaefer & Lazarus, 1981)

A list of daily hassles was presented to respondents on two occasions (approximately three weeks after the commencement of the study and three weeks before the end of the season). Respondents checked the items for personal relevance and indicated the degree to which they were negatively affected on a 7-point Likert type scale ranging from ("little negative influence" to "excessive negative influence"). Examples of daily hassles are: Losing your keys, having to wait a long time for somebody, malfunctioning of a household appliance. Respondents could also report their own personal hassles. The number of hassles reported was multiplied with the rating of negative influence to calculate a total score. The sum of the totals of both lists was used in the analysis of data.

Injury Report Forum

This questionnaire was administered 5 times at regular intervals (approximately every three weeks) during the season. Respondents were asked to report the incidence of personal illness/injury. Respondents indicated the date and nature of illness/injury as well as its perceived severity on a 7-point Likert-type scale ranging from "Not at all serious" to "Very serious". The number of illnesses/injuries was multiplied with perceived seriousness to calculate a total illness/injury score. The sum of the scores of the five reports was used in the final analysis of results.

Results

A distinction was made between illness (e.g. influenza) and injury. A total of 58 illnesses/injuries were recorded over a period of four months. All of the subjects suffered some form of illness or another during the course of the season. Only 7 (16%) of the 44 athletes enjoyed an injury-free season. The following were the most prevalent illnesses/injuries reported: common cold (13), calf muscle injury (7), influenza (6), knee injury (5), hamstring injury (4), Achilles tendonitis (3), Iliar Tabular band syndrome (3), and groin injury (3).

The notable absence of shin splints among this group of runners cannot be explained. The relationship between the psychological variables of competitive anxiety, locus of control, extroversion, neuroticism, and illness/injury scores was investigated (Table 2). A significant, but modest correlation, was found between competitive anxiety and the combined illness/injury scores (r=.313; p<.05), as well as the injury-only scores (r=.302; p<.05). The data revealed an interesting positive relationship between competitive anxiety (SCAT) and daily hassles scores (r=.464; P<.001). No significant relationships were found between the other variables and illness/injury scores.

TABLE 1
CORRELATIONS (PEARSONS'S r) BETWEEN PSYCHOLOGICAL VARIABLES AND ILLNESS/INJURY

	Combined Illnesses/Injuries	Injuries only
SCAT	.313*	.302*
Locus of control	-.009	.117
Extroversion	.183	.109
Neuroticism	.123	.087
Life events	.103	-.027
Daily hassles	.334*	.299*

* p < 0.5

No significant relationships were found between life events and the number of combined illness/injuries (r=.145; p>.10). When analysing the total illness/injury scores (which take into consideration the incidence and perceived impact of illness/injury), no significant relationship was found with life events scores (r=.103; p>.10). However, significant relationships were found between daily hassles scores and combined illness/injury scores (r=.334; p<.05) and injury only scores (r=.299; p<.05).

TABLE 2
COMPARISON OF COMBINED ILLNESS/INJURY SCORES OF LOW AND HIGH ILLNESS/INJURY RATE ROAD RUNNERS

Variable	N	Mean	SD	*t*	*p*
Life events					
Low rate	24	8.13	16.88	1.09	.282
High rate	20	33.80	17.55		
Daily hassles					
Low rate	24	57.75	31.34	2.22	.032
High rate	20	78.75	31.22		

The effect of life events and daily hassles on illness/injury was investigated further. The median score of the sample for the combined illness/injury scores was 7, while the median for injuries only, was 3. The median scores were used to divide the sample into two groups, viz. a high illness/injury group (scores above the median) and a low illness/injury group, who produced scores at and below the median. No significant difference was found between the mean scores of the high injury and low injury groups regarding life events (Table 2). However, the high injury group produced significantly higher daily hassles scores than the low injury group, both in terms of combined illness/injury and injury only scores (Table 3).

TABLE 3
COMPARISON OF INJURY-ONLY SCORES OF LOW AND HIGH INJURY RATE ROAD RUNNERS

Variable	N	Mean	SD	*t*	*p*
Life events					
Low rate	24	31.00	17.93	0.123	.903
High rate	20	30.35	16.79		
Daily hassles					
Low rate	24	58.21	33.26	2.098	.042
High rate	20	78.20	29.14		

Conclusion

With the exception of competitive anxiety, the results of this study did not indicate significant relationships between the psychological variables under investigation, namely locus of control, extroversion, and neuroticism.

The failure to find a relationship between locus of control and the incidence of illness/injury is consistent with the results of earlier investigations (Lysens, Auwelle & Ostyn, 1986; Passer & Seese, 1983). The relationship between competitive anxiety and illness/injury partly supports the findings of Blackwell and McCullagh (1990), who concluded that competitive trait anxiety influences the severity but not the frequency of injury. However, in the present study, there may have been an overlap in the effect of anxiety and daily hassles scores and consequently both these variables could possibly explain a significant portion of variance in illness/injury occurrence. The injury/illness could render the athletes sensitive to daily hassles rather than the converse.

The failure of the present investigation to find a relationship between major life events and illness is not consistent with the earlier findings of Kerr and Minden (1988) with gymnasts. The retrospective design of the gymnastic studies may account for these inconsistencies.

A problem in studying multiple moderator variables is the need for large sample sizes. This would necessitate investigations that cover larger groups in diverse geographical areas. In addition, in order to gather objective data, rather than subjective and retrospective information, a more stringent method of recording illness/injury incidence is needed than utilising the personal reports of the athletes themselves.

Despite the failure of this investigation to unequivocally identify the moderator psychological variables associated with injury, the association found between daily hassles and illness/injury points to the importance of coping resources in the stress-illness relationship. Coping resources comprise a variety of behaviours and social support systems that assist the individual in dealing with life's day to day problems. The presence of a supportive social network of family, friends, coach and team-mates may have a positive effect on the stress-injury relationship. It is therefore desirable to assist athletes psychologically to counter the effects of daily stress.

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Inspired air humidity effect on respiratory function in normal adults during exercise

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Abstract
Inspired air humidity effect on respiratory function and expired air water vapour pressure (EWVP) during submaximal exercise was assessed at fixed inspired air temperature.

Spirometry in six healthy male subjects was performed at rest (baseline), after each of four consecutive ten minute exercise sessions (phases I-IV) and ten minutes post exercise (recovery), on humid inspired air (HIA), and on dry inspired air (DIA) one week thereafter.

Peak expiratory flow rate, forced vital capacity and forced expiratory volume in one second values were not significantly different between the DIA and HIA groups when compared with each group's baseline. EWVP with exercise was significantly decreased at phase IV versus phase II within the DIA group. No significant decrease in EWVP on HIA was noted.

Decreasing inspired air humidity, without simultaneously changing inspired air temperature, does not significantly influence respiratory function at submaximal exercise in normal adults.

Key words: *Humidity, Spirometry, Exercise.*

Introduction
The humidity and temperature of inspired air varies widely with ambient conditions and pharyngeal discomfort caused by the passage of under-humidified air is a common complaint amongst exercising laboratory subjects during oral breathing through a two-way non-rebreathing valve, and during the use of self-contained breathing apparatuses. Decreased forced expiratory volume in one second (FEV_1) and an increased airways resistance with cold dry (10°C, 10% Relative Humidity (RH)), cold humid (10°C, 50% RH) and hot dry (37°C, 15% RH) inspired air compared with hot humid inspired air in exercising asthmatic patients, and an increased FEV_1 with hot humid (37°C, 60% RH) inspired air compared with hot dry and cold dry inspired air in normal exercising subjects, has been noted.[1] However, most studies have concentrated on changing inspired air humidity and inspired air temperature simultaneously, and then examined the effects of these on asthmatic patients only.[2,3,4] One investigator[5] has shown that inspired hot humid (45°C, 95% RH) air during exercise increases tidal volume and decreases respiratory rate[5,6] as compared with cool dry (26°C, 60% RH) air, in normal subjects. Most research experiments are conducted in a laboratory with a constant room temperature.

The effect on pulmonary function and expired air water vapour pressure of breathing DIA and HIA through a two-way non-rebreathing valve, at equal inspired air temperatures, was assessed in normal persons during prolonged submaximal laboratory-based exercise.

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METHODS

Subjects
The protocol was approved by the Committee for Research on Human Subjects of the University of the Witwatersrand. Subjects with a history of health problems; pulmonary disease, asthma, smokers and current use of any medication, were excluded from the study. After initial spirometry instruction, subjects failing to demonstrate reproducible flow-volume loops were excluded. Six volunteer consenting healthy sedentary male subjects aged between twenty and twenty-two years, 175,1 ± 3,2 cm in height and weighing 69,4 ± 4,7 kg participated in the study after written informed consent was obtained.

Procedure
Subjects underwent spirometry (Schiller SP 200, Switzerland), in accordance with the American Thoracic Society guidelines[7] after each of four consecutive ten minute bicycling periods (Medifette 400L ergometer, Netherlands) at 75%

of maximal predicted heart rate ((220 minus age) beats/min). There was a three minute rest period in between each ten minutes of exercise followed by three consecutive spirometry recordings performed at rest each made one minute apart, on room air. Measurements were taken prior to the first exercise period (baseline), three minutes after each ten minute exercise period (phases I-IV) and ten minutes post the fourth exercise period (recovery) whilst breathing oral humid inspired air (HIA), or oral dry inspired air (DIA) one week thereafter at the same exercise workload. Mean expired air water vapour pressure (EWVP) was measured over each ten minute exercise period.

Apparatus

Compressed air (Afrox Medical Air, South Africa) was bubbled through two in-series triple-distilled water cylinders at room temperature to two reservoir anaesthetic bags, from which the subjects inhaled air at 85% RH or at 0% RH by circumventing the water cylinders (Fig. 1). Air flow was regulated to be close to the subjects' minute ventilation at all times. The subjects inspired and expired through a two-way non-breathing T-shape valve (Hans Rudolf 2700, MO, USA), the expiratory circuit of which was heated to between 38°C and 42°C to prevent condensation on the humidity and temperature sensor inserted therein (Solomat MPM 500E, Devon, UK). The humidity sensor was calibrated using 2% RH and 75% RH saturated salt solutions, and readings were averaged over each ten minute exercise session from which mean EWVP in kPa was calculated using psychrometric charts. Inspired air temperature was measured using a thermocouple inserted into the mouthpiece of the subject and attached to an electronic ice-point reference (Physitemp Bat-12, NJ, USA) plus calibrated analogue recorder (Hellige Servomed, Freiburg, Germany), and averaged every thirty seconds during exercise. Standard bipolar electrocardiographic measurements of heart rate were recorded.

Statistical Analysis

All data are expressed as absolute sample mean ± SEM, or the absolute change (Δ) in sample mean ± SEM at any phase compared to baseline values for spirometry (Δphases I-IV) and compared to phase I values for heart rate and EWVP (Δphases II-IV). The highest of the three spirometry measurements' values at each phase was used for analysis. Data within the HIA and DIA groups were analyzed by repeated measures ANOVA followed by Student's paired t test and Student-Newman-Keuls post-test correction for multiple comparisons,[8] and between groups using Student's paired t test. Two tailed probability values of $p < 0.05$ were considered significant.

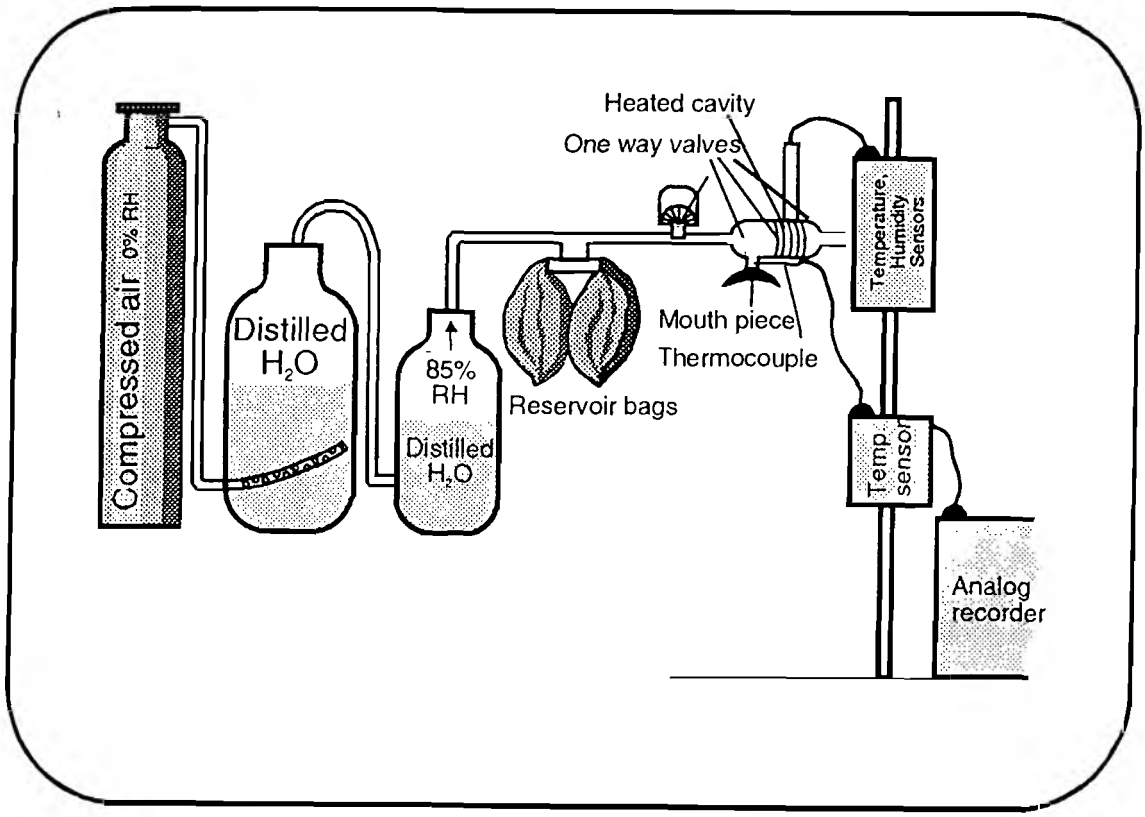

Figure 1. *Schematic representation of apparatus for supply of 0% and 85% RH inspired air. RH = relative humidity.*

Results

Room air temperature was 22-23 °C and room %RH 48,9 ± 2,8 in the vicinity of the subjects. Mean inspired air temperature was recorded as 26,7 ± 0,4°C and 27,1 ± 0,03°C for the HIA and DIA groups respectively. The subjects' submaximal workloads ranged from 100-130 watts and heart rates between the two groups were not significantly different for any of Δphases II-IV.

All spirometry data at baseline were not significantly different between the two groups (Table I). Peak expiratory flow rate (PEFR), forced vital capacity (FVC) and FEV_1 were not significantly different between the groups for any Δphase I-IV values. There were no significant differences between the groups for any Δphase I-IV values of forced expiratory volume in 0,5 second ($FEV_{0.5}$), average flow rate between 0,2 litres and 1,2 litres ($FEF_{0.2-1.2}$), 25% and 75% ($FEF_{25-75\%}$), and 75% and 85% ($FEF_{75-85\%}$) of forced expired vital capacity, maximum expiratory flow rate at 75% ($MEF_{75\%}$), 50% ($MEF_{50\%}$) and 25% ($MEF_{25\%}$) of forced vital capacity. Peak inspiratory flow rate recovery minus baseline value (Δrecovery) was significantly more negative ($p<0,05$) on DIA than on HIA (-0.45 ± 0,27 vs +1.01 ± 0.64 ℓ/min) but there were no differences in any of the Δphase I-IV values.

EWVP absolute values at phases I-IV were significantly lower ($p<0,005$) for DIA compared to HIA. The EWVP phase IV absolute value and the Δphase IV value within group DIA (3,92 ± 0,01 and -0,19 ± 0,11 kPa respectively) were significantly decreased ($p<0,05$) compared with the absolute phase II and the Δphase II values (4,16 ± 0,10 and +0,04 ± 0,06 kPa respectively), however there were no significant differences between the groups for any Δphase II-IV values. There was no significant change in the absolute or Δphase II-IV EWVP values within the HIA group with exercise.

Discussion

In this study we have shown that, despite the laryngo-pharyngeal discomfort experienced by many subjects from the oral breathing of air through a two-way non-rebreathing valve during laboratory-based exercise, indices of pulmonary function do not change following alterations in inspired air humidity at constant inspired air temperature. No significant difference in heart rate was observed between the two inspired humidity groups at constant work loads. Inspired air humidity therefore had no effect on heart rate, and this outcome correlates with the finding that certain changes in the responses to exercise are related to inspired air temperature rather than to inspired air humidity.[1]

Decreased FEV_1 in asthmatic patients during inspiration of cold dry air has been documented,[2,3] whilst changes in inspired air temperature alone are noted to alter pulmonary function in patients with exercise-induced asthma (EIA).[4] However, it has been suggested that airway water loss itself, and not the changing temperature of inspired air, is important in exacerbating EIA.[4,9,10] Under ideal conditions of inspired humidity and temperature there is a certain degree of bronchodilation secondary to exercise,[11] however bronchoconstriction has been shown to predominate under conditions of decreased inspired air water content,[1] whilst the bronchospasm precipitated by altered inspired air temperature in EIA has been noted to be reduced by the addition of water vapour to the inspired air.[4]

The exact mechanism by which a decreased inspired water vapour content may affect pulmonary function is unclear. Experiments designed to demonstrate an impairment of pulmonary function in normal persons by nasal obstruction have produced inconsistent results, and interpretation is further complicated by the dissimilar variables

Table 1.
SPIROMETRY BEFORE AND AFTER SUBMAXIMAL EXERCISE ON HUMID AND DRY INSPIRED AIR

Variable	BASELINE		ΔPHASE IV	
	HIA	DIA	HIA	DIA
PEFR (ℓ/min)	9,55 ± 0,40	9,69 ± 0,49	+0,57 ± 0,34	-0,07 ± 0,07
FEV_1 (ℓ)	4,84 ± 0,21	4,69 ± 0,20	+0,01 ± 0,08	-0,07 ± 0,04
FVC (ℓ)	5,26 ± 0,29	5,10 ± 0,23	+0,02 ± 0,06	-0,13 ± 0,11
$FEF_{25-75\%}$ (ℓ/min)	5,74 ± 0,38	5,59 ± 0,32	-0,21 ± 0,26	+0,12 ± 0,18
$MEF_{50\%}$ (ℓ/min)	6,28 ± 0,47	6,15 ± 0,51	-0,41 ± 0,43	+0,03 ± 0,28
PIFR (ℓ/min)	8,73 ± 0,78	9,87 ± 0,55	+1,21 ± 0,87	-0,37 ± 0,32

BASELINE = at rest before exercise; ΔPHASE IV = absolute change from baseline at phase IV measurements; HIA = humid inspired air; DIA = dry inspired air; PEFR = peak expiratory flow rate; FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity; $FEF_{25-75\%}$ = average flow rate of expired air between 25 and 75% of FVC; $MEF_{50\%}$ = maximum expiratory flow rate at 50% of FVC; PIFR = peak inspiratory flow rate.

measured by different researchers.[12-15] In assessing the role of nasal humidification of dry air in healthy persons at rest, a decreased dynamic lung compliance was recorded while orally breathing dry air compared with humidified air[15] (the inspired air temperature was not stated in this study), however no change in spirometry or functional residual capacity was noted. The researchers concerned suggested that during dry air breathing increased evaporation from the lungs could increase the concentration of surfactant thereby decreasing the lung compliance, and that DIA could be a stimulus for bronchoconstriction via desiccation and cooling of the smaller airways.

The consistently lower EWVP phases I-IV absolute values on DIA compared with HIA, in the absence of a significant difference in the EWVP Δphases II-IV values between the groups, was related to the DIA supply present in the apparatus dead space of the T-shape two-way valve, which effect has also been noted by other researchers.[9] Although there was no significant difference between the groups' EWVP Δphase II-IV values, the trend towards a decreased expiratory vapour pressure within the DIA group over time, and the late significant EWVP decrease within the group DIA (DIA absolute phase IV and Δphase IV versus DIA absolute phase II and Δphase II) may be explained by the airway luminal surfaces dehydrating due to oral DIA breathing,[16] the dehydration being exacerbated by the increased minute ventilation of exercise in our study, thereby decreasing the humidity of the airway dead space air. The role of the humidification of dead space air by the non-nasal airways has been shown to be limited,[15] inspired air being largely humidified by the nasal passages, in the absence of which pulmonary tract dehydration ensues resulting in a limited ability of the non-nasal respiratory airways to humidify expired air. Decreased mean expiratory %RH from breathing hot dry air in exercise has also been attributed to a change in the secretion of water by the mouth, pharynx and upper airway epithelium.[5] Decreased EWVP when exercising on DIA may in addition be due to the reabsorption of water by the dehydrated respiratory tract and mouth from the expired humidified alveolar air.

In summary, this study shows that, in normal adults exercising submaximally under laboratory-based conditions, decreasing inspired air humidity without concomitantly altering inspired air temperature does not significantly affect respiratory function with respect to spirometry or the humidification of dry inspired air.

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