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Assessing the efficacy of a specific physiotherapy intervention for the prevention of low back pain in female adolescent rowers: A field study

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Opportunities and Challenges

As we head into a new decade, sports and exercise medicine is at an interesting point. Since I graduated in 1980 our discipline has undergone immense change. I will outline some of the challenges below and the opportunities they represent.

Firstly, in the last 20 years there has been a major development in the professionalism of sport within this country. Athletes who are professionals have demanded a higher level of professional expertise from their clinicians and, in turn, the clinicians have developed the expertise to manage injuries and medical problems to a much better standard. When I was completing my Diploma course in London in the mid 1980s, ACL injuries fell into the rule of thirds: one-third of athletes could be expected to return to their previous level of sport, a further third return to a lesser level and the final third would have to give up sport completely. I am pleased to report that with developments in surgical technique and post-operative rehabilitation, over 80% of athletes with an ACL rupture in this decade can now expect to return to their previous level of competition. This is impressive and mirrors the progress made in management of other important conditions, e.g. shoulder instability, achilles tendinopathy and most lumbar spinal problems.

Injury management has been enhanced with ACC funding and this has enabled the proliferation of sports and exercise medicine and musculoskeletal practitioners in many centres throughout the country. These clinicians have in turn developed greater expertise in managing a variety of problems and developed patient education skills which could be further enhanced. The challenge is for us to continue to develop home based exercise regimes, particularly in an era where there will be continuing constraints on funding.

An additional challenge over the last few decades has been that of rising healthcare costs. When I graduated there was a significant issue with things that were not technically possible; now the major challenge is what is financially affordable. This is an issue which successive governments have failed to address and funding still appears to be applied in a relatively ad hoc fashion with response to political pressure, e.g. the Herceptin debate. Fortunately there has been some incisive analysis of the situation by Gareth Morgan, a respected economist, and I will comment on his work in a later editorial. The challenge for us, as clinicians, is to practice in a cost-effective manner and educate our patients on an individual basis where appropriate.

A further challenge to clinicians is the rising burden of arthritis, both degenerative, posttraumatic and of the inflammatory variety. The development of progressively safer and more effective NSAIDs, and more recently cox-2 agents, has provided clinicians with a better than ever range of options for our patients. Phenylbutazone, the first widely available anti-inflammatory tablet, is now only a distant memory for retired clinicians - today's patients don't know how lucky they are. The challenge for today's clinicians is to provide appropriate advice for those with early degenerative joint disease so as to maintain the lifespan of their native joints as far as is practicable, whilst still enabling them to maintain an active lifestyle.

A further challenge for us is the increasing burden of chronic disease which results from inactivity. This, coupled with what has been described as an obesogenic environment which discourages habitual physical activity, provides an opportunity for healthcare providers to get involved with some innovative solutions. Patients can be encouraged to walk the last few hundred metres to and from their car, to consider cycling to or from work, or to take a longer lunch hour and go for a walk, run, bike ride or swim.

These are but some of the challenges and opportunities open to sports medicine clinicians. Fortunately, we have a cadre of well-trained doctors, physiotherapists, podiatrists, chiropractors, osteopaths, nutritionists, psychologists and exercise scientists who can provide appropriate professional advice for all those who wish to be active, whether they are an elite athlete or weekend warrior. Long may this situation continue.

Chris Milne
Editor

January

The January 2009 issue of BJSM focused on 'exercise as medicine'. The editorial for this themed issue was written by Dr Bob Sallis, and he states that 'physical inactivity has become the greatest public health problem of our time, and finding a way to get patients more active is absolutely critical to improving health and longevity in the 21st Century'.

In the same article, Professor John Campbell from Dunedin School of Medicine lays out his recommendations for what he terms 'assertive screening'. He is opposed to unnecessary safety screening and detailed questionnaires, and I would be in agreement with this philosophy. Years ago one of our forebears intoned that it was more important to obtain a 'medical clearance' if one wanted to be slothful than to be physically active. It's good to see that such recommendations live on in 2009.

February

In the February 2009 issue, the focus was again on exercise and health. The cover had a caption – The exercise pill – time to prescribe it. The editorial is headed up 'Limiting our daily sitting/lying to just 23.5 hours – too ambitious?' This provocative title is a clever way of making the point that sedentary behaviour itself is an independent risk factor for chronic disease. Later in the same issue, Professor Tim Church and Dr Steven Blair make a strong case for treating physical activity as a legitimate medical therapy, even though it does not come in a pill. They make the point that whilst creating a pill that would have the many positive benefits of exercise is near impossible, creating a more physically active environment and society is a very achievable goal. There is a need to refine the exercise prescription for various groups across the lifespan.

In the same issue, Lobelo and colleagues comment that the physical activity habits of doctors and medical students influence their counselling practices. In other words, you are more likely to receive appropriate exercise advice from a doctor who is physically active than a couch potato – sounds pretty plausible to me! Likewise, physiotherapists and exercise psychologists can help promote physical activity, and two further articles in the same issue attest to that.

March

The March issue concentrated on groin and knee problems. There was a major article by Bill Vincenzino and colleagues highlighting the role of foot orthoses and physiotherapy in the management of patellofemoral pain. Later in the same issue, Falvey and colleagues introduce the concept of the groin triangle to try and improve the diagnosis of groin problems. They describe groin, gluteal and greater trochanter triangles to help the clinician focus on likely structures where symptoms may be arising from. There is more on this in the June issue, where there are some useful tables listing gluteal problems in their order of frequency in the athletic population.

April

The April issue focused on tendinopathies. Karim Khan was co-author of an article entitled 'Mechanotherapy: how physical therapists' prescription of exercise promotes tissue repair'. The article describes the process by which physical loading causes a perturbation to tendon cells which then communicate with one another via small charged particles and lay down new tissue. The article is very well illustrated. Bill Vincenzino and colleagues describe a new integrative model for lateral epicondylitis. They identify tendon pathology, pain system changes (what some clinicians call 'wind up') and motor system impairments as important contributors to tennis elbow.

Jeremy Lewis, a senior research physiotherapist in the UK, takes a relatively nihilistic approach to current clinical testing for rotator cuff tendinopathy and subacromial impingement. He describes clinical techniques such as repositioning the humeral head with seatbelt strapping, or neck retraction which can reduce the patient's symptoms in the clinic and be used as an adjunct to management.

May

The May issue addressed the theme of ACL injuries; Quatman and Hewett argued that 'valgus collapse' is a sex-specific mechanism of ACL injury in females, whereas a sagittal plane oriented loading mechanism appears to predominate in men. As a sideline netball observer, it still seems to me that overload of the ACL in the sagittal plane is still a fairly big contributor to ACL injuries in women in that sport. The ACL reconstruction options can be broadly divided into patella tendon or hamstring grafts. Each has their advocates, and in this issue Merv Cross argues for the patella tendon and Leo Pinczewski for the hamstring graft. Surgical techniques have been refined steadily over the past decade, so early studies need to be interpreted in that light. My reading of these papers is that hamstring grafts are probably preferred for those who need to do a lot of kneeling, but both procedures are equally effective at reducing episodes of instability.

There was a supplement to the May issue dealing with concussion in sport. The most important paper to be aware of is the Consensus Statement issued after the 3rd International Conference on Concussion held in Zurich in November 2008. The SCAT card (Sport Concussion Assessment Tool) has been modified since the previous version, and now incorporates specific tests of balance and coordination that replace the 'pronator drift' assessment of the previous version. Coordination and balance are known to be impaired after concussion, so it is important for clinicians to become familiar with these tests.

June

The June issue contained a useful article by Martin Schwellnus on exercise associated muscle cramps. Hitherto, cramps have been thought to be due to dehydration and electrolyte depletion. This article discusses this hypothesis, along with the alternative view that these cramps are due to fatigue and altered neuromuscular control. This second hypothesis is favoured by Schwellnus in view of the superior quality of evidence available, and I tend to agree with him.

Later in the same issue, Jill Cook describes a pathology model to explain the varied presentation of load-induced tendinopathy. Her model proposes that tendons go through three phases of injury – reactive tendinopathy (which recovers promptly), tendon dysrepair and finally degenerative tendinopathy (which is very slow to recover). She supplies good evidence from clinical, imaging and histopathology studies and then tailors the treatment to the stage of tendinopathy.

Finally in this issue there is an excellent short summary of cervical disc prolapse using a case example and expert commentary.

My pick for MVA (most valuable article) for the six months would be that on the Zurich consensus guidelines for consultation management in the May supplement. Every clinician who sees concussed athletes (and that's most readers of this journal) needs to be familiar with the updated SCAT card.

July

The July issue focused on the general acceptance that physical inactivity is the biggest public health problem of the 21st Century. Karim Khan, in his editorial, made mention of the fact that it is now 10 years since Dr Frank Booth and colleagues wrote in the Journal of Applied Physiology that we should declare war against physical inactivity. The American College of Sports Medicine is promoting a new initiative entitled Exercise is Medicine.

The big news in the July issue is an article by Byberg and colleagues who studied 2,205 men in Uppsala, Sweden from the 1970s until relatively recent years. They found that increased activity in middle life increases longevity, even if those men only take up exercise in their 50s. Effectively, this means that middle aged men can obtain a new lease of life with just 10 years of physical activity. This follow up has been carried out in the typical detailed Scandinavian fashion over 35 years. One might suppose that these same benefits would accrue to women but this has yet to be proven.

A second article of widespread interest in this issue is a systematic review of four injection therapies for lateral epicondylosis; these included prolotherapy, Polidocanol, whole blood and platelet rich plasma. The authors concluded that there is strong pilot level evidence supporting the use of all four of these interventions but at present we do not have any large definitive trials. This is an area of intense research and I suspect we will have a lot more information to offer in a few years' time.

August

The August issue discussed the very important issue of prophylactic use of NSAID by athletes. It is well-known that athletes will often take NSAID before exercise. This article supports the use of NSAID in the presence of inflammatory symptoms and signs (active swelling, and resting and/or night pain). In the absence of previous NSAID side effects or risk factors then they can be used alone. But if, for example, dyspepsia has been noted, NSAID should be combined with a protective agent or an alternative agent, e.g. paracetamol, should be used. The article tends to gloss over the important issue of prostaglandin inhibition abolishing some of the protective mechanisms of the kidney. Therefore any athlete taking NSAID, particularly for prolonged activity, needs to be especially rigorous with rehydration, as a depleted blood volume is known to be associated with an increased risk of renal shutdown.

In the same issue was a review of rugby league injuries in New Zealand based on eight years of ACC data. King and co-authors showed that the knee was the most common injury site, and their study also showed that the injury rate and average age of injured rugby league players both increased over time. Their data were consistent with other studies of rugby league injuries.

A UK based group studied 73 athletes of both genders, 95% male, who underwent an inguinal release procedure for groin pain. On average, athletes were able to return to light training at one week, full training at three weeks and competition at four weeks; 73% reported complete absence of symptoms and 97% thought the operation had improved their symptoms. These are very impressive data and probably reflect a highly selected group as most studies do not have quite this high success rate.

September

The September issue marked the inauguration of a new series entitled A to Z of Nutritional Supplements. Put together by Louise Burke and a couple of other respected nutritionists, this series will systematically evaluate the issues regarding supplementation on an individual supplement basis. It goes right from alpha-ketoglutarate to zinc. Follow it closely over the next few issues if you have an interest in this area.

There was also an article from the University of Melbourne group on hip flexibility and strength measures and, in particular, their reliability and association with athletic groin pain. These authors found that only the squeeze test discriminated between football players with and without groin pain.

The September supplement concentrated on injury prevention and health protection. In particular, there was a 13 page IOC consensus statement on the periodic health evaluation of elite athletes that had been promulgated in March 2009. This replaces the previously recommended pre-participation examination and Martin Schwellnus spoke to this issue at the Sports Medicine Conference in Rotorua last year. All those caring for elite athletes, particularly team doctors for our National and other professional teams, should be familiar with the recommendations in this article. I will attempt to provide a summary in a later issue of this journal.

There are useful articles on prevention of sudden cardiac death. This issue was brought to prominence by the death of Marc-Vivien Foe during the FIFA Confederations Cup in France in 2003 when CPR was correctly performed on the sideline but, despite that, he died. Since then FIFA have made it mandatory to equip all stadia with automated external defibrillators (AEDs) and also made it compulsory for emergency medical practitioners to be present at all FIFA international tournaments. These procedures were in place during the FIFA U17 Women's World Cup in New Zealand in 2008.

There are significant issues in interpretation of ECGs in athletes and several articles discuss this issue. The most useful is by Corrado and colleagues and this is entitled 12-Lead ECG in the Athlete: physiological versus pathological abnormalities. Common training related ECG changes include sinus bradycardia, first-degree AV block, incomplete right bundle branch block, early repolarisation and, finally, isolated QRS voltage criteria for left ventricular hypertrophy. A more sensitive measure of left ventricular hypertrophy is an echocardiogram.

October

The October issue contained a very useful pictorial essay on interpretation of MRI scans in athletes with suspect ACL injuries. Compiled by the senior radiologists from the University of British Columbia group, this article is an excellent summary for clinicians and all of us who have to look at MRI scans could benefit from reading through it.

There is also a useful article on central aponeurosis tears of the rectus femoris from a Spanish group. These authors found that the average time to return to competition is 45 days with a 4cm tear; for each 1cm increase in the length of tear one should add another five days onto the recovery period. These data are similar to those produced by Tom Cross in Australia, although his athletes seemed to recover in half the time of the Spanish athletes studied here.

Bruce Hamilton and myself collaborated with John Orchard and Justin Paolini in putting together some guidelines for practical but sensible use of NSAID in sports. The evidence suggests that NSAID are useful in treating inflammatory problems such as tenosynovitis and impingement, but not in tendinopathy. They are contraindicated in the early phase of fracture healing.

November

In the November issue there was a very useful article on the beneficial effects of exercise in overweight men and women with an average BMI of 31.8. These authors shifted the focus from bodyweight to other markers of health and found that meaningful health benefits could be achieved, even in the presence of lower than expected weight loss following an exercise programme. They recommend, from a public health perspective, to encourage exercise and pay less attention to weight loss. I would support this.

The regular series on A to Z of Nutritional Supplements featured antioxidants and arnica in this issue. A recent Cochrane Review found little or no difference in the mortality between antioxidant administration and those taking a placebo. In fact, mortality was slightly higher in the antioxidant group. The authors conclude that at present there is limited scientific evidence to recommend antioxidant supplements to athletes or other physically active individuals and, in fact, they urge caution when considering supplementation with high doses of antioxidants. They have a similar sanguine view of the use of arnica. There are numerous anecdotal reports of its benefit but the results of trials are contradictory, and two independent systematic reviews concluded that the effectiveness of homeopathic arnica is unproven.

December

In the December issue there was a useful update on the status of sports and exercise medicine as a specialty in Europe and also in the UK. Michael Cullen, a senior consultant in Belfast, describes the UK scene where there is a significant emphasis on the role of exercise as a driver for health and in the management of chronic disease. From my time in the UK, my impression is that citizens of that country are probably less inclined to exercise than their counterparts in New Zealand and Australia, but the focus on exercise as medicine is appropriate for all Western countries.

Later in the same issue there is an article by Fabio Pigozzi in Rome summarising the status of sports medicine specialisation in various European countries. This is a useful reference.

Also in this issue of the journal there is an article on asthma in elite athletes. A Danish group found that symptoms of asthma in elite athletes are not necessarily associated with classic features of asthma and should not be relied upon to make a diagnosis; they recommend the use of objective measurements, e.g. spirometry and provocation testing. However, it is well-known that these tests do not pick up all affected athletes either. The area is one of considerable controversy and emphasises the point that the most accurate assessment is based on clinical features plus the results of investigations.

The December supplement focused on the Vancouver Winter Olympics coming up in February. An introductory article discussed the importance of sports medicine for the Vancouver Olympic Games. There were articles on injuries in alpine skiers that confirm much of the early data presented to us years ago by Mike Lamont.

Roald Bahr, who will be known to all of those who attended the 2007 Sports Medicine New Zealand Conference, has a useful article on the methodology for reporting overuse symptoms in sports. He recommends prospective studies with continuous serial measurements of symptoms. These should be combined with valid and sensitive scoring instruments and prevalence, not incidence, should be used to report injury risk. He recommends assessment of severity based on functional level and not time lost from sport.

The most provocative article is that by John Orchard entitled On the Value of Team Medical Staff: can the “Moneyball” approach be applied to injuries in professional football. John asks the question, how important is the performance of the medical staff to professional sporting teams’ success? He evaluates the literature, particularly with regard to the management strategy behind the Oakland Athletics basketball team for five years from the late 1990s. During that time the Oakland team was one of the most successful in major league basketball, despite having one of the lowest payrolls. Analysis of the data showed that Oakland’s philosophy of buying players that were undervalued statistically and selling players that were overvalued enabled them to punch above their weight, i.e. be more successful than one would have anticipated. John goes on to look at what he terms player durability (availability through not being injured). He found that this may be under-recognised as an important factor in team success. Leading on from this, he has emphasised the importance of the medical staff in improving availability of players. Further, he states that medical staff may be currently undervalued by football team management. From my knowledge of various sporting codes in New Zealand, I would say “hear hear”. Partly this is because we do sports medicine because we love sport and the contact with athletes, however, over time we have often allowed our services to be undervalued, and sports administrators are quite happy to pay the minimum amount they can get away with. Overall, a very good read.

My pick for MVA (Most Valuable Article) in the six month period under study would that by John Orchard. I believe it raises important questions and he has looked at the issues surrounding our professionalism from a fresh viewpoint. Read this and see what you think.

Chris Milne
Sports Physician
Hamilton

ARTICLE

Assessing the efficacy of a specific physiotherapy intervention for the prevention of low back pain in female adolescent rowers: A field study

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ABSTRACT

Objectives: **To determine the efficacy of a specific physiotherapy intervention administered to adolescent female rowers with the aim to decrease the prevalence of low back pain (LBP) and associated levels of pain and disability.**
Design: A non randomized controlled field study in adolescent female rowers with and without LBP.
Setting: Curtin University of Technology, Western Australia and the participating private school boatshed, Perth, Western Australia.
Participants: **Participants were 82 adolescent female rowers, with and without LBP. These participants attended the same school and were aged between 13 – 17 years [experimental group 13.9(0.9) years, control group 13.8(1.0) years]**
Outcome Measures: **Primary outcome measures in this study included; LBP point prevalence, pain intensity (utilizing a visual analogue scale) and disability level (utilizing a modified Oswestry questionnaire). These measures were taken at four time points over the rowing season.**
Results: **The experimental group demonstrated a significant reduction in the prevalence of LBP across the rowing season. [48% to 19% pre-season to mid-season and from 48% to 24% pre-season to end-season]. The prevalence**

of LBP in the control group slightly increased [22% to 25% pre-season to mid- season and was unchanged at 22% pre-season to end-season]. A significant increase in the proportion of subjects pain-free was shown in the experimental group at mid-season compared with pre-season (p=0.007), but no change thereafter (p>0.05). In the control group the proportion pain-free remained relatively stable across the four time points (p>0.2 for changes between consecutive times). The experimental group rowers demonstrated reduced pain intensity over the course of the rowing season (p=<0.05) compared to the control group [mean pre-end season exp 6.4(21.0), control -2.7(17.6) Z= -2.283, p = 0.022].Levels of disability did not differ between the groups across the rowing season.
Conclusions: **A specific physiotherapy exercise intervention was effective in reducing the prevalence of LBP in a population of adolescent female rowers and reducing pain intensity levels in subjects who complained of LBP at the commencement of the rowing season. A randomized trial to test the intervention under more rigorous scientific conditions is recommended.**
Key Words: **low back pain, rowers, exercise intervention, female, adolescent**

INTRODUCTION

Low back pain (LBP) in adolescence has been shown to be a significant risk factor for LBP in adulthood, with the strongest predictor of future LBP being a prior history of LBP (Hestbaek et al, 2006). High prevalence rates of LBP (24%) among children and adolescents have been demonstrated in several studies (Balague, 1999, 2003; Hestbaek et al, 2006); with the highest prevalence amongst girls than boys (Balague et al, 1999).

Participants in particular sports such as rowing have shown a greater disposition to reporting LBP (Burnett et al, 2008). A recent study of adolescent female rowers demonstrated a LBP point prevalence rate of 47.5% as compared to 15.5% in equally active non-rowers (Perich et al, 2006). These findings are supported by other research involving rowers of all ages and abilities (Howell, 1984; Perich et al, 2006; Stutchfield & Coleman, 2006).

Research has shown that *sports specific risk factors* associated with rowing, such as repeated lumbar flexion and loading often with the addition of spinal rotation and high levels of training times and volumes can potentially increase LBP prevalence (Adams & Dolan, 1995; Reid & McNair, 2000; McGregor et al, 2007). The lumbar spine may also be predisposed to strain as a result of *individual risk factors* of the rower, such as: habitual sitting postures (Perich et al, 2006), deficits in lumbo-pelvic motor control (McGregor et al, 2002; Caldwell et al, 2003), limitations in anterior pelvic tilt (Reid and McNair, 2000; Caldwell et al, 2003; McGregor et al, 2007), deficits in back muscle and lower limb endurance (Roy et al, 1990; Perich et al, 2009) and a lack of flexibility in the hamstrings (Gajdosik, 1994; Reid and McNair, 2000) have all been reported in the literature as causative factors for LBP prevalence in rowers.

There are limited studies demonstrating the efficacy of *specific exercise interventions* for prevention of LBP in athletic populations. Generic programs with mixed outcomes have been reported such as: general core strengthening exercise programs in rugby players (Cusi et al, 2001) and in collegiate athletes (Nadler et al,2002); trunk endurance exercises in college rowers (Tse et al, 2005); hamstring strengthening in female rowers (Koutedakis et al, 1997); muscle-specific exercises aiming at segmental control in young female gymnasts with and without LBP (Harringe et al, 2007). These studies had mixed outcomes and failed to demonstrate efficacy.

To date, only one study has tested an individually applied, specific physiotherapy exercise intervention as part of a multi-dimensional intervention in a rowing population. Perich et al (2009) demonstrated a significant lower prevalence of LBP in the intervention group (26%) as compared to the control group (46%). However, the education, physical conditioning, coaching or rowing/ergometer sessions were not controlled for, making it unclear as to the specific effect of the exercise intervention itself, in reducing LBP prevalence. Therefore, the aim of the current study was to determine the efficacy of the specific physiotherapy exercise intervention undertaken in the previous study (Perich et al, 2009) within a field study, whilst controlling for the other components of the multi-dimensional intervention program.

METHODS

Ethical Approval

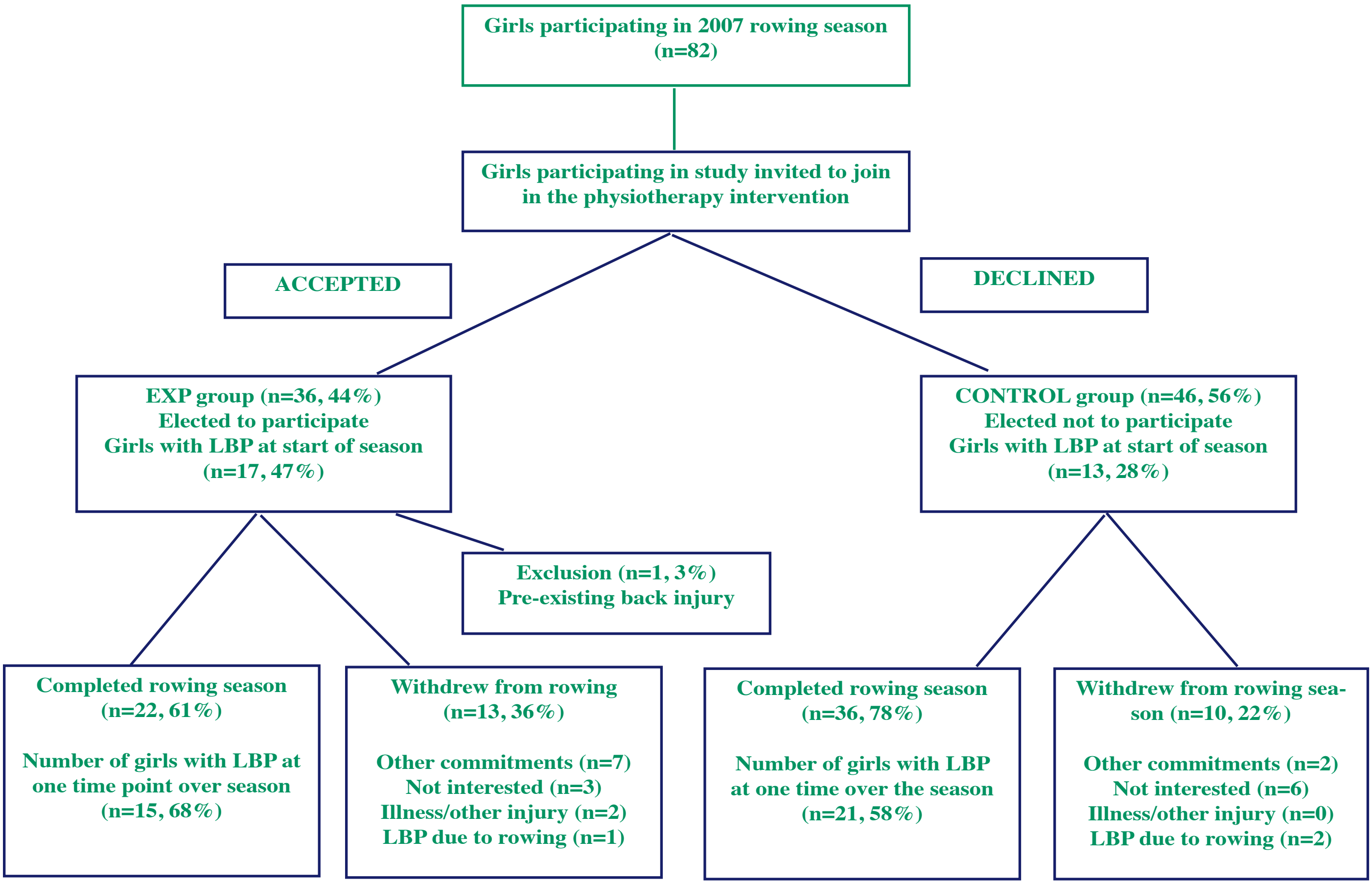
Ethical approval was granted by the Human Research Ethics Committee at Curtin University of Technology, Perth, Western Australia.

Subjects

There were 82 girls with and without LBP, aged 13-17 years, from a private school in Perth, Western Australia participating in the Independent Girls Schools Sports Association 2007 rowing season.

A non-randomised controlled field study design was chosen as it best replicates real life clinical practice. All subjects (n=82) were offered the opportunity to participate in the physiotherapy intervention, with the fees associated being standard physiotherapy consultation charges. Subjects who indicated a willingness to undergo the intervention (n=36) were assigned to the experimental group while those who did not were assigned to the control group (n=46). Subjects and their parents were provided with an information sheet outlining the purpose of the study and an informed consent was completed prior to participation in the study. Subjects were excluded from the study if they had pre-existing LBP due to previous spinal trauma unrelated to rowing, or diagnosed specific lumbar spine disorders such as spondylolisthesis, inflammatory disorders or neurologic conditions (n=1). The subjects who withdrew from each group throughout the rowing season were requested to fill in a questionnaire outlining the reason for their withdrawal. These were excluded from further analysis (exp n=13, control n=10).

FIGURE 1: The flow of subjects through this non-randomised controlled trial outlining withdrawals, reasons for withdrawal and the proportion of subjects with LBP in each group.



Intervention

The format for the study was:

i education session (experimental and control groups)

ii physiotherapy exercise intervention program (experimental group)

iii physical conditioning program (experimental and control groups)

Education Session

The education session was conducted by the same experienced physiotherapist involved with the musculoskeletal screening in the previous study (Perich et al, 2009). The session was of 1 hour duration and all subjects from both groups, their parents, physical education staff and rowing coaches were requested to attend. This session covered concepts such as: basic spinal mechanics, injury risk in rowing, potential LBP mechanisms in rowing, spinal posture education (while sitting, rowing and lifting), attitudes and coping strategies with regards to the management of LBP. This included seeking prompt advice, informing parents and coaches of the presence of LBP and the use of early symptomatic relief.

Physiotherapy Intervention Program

Subjects in the experimental group were then required to undergo a musculoskeletal screening by an experienced physiotherapist. A team of 8 musculoskeletal and sports physiotherapists who were experienced in the assessment and exercise prescription protocol participated in the physiotherapy intervention. Initially, an *interview* was performed to assess current and previous history of LBP, pain location, aggravating and easing factors for LBP, as well as treatment history, attitudes towards LBP, current levels of rowing training and general activity. Subsequently, a musculoskeletal *physical examination* was performed examining spinal range of movement, directional pain provocation, habitual spinal postures in sitting and standing and spinal repositioning sense in sitting. Lumbo-pelvic motor control was assessed by the ability to maintain a neutral lumbar spine with a relaxed thorax in sitting, whilst performing active hip flexion and knee extension, forward reach in sitting, sit to stand, half squat (with 90 degrees hip and knee position), squat with forward reach and the row position. Rowing technique and the ability to control end range lumbar flexion postures whilst rowing was assessed visually on the rowing ergometer.

An *individually prescribed exercise program* was instituted for subjects with and without LBP, based on the findings identified in the musculoskeletal screening. Each subject received a total of three physiotherapy sessions, with review at one and three weeks following the initial screening to ensure adherence and understanding of the exercise programs and to progress the exercises. There was no other physiotherapy treatment or manual therapy administered. Individual exercises which formed part of the physiotherapy intervention are illustrated in figure 2.

FIGURE 2: Rowing exercises included in the physiotherapy intervention program.

Subjects in the experimental group received an exercise sheet with their individual physiotherapy intervention presented in picture form, with a brief explanation of the exercises. They were requested to undertake the exercises marked by the physiotherapist daily throughout the course of the season. The exercises and repetitions were assessed and progressed at each session to enhance both spinal motor control and lower limb and back muscle endurance. Subjects were asked to fill in a compliance sheet to indicate if the exercises were undertaken, however this data was not collected as there was poor adherence to completing the compliance sheet. In spite of this most girls indicated and demonstrated that they had complied with the program.

Physical Conditioning Program

The physical conditioning program used in this study was the same as that used in the previous study by Perich et al (2009) which was designed to increase lower limb and back muscle endurance, enhance general conditioning and improve postural awareness. The implementation of the program was undertaken by the school's rowing coaches and physical education staff and remained a constant between groups in this study. Activities included in this program consisted of aerobic conditioning, hill running, strength and conditioning circuits and flexibility training. The principal researcher also attended two physical conditioning sessions after the completion of the physiotherapy reviews to reinforce compliance with the intervention program in the experimental group.

Data Collection and Analysis

Anthropometric Measures

Age, height and weight measures were collected to ensure similarities in the trial sample between both groups.

LBP Prevalence and Aggravating Factors

LBP prevalence was determined by asking the question at baseline pre-season "Have you experienced low back pain?" and at mid-season (week 11), end-season (week 23) and post-season (week 33) asking the question "Have you experienced back pain since the commencement of the rowing season?" A questionnaire was completed examining factors that 'bring on' or 'exacerbate pain' in relation to rowing, ergometer training and lifting the boats.

LBP Location

Subjects were requested to fill in a body chart illustrating the area they currently experienced back pain.

LBP Intensity

Subjects were asked to rate their pain on a 10cm visual analogue scale (VAS) with the start point on the left side of the horizontal line being "no pain" and the end point on the right side of the line being the "worst pain imaginable". A percentage score was recorded. This method of measuring pain intensity is both reliable and valid (Jensen et al, 1989; Ogon et al, 1996).

LBP Disability

Subjects were asked to complete the modified Oswestry questionnaire which is a reliable and valid measure of function consisting of nine sections: pain intensity, personal care, lifting, walking, sitting, standing, sleeping, social life and traveling.

(Fairbank & Davies, 1980). Subjects select the statement in each section which is most appropriate to their level of disability and a percentage score was recorded. The section relating to sex life was deleted for this study, however the test still retains its validity (Page et al, 2002).

These questionnaires, body chart and visual analogue scale were collected at the beginning of the rowing season (week 1), mid season (week 11), end of season (week 23) and 10 weeks post season (week 33)

Statistical Analysis

An independent t-test was performed on the baseline data, to assess for group differences with regard to anthropometric data of height, weight and age at entry to the trial.

To assess for group differences with regard to VAS and Oswestry scores at entry to the trial an independent t-test was performed on the baseline data of subjects with LBP in each group.

Point prevalence was compared at four occasions over the rowing season between the control and experimental groups. Prevalence of LBP was defined as pain intensity of a VAS score greater than 1, in order to eliminate low levels of discomfort associated with rowing and muscle fatigue. Logistic regression was used to model the proportions pain-free over the 4-time points for both groups. Within-subject correlation over the times was taken into account for calculation of standard errors and assessment of parameter estimates using a Wald Chi-square test statistic.

A Mann Whitney-U non parametric test was used to assess differences in change scores of pain intensity and disability levels between the two groups, in subjects with LBP, across the time frame (the difference between the baseline pre-season score and the mid-season, end-season and post-season score for each individual) of each measure, to assess differences between the two groups after the intervention period. This test was performed to account for the large number of zero scores for subjects without back pain, resulting in a zero inflation effect.

The level for statistical significance was set at the 95% confidence limit. Statistical analysis was performed using Microsoft Excel and SPSS software version 15.

RESULTS

Baseline Data

There was no significant difference in baseline data illustrated in table 1 with regards to the anthropometric data between groups. Pre-existing LBP prevalence (VAS score greater than 1 out of 10) was recorded at baseline pre-season. Statistical analysis demonstrated no significant difference in baseline levels of pain intensity and disability in subjects with LBP, between groups. Differences in prevalence at baseline were found to be statistically significant and will be discussed further below.

TABLE 1: Baseline measures of anthropometric data, prevalence, pain intensity and disability of all subjects involved in the 2007 school rowing program.

	EXPERIMENTAL (n=36)Mean (SD)	CONTROL (n=46)Mean (SD)
Age	13.9(0.9)	13.8(1.0)
Height	167.4(10.6)	168.1(8.2)
Weight	58.9(8.0)	58.4(9.04)
Prevalence VAS > 1	48%	22%
Pain IntensityMean VAS (mm)	32.5(14.5)(n=10)	25.4(7.7)(n=8)
DisabilityMean Oswestry (out of 100)	5.3(4.6)(n=10)	5.4(4.3)(n=8)

Pain intensity (mean VAS) and disability (mean oswestry) refer to those subjects at baseline who currently experienced LBP.

TABLE 2: Baseline measures of anthropometric data, proportion of withdrawals, pain intensity and disability of subjects who withdrew from the 2007 school rowing program.

	EXP (n=12)Mean (SD)	CONTROL (n=10)Mean (SD)
Age	14.2(1.4)	13.5(0.5)
Height	166.2(6.6)	165.0(6.0)
Weight	55.5(8.4)	51.3(7.4)
Proportion of Subjects who Withdrew	33%	22%
Pain Intensity Mean VAS (mm)	46.1(22.4)	13.9(5.6)
Disability Mean Oswestry (out of 100)	5.6(7.0)	4.0(0)

Withdrawals

Withdrawals in each group were requested to fill in a questionnaire outlining the reason for their withdrawal from the school rowing program (which is cited in figure 1) and were excluded from further analysis in this study. One subject in the experimental group was excluded from the study due to sustaining a traumatic lifting injury to the low back (independent of rowing) prior to the commencement of the 2007 rowing season.

Aggravating Factors and Location of Pain

The self-reported factors related to rowing that caused the initial onset or exacerbated LBP: lifting the boat (exp n=6, control n=3), sweep rowing (exp n=8, control n=4), rowing in quadruple or single scull (exp n=7, control n =1), ergometer rowing (exp n=11, control n=4), long rowing sessions (exp n=6, control n=4) or other issues related to sustained postures or weight training (exp n=4). The location of pain was identified as lumbar (exp n=13), thoracic (exp n=4), buttock (exp n=1). Six subjects also reported shoulder pain.

LBP Prevalence

The experimental group demonstrated a significant reduction in the prevalence of LBP across the rowing season (Fig.2) from 48% to 19% pre-season to mid-season and from 48% to 24% pre-season to end-season while in the control group the prevalence of LBP slightly increased from 22% to 25% pre-season to mid-season and was unchanged at 22% pre-season to end-season. This was in spite of a significantly greater number of subjects in the experimental group having pre-existing LBP.

A between-group comparison of the proportions pain-free for each of the 4 time points found the two groups differed significantly only at baseline pre-season (p=0.05). Modeling within-group changes across the times revealed there was a significant increase in the proportion pain-free in the experimental group at mid-season as compared to pre-season (p=0.007) but no change thereafter (p>0.05), indicating a significant reduction of LBP prevalence in the experimental group after the intervention. Within the control group the proportion pain-free remained relatively stable across the 4 times (p>0.2) for changes between consecutive times.

Changes in Pain Intensity and Disability

Differences in change scores (the difference between the baseline pre-season score and the mid-season, end-season or post-season score) of pain intensity and disability levels between groups across the time frame are illustrated in figures 4 and 5.

FIGURE 3: LBP Point Prevalence VAS>1 (n=58). Point prevalence values were recorded for subjects in both groups who completed the rowing season.

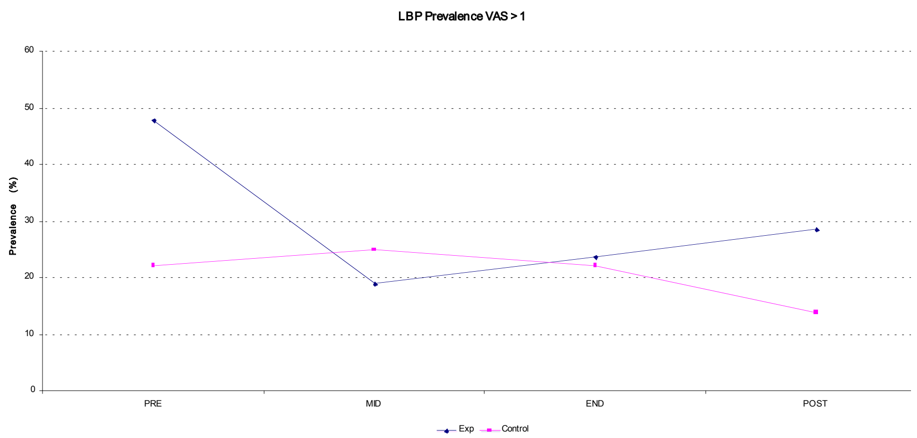


FIGURE 4: Pain intensity change scores for subjects with LBP in each group. The change in VAS score is defined as a positive score for a reduction in pain intensity and defined as a negative score for an increase in pain intensity.

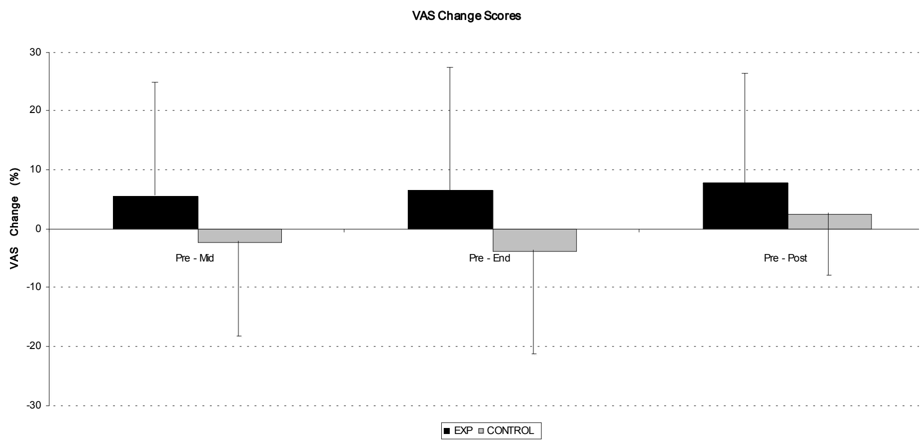
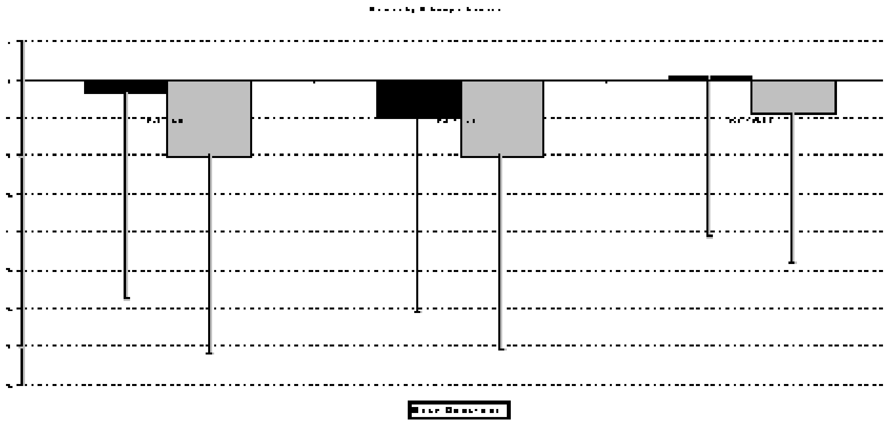


FIGURE 5: Disability level change scores (Oswestry) for subjects with LBP in each group. The change in Oswestry score is defined as a positive score for a reduction in disability level and defined as a negative score for an increase in disability level.



Six subjects in the experimental group and 10 subjects in the control group reported no pain at any stage during the rowing season.

Changes in VAS score across the rowing season was defined as a positive score for a reduction in VAS (decrease in levels of pain) and defined as a negative score for an increase in VAS. There was a significant difference in pain intensity VAS change scores between groups, when comparing end-season score with baseline pre-season score [mean pre-end season exp 6.4(21.0), control -2.7(17.6) Z= -2.283, p = 0.022]. This illustrates a significant reduction in pain intensity over the course of the rowing season for the experimental group who received the physiotherapy intervention.

Changes in Oswestry scores across the rowing season were defined as a positive score indicating reduced levels of disability and defined as a negative score indicating an increase in levels of disability. Statistical analysis of changes in the level of disability (Oswestry) mid-season, end-season and post-season when compared to baseline pre-season did not illustrate significant differences between groups. However, the trend illustrated by the graph in figure 5 suggests a trend for slightly greater levels of disability in the control group across the rowing season as compared to the experimental group.

DISCUSSION

The current study tested the efficacy of the specific physiotherapy intervention in isolation that was a component of the multi-dimensional study previously undertaken in the study by Perich et al (2009). Although it is acknowledged that there are inherent limitations with a 'field study' design, the advantage of this methodology is that it closely reflects 'real life' clinical practice.

LBP Prevalence

The specific physiotherapy intervention resulted in a reduction in the prevalence of LBP across the rowing season in the experimental group as compared to the control group. This change revealed a 29% reduction in LBP prevalence in the experimental group with a 3% increase in the control group across the rowing season. It is acknowledged that the self-selection bias for the physiotherapy intervention may have had the potential to provide a greater chance for the experimental group with a higher pain prevalence and an expectation for treatment to improve their status. In spite of this, whilst controlling for the education session, physical conditioning programs and coaching components of the intervention the findings support that the physiotherapy intervention had a significant impact on the prevalence of LBP in this population. This is in line with previous research by Perich et al (2009), which demonstrated a reduction in prevalence of up to 12% across the rowing season in the experimental group following the multi-dimensional intervention.

In addition to the change in prevalence there was also a reduction in pain intensity in the experimental group although differences were small. In contrast the baseline level of disability was low and remained low and unchanged throughout the rowing season suggesting that the LBP experienced had little impact on activities of daily living as measured by the modified Oswestry questionnaire. This finding may reflect a lack of sensitivity of the questionnaire to disability related to rowing in an adolescent population. It is reported that the Oswestry questionnaire is more sensitive to moderate rather than low levels of disability (Roland & Fairbank, 2000). Conversely it may be that these subjects coped well with their LBP.

Exercise Interventions

Overall knowledge of the impact of specific physiotherapy exercise interventions on the effect on LBP in sporting populations is very limited. As part of clinical physiotherapy practice, exercise prescription by physiotherapists formulates a significant portion of the treatment and management of athletes. The lack of evidence base reported in the literature questions the validity of the many physiotherapy exercise interventions which are prescribed on a daily basis.

Many sporting groups undertake 'trunk strengthening' or 'core stability training' in an attempt to reduce pain prevalence, intensity and disability associated with LBP, however no significant benefits from these interventions have been demonstrated (Nadler et al, 2000; Cusi et al, 2001). Specific muscle control exercises delivered in a group setting as part of weekly training regime were of benefit in reducing pain intensity and functional disability in adolescent gymnasts, however the authors acknowledge a number of methodological limitations in the research design (Harringe et al, 2007).

The findings of the current study adds to the evidence base for specific physiotherapy exercise interventions in sporting groups and is consistent with the previous study in adolescent female rowers (Perich et al, 2009).

Limitations

The present study has a number of methodological limitations which need to be acknowledged. This non-randomised controlled study was conducted as a field based study where the subjects elected to participate in the physiotherapy intervention. The uneven group size was the result of more subjects with LBP self selecting for the physiotherapy intervention, causing a self-selection bias. This may have the potential to provide more chance for the experimental group with a higher pain prevalence to improve pain prevalence itself. Despite this, the significant reduction in LBP prevalence and pain intensity levels at the end of season in the experimental group suggesting that the physiotherapy intervention was effective.

Low numbers in each group who completed the rowing season were due to a high number of withdrawals from each group, with the baseline pain intensity level in the subjects who withdrew, higher in the experimental group. In spite of this, it is interesting to note that LBP was cited as a reason for withdrawing from the rowing program in only 3 subjects (exp group n=1, control group n=2).

The lack of randomization and blinding in this study is also considered to be a limitation. Although it was possible that some of the exercises in the intervention may have been shown to the control group subjects, the fact that the physiotherapy intervention was based on individual screening and an individually prescribed exercise program, it is unlikely subjects in the control group had the understanding and knowledge to adequately perform the physiotherapy intervention as a result of observing subjects in the experimental group do their exercises.

Statistical analysis involved multiple testing of point prevalence, pain intensity and disability level increasing the chance of error. A confidence interval was set at 95%, which was based on the point prevalence findings in the previous study by Perich et al (2009), with the potential for a significant result occurring by chance with p-value set at 0.05.

As standard physiotherapy consultation fees were charged to the subjects receiving the physiotherapy intervention, the potential self selection bias could be based on an economic decision by the family of the subject.

Because of the study design limitations, the strength of these findings is reduced. Questions such as the input and level of support from the physical education and coaching staff within the school and level of compliance amongst subjects in the experimental group have not been answered.

Recommendations

Future studies with a randomized controlled design and long term follow-up using a patient functional specific questionnaire as a measure of disability would assist in evaluating the true effectiveness of this physiotherapy exercise intervention. However the results of the study reflect the real life situation of clinical practice and exercise prescription in a sporting environment.

CONCLUSIONS

The finding of this field study support that a specific individually prescribed physiotherapy exercise intervention was associated with a reduction in the prevalence of LBP in a population of adolescent female rowers across a rowing season. Reduced levels of LBP intensity in subjects who received the physiotherapy intervention was also observed although there were no statistically significant changes in disability levels across the rowing season. These findings need to be supported by randomized controlled study before their validity can be assured.

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ARTICLE

Rugby league injuries in New Zealand: Variations in injury claims and costs by ethnicity, gender, age, district, body site, injury type and occupation

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ABSTRACT

Aim: **This paper provides an overview of the epidemiology of New Zealand rugby league injuries requiring medical treatment and associated costs analysed by ethnic groups.**

Method: **New Zealand national Accident Compensation Corporation injury data for the period 1999 to 2007 were searched for rugby league injury cases. Data were analysed by ethnic groups for demographics, body region, nature/severity of injury, and medical procedure and costs.**

Results: **New Zealand Maori accounted for 39.8% of the number of total injury claims and 43.5% of the total injury entitlement costs but were recorded as only 13.2% of the total New Zealand population. Accounting for only 3.2% of the population distribution living in Auckland, New Zealand Maori recorded 11.7% of the total injury claims in the Auckland district. Soft tissue injuries accounted for 10.6% (±8.5%) of injury claims and 7.9% (±6.7%) of injury entitlement costs for all ethnic groups. New Zealand Maori recorded more injury claims for the knee than all other ethnic groups. Injury claims for New Zealand Europeans recorded more trade occupations, New Zealand Maori more plant and machinery occupations and Pacific peoples more elementary occupations. New Zealand Maori recorded significantly more injury claims for both males and females than all other ethnic groups over the study period.**

Conclusions: **This study identified the number of injury claims, and associated costs of the injuries, by ethnic group that have occurred from participation in rugby league activities in New Zealand over an eight year period. NZ Maori are disproportionately participating in rugby league in NZ, but the proportions injured are consistent with reported proportions playing the game. Further research is warranted to fully explore the differences in injury rate between the ethnic groups and to what extent these differences in levels of participation in rugby league activities.**

Key Words: **rugby league, ethnicity, injury, incidence**

INTRODUCTION

The use of sports injury surveillance to provide epidemiological data is useful in describing the occurrence of injuries and associated risk factors[27] and in directing and monitoring the effectiveness of programs designed to reduce the risk of injury from participation in sporting activities.[2, 32] Sports injury reduction programs include modifying coaching material and training resources,[10] modifying training techniques[23, 25] and equipment[19] and implementing law changes in sporting activities.[30]

Studies undertaken on rugby league injuries have looked at the incidence of injuries in both the match and training environments;[5, 6, 20] changes in injury incidence as a result of changes in playing season,[12, 16] rule changes,[26] different participation levels;[5, 6, 28] anthropometric and physiological characteristics of rugby league participants,[7] and the cost of injuries from rugby league participation.[4, 24] A recent national epidemiological analysis of eight years (1999-2007) of moderate to serious injury claims lodged with the Accident Compensation Corporation (ACC) for rugby league injury in New Zealand[21] showed that the knee was the most common injury site and soft tissue injuries were the most common injury type. Concussions contributed 1.8% of total injury claims but 6.3% of injury entitlement costs and had the highest mean cost per claim ($25,347 [£9,420]). Despite the increasing number of international studies on rugby league no study has looked at the incidence of injuries of different ethnic groups as a result of participation in rugby league activities. We were interested in any differences in injury incidence and characteristics by ethnicity given the differences in proportions of ethnicities playing rugby league[3] compared to the proportions of ethnicities in the New Zealand population.[1] In the New Zealand population of 4.1 million[1] in 2006 the reported four largest ethnic groups were New Zealand Europeans (67.6%), New Zealand Maori (14.7%), Pacific Peoples (6.9%) and Asian (9.2%), whilst in the New Zealand Rugby League participation population 42% were reported as New Zealand Maori, 32% New Zealand European, 22% Pacific and 4% are other[3].

With this in mind, the aim of this study was to provide an epidemiological overview of rugby league injuries and associated costs in one country (New Zealand) by ethnic groups over an eight year period (1999 to 2007). New Zealand's national no-fault injury compensation system administered by the Accident Compensation Corporation (ACC) means that New Zealand is uniquely positioned to provide detail descriptive epidemiological data, including costs associated with treatment, on sports (such as rugby league) injuries. Using this data, comparisons by ethnic group for the rate, site, type, cost, location and occupation of players for rugby league injuries were conducted.

Data Limitations

Epidemiological studies are dependant on data quality for any analysis to be undertaken.[9] The data provided for the analysis in this study are from the ACC database and are dependant on several factors.[21] Additionally, data were limited to the ACC database only as there were no other available databases for the collection of player specific data such as: (1) Numbers participating in rugby league activities; (2) Age of players participating; (3) Identification of the ethnicity of players; and (4) Number of matches played and trainings completed enabling match and training exposure hours.

METHODS

ACC cover is available to any person, including visitors to New Zealand, that suffers any accidental personal injury at any time when in New Zealand. People who have a personal injury can make a claim to ACC at the time of seeking medical treatment from over 30,000 registered health professionals throughout New Zealand. When making a claim, information about the injury is collected using a standard ACC 45 injury reporting form to ensure levels of consistency for data recording and analyses. The injured person (unless impaired) completes information about the activity surrounding the injury (e.g., location, activity prior, cause, narrative) along with their personal details (e.g., age, gender, ethnicity, contact details). The registered health professional completes the form by providing information regarding initial diagnosis and other relevant medical information (e.g., surgical procedure). The claim is then filed with ACC and details are entered into a central database. ACC makes no disincentive for making claims nor are people risk-rated or penalised for the amount of claims they make.[8] Personal injury coverage is guaranteed by ACC but this is offset by the restriction to sue for personal injury, except in rare circumstances, for exemplary damages.[8] ACC categorises the claims made as minor or moderate to serious claims (MSC) and as new or ongoing injury claims. Minor injury claims reflect those injuries that require the initial medical treatment or service and may only require one or two visits to a health professional. Moderate to serious injury claims reflect those injuries that require additional treatment than the initial medical treatment or service, for example income replacement if the injury requires more than 5 consecutive days off work. Serious injury claims are permanent brain injury or spinal damage. New claims are those made in the reporting period and are reflective of the number of new injuries from rugby league participation. Ongoing injury claims are reflective of the severity of the injury claim and can carry on over the reporting period. In this study MSCs are reported with new and ongoing injuries combined per reporting period.

Throughout the study period there were 42,754 new and ongoing claims costing ACC $NZD 48,704,704 [£GBP18,099,470]. MSC represented 13.9% (5,941) of the number of total claims but 87.9% ($NZD 42,822,048 [£GBP15,916,048]) of the costs. For this study we focused on MSC rather than minor claims as defined by the ACC for its sports cost outcome model,[8] that occurred between 1st July 1999 and the 30th June 2007 as a result of participating in rugby league activities. ACC data were analysed by ethnic groups for: (1) Region/District; (2) Injury type; (3) Injury site and (4) Occupation[15] (see Table 1). A potential limitation to the ACC data are the way the data are retrieved to protect client confidentiality by limiting the access to low level results under four injury claims. Therefore any data less than or equal to three injury claims have been rounded to represent three claims only. New Zealand population data were obtained from official government data. This data set provides estimates of resident populations between each five year census.[1] The population of New Zealand over the study period was approximately 4.1 million people based on the 2006 census.[1] Rugby player participation proportions were based on the published work of Falcous.[3]

TABLE 1: Data categories analysed.

Ethnicity Group includes	Region Area/District	Injury Site Group/Sub Group	Injury Type	Occupation NZSCO Categories[15]
New Zealand European	**North Island**	**Head/Neck**	Soft tissue injury	Legislators, Administrators, and Managers
New Zealand Maori	Northland	Head (except face)	Corrosive injuries	Professionals
Pacific Peoples	Auckland	Face	Dental injuries	Technicians and Associate Professionals
Cook Island Maori	Waikato	Eye	Fracture / Dislocation	Clerks
Tongan	Bay of Plenty	Nose	Hernia	Service and Sales Workers
Niuean	Gisborne	Ear	Laceration, puncture, wound, sting	Agriculture and Fishery Workers
Fijian	Hawke's Bay	Neck / Back of head / vertebrae	Foreign body in orifice / eye	Trades Workers
Tokelauan	Taranaki	**Upper Limb**	Concussion / brain injury	Plant and Machine Operators and Assemblers
Other Pacific	Manawatu-Wanganui	Shoulder (including Clavicle/Shoulder blade)	Amputation	Elementary Occupations
Asian	Wellington	Upper and Lower Arm	Deafness	Unknown
South East Asian	**South Island**	Elbow	Other/Unknown	
Indian	Tasman	Hand / wrist		
Other/Unknown	Nelson	Finger / Thumb		
	Marlborough	**Lower Limb**		
	Canterbury	Hip/Upper leg/Thigh		
	West Coast	Knee		
	Otago	Lower Limb		
	Southland	Ankle		
	Other/Unknown	Foot		
		Toes		
		Other		
		Abdomen / Pelvis		
		Chest		
		Back / Spine		
		Internal organ		
		Multiple locations		
		Unknown		

Ethical Consent

Ethical consent for the research was obtained from the Auckland University of Technology Ethics Committee. Informed consent from the injured participants was not obtained as data were collected from the ACC data base without individual player identification or follow-up.

Statistical Analyses

All the data collected were entered into a Microsoft Excel spreadsheet and analysed with SPSS v.16.0 (SPSS Inc, Chicago, Illinois, USA). Injury rates were calculated as the number of injuries per 1,000 injury claims.[11, 17, 18] Data are reported as means with 95% confidence intervals (CI).[31] The injury rates and patterns were compared between reporting years using a one sample chi-squared (^{2}) test. Significant p values reported in the text are less than 0.0001 if they are not specifically stated. Injury incidence was not calculated for the study as rugby league participation rates were not available as part of the data analysis. All costs are reported in New Zealand Dollars ($) and Great Britain Pounds (£) unless otherwise indicated.

RESULTS

Over the study period, there were 5,941 new and ongoing moderate to serious injury claims recorded. New Zealand Maori recorded significantly more injury claims than all other ethnic groups (see Table 2).[1] They accounted for 39.8% of the number of total injury claims and 43.5% of the total injury entitlement costs but were recorded as only 13.2% of the total population. All other ethnic groups accounted for fewer claims and costs. New Zealand European (2=267, df=7) and New Zealand Maori (X^2=183, df=7) recorded a decrease in the incidence of injury claims over the duration of the study while Pacific Peoples (X^2=169, df=7) and Asian (X^2=18, df=7) recorded an increase (see Table 3).

TABLE 2: Total injury rate per 1,000 entitlement claims, 95% Confidence Intervals and percentage of total claims, costs, percentage of total costs and mean cost per claim by ethnic groups from 1999 to 2007.

		INJURY CLAIMS						DIFFERENCE FROM ETHNIC GROUP[1]				
Ethnic Group	Population Distribution %[1]	Number	Total Claims Rate (95% CI)	%	Total Costs $(000) $(000)	%	Mean	NZ European X^2 (df=1)	NZ Maori X^2 (df=1)	Pacific people X^2 (df=1)	Asian X^2 (df=1)	Other X^2 (df=1)
New Zealand European	61.2	1,839	309.6 *(295.8-324.1)*	31	12,377	28.9	6.7	-	65	100	1742	88.4
New Zealand Maori	13.2	2,365	398.1 *(382.4-414.5)*	39.8	18,615	43.5	7.8	65	-	323	2268	1350
Pacific Peoples	6.2	1,280	215.5 *(204.1-227.7)*	21.5	8,304	19.4	6.4	100	323	-	1184	430
Asian	8.4	33	5.9 *(4.2-8.2)*	0.6	113	0.3	3.2	1742	2268	1184	-	335
Other/Unknown	10.9	424	71.7 *(65.2-78.8)*	7.1	3,413	7.9	8	88.4	1350	430	335	-
Total	**100**	**5,491**	**1,000.0** ***(974.9-1025.8)***	**100**	**42,822**	**100**	**7.2**					

CI: Confidence Interval. Rates are per 1,000 injury claims. 1 = p values all <0.0001.

TABLE 3: Total injury claims by reporting year per 1,000 entitlement claims, 95% Confidence Intervals and costs for ethnic groups from 1999 to 2007.

	NEW ZEALAND EUROPEAN			NEW ZEALAND MAORI			PACIFIC PEOPLES			ASIAN			OTHER		
Yr	No	Rate (95% CI)	$(000)	No	Rate (95% CI)	$(000)	No	Rate (95% CI)	$(000)	No	Rate (95% CI)	$(000)	No	Rate (95% CI)	$(000)
99-00	228	325.3 (319.4-414.0)	1,312	245	390.7 (344.8-442.9)	1,681	110	175.4 (145.5-211.5)	626	3	4.8 (1.5-14.8)	2	43	68.6 (50.9-92.5)	188
00-01	155	363.6 (245.7-336.6)	911	229	424.9 (373.2-483.6)	1,772	112	209.6 (174.3-252.1)	706	5	11.1 (5.0-24.8)	25	40	74.2 (54.4-101.2)	202
01-02	138	287.6 (232.2-324.2)	841	235	467.2 (411.1-530.9)	1,724	107	214.7 (177.8-259.3)	890	0	0.0 -	0	22	43.7 (28.8-66.4)	227
02-03	142	274.4 (250.3-347.4)	1,064	193	400.0 (347.5-460.4)	1,754	115	239.2 (199.4-286.9)	673	0	0.0 -	0	32	68.0 (48.4-95.7)	147
03-04	160	294.8 (221.4-301.8)	1,142	275	444.3 (394.7-500.0)	2,080	139	224.6 (190.2-265.2)	861	3	4.8 (1.6-15.0)	2	42	67.9 (50.1-91.8)	240
04-05	305	258.5 (288.5-361.1)	1,784	356	376.7 (339.6-418.0)	2,452	200	211.6 (184.3-243.1)	1,207	5	6.3 (2.9-14.1)	16	77	81.5 (65.2-101.9)	343
05-06	322	322.8 (271.6-337.9)	2,405	410	385.7 (350.1-424.9)	2,630	243	228.6 (201.6-259.2)	1,565	8	7.5 (3.8-15.0)	36	80	75.3 (60.4-93.7)	720
06-07	390	302.9 (303.9-370.6)	2,913	422	363.2 (330.1-399.5)	4,517	254	218.6 (193.3-247.2)	1,781	9	7.7 (4.0-14.9)	28	88	75.7 (61.5-93.3)	1,294
Total	**1,839**	**309.6 (295.89-324.1)**	**12,377**	**2,365**	**398.1 (382.4-414.5)**	**18,615**	**1,280**	**215.5 (204.1-227.7)**	**8,304**	**33**	**5.9 (4.2-8.2)**	**113**	**424**	**71.7 (65.2-78.86)**	**3,413**

CI: Confidence Interval. Rates are per 1,000 injury claims.

TABLE 4: Total population percentage by district and injury claims by district per 1,000 entitlement claims, 95% Confidence Intervals, costs and percentage of injury claims for ethnic groups from 1999 to 2007.

	NEW ZEALAND EUROPEAN				NEW ZEALAND MAORI				PACIFIC PEOPLES				ASIAN				OTHER/UNKNOWN			
	P%	No. [Rate(95% CI)]	$(000)	%	P%	No. [Rate(95% CI)]	$(000)	%	P%	No. [Rate(95% CI)]	$(000)	%	P%	No. [Rate(95% CI)]	$(000)	%	P%	No. [Rate(95% CI)]	$(000)	%
North Island	**43.9**	**1129 [190.0(179.3-201.5)]**	**6949**	**19.0**	**11.5**	**2021 [340.2(325.7-355.3)]**	**15584**	**34.0**	**5.8**	**1082 [182.1(171.6-193.3)]**	**6553**	**18.2**	**7.4**	**29 [4.9(3.4-7.0)]**	**86**	**0.5**	**7.6**	**295 [49.7(44.3-55.7])**	**3012**	**5.0**
Northland	2.2	46 [7.7(5.8-10.3)]	183	**0.8**	1	145 [24.4(20.7-28.7)]	499	**2.4**	0.1	18 [3.0(1.9-4.8)]	190	**0.3**	0.1	0 [0.0-]	0	**0.0**	0.4	31 [5.2(3.7-7.4)]	47	0.5
Auckland	16.4	637 [107.2(99.2-115.9)]	4585	**10.7**	3.2	698 [117.5(109.1-126.6)]	6113	**11.7**	4.2	817 [137.5(128.4-147.3)]	3997	**13.8**	5.5	20 [3.4(2.2-5.2)]	56	**0.3**	2.8	148 [24.9(21.2-29.3)]	1629	2.5
Waikato	6	133 [22.4(18.9-26.5)]	525	**2.2**	1.8	338 [56.9(51.1-63.3)]	2599	**5.7**	0.3	35 [5.9(4.2-8.2)]	197	**0.6**	0.4	2 [0.3(0.1-1.3)]	1	**0.0**	1	41 [6.9(5.1-9.4)]	869	0.7
Bay of Plenty	3.9	73 [12.3(9.8-15.5)]	529	**1.2**	1.6	231 [38.9(34.2-44.2)]	1611	**3.9**	0.2	38 [6.4(4.7-8.8)]	167	**0.6**	0.2	0 [0.0-]	0	**0.0**	0.7	21 [3.5(2.3-5.4)]	118	0.4
Gisborne	0.5	8 [1.3(0.7-2.7)]	6	**0.1**	0.5	62 [10.4(8.1-13.4)]	275	**1.0**	0	2 [0.3(0.1-1.3)]	44	**0.0**	0	0 [0.0-]	0	**0.0**	0.1	5 [0.8(0.4-2.0)]	67	0.1
Hawke's Bay	2.3	16 [2.7(1.7-4.4)]	85	**0.3**	0.8	86 [14.5(11.7-17.9)]	374	**1.4**	0.1	13 [2.2(1.3-3.8)]	101	**0.2**	0.1	0 [0.0-]	0	**0.0**	0.4	5 [0.8(0.4-2.0)]	45	0.1
Taranaki	1.8	55 [9.3(7.1-12.1)]	205	**0.9**	0.4	123 [20.7(17.4-24.7)]	1008	**2.1**	0	6 [1.0(0.5-2.2)]	17	**0.1**	0.1	0 [0.0-]	0	**0.0**	0.3	9 [1.5(0.8-2.9)]	58	0.2
Manawatu	3.7	77 [13.0(10.4-16.2)]	445	**1.3**	1	139 [23.4(19.8-27.6)]	1662	**2.3**	0.1	20 [3.4(2.2-5.2)]	75	**0.3**	0.2	0 [0.0-]	0	**0.0**	0.7	10 [1.7(0.9-3.1)]	16	0.2
Wellington	7.1	84 [14.1(11.4-17.5)	386	**1.4**	1.3	199 [33.5(29.2-38.5)]	1443	**3.3**	0.8	133 [22.4(18.9-26.5)]	1765	**2.2**	0.9	7 [1.2(0.6-2.5)]	29	**0.1**	1.2	25 [4.2(2.8-6.2)]	163	0.4
South Island	**17.3**	**678 [114.1(105.8-123.0)]**	**4734**	**11.4**	**1.7**	**306 [51.5(46.0-57.6)]**	**2437**	**5.2**	**0.4**	**158 [26.6(22.8-31.1)]**	**1287**	**2.7**	**1**	**4 [0.7(0.3-1.8)]**	**21**	**0.1**	**3.3**	**122 [20.5(17.2-24.5)]**	**714**	**2.1**
Tasman	0.8	1 [0.2(0.0-1.2)]	7	**0.0**	0.1	2 [0.3(0.1-1.3)]	6	**0.0**	0	0 [0.0-]	0	**0.0**	0	0 [0.0-]	0	**0.0**	0.2	0 [0.0-]	0	0.0
Nelson	0.8	8 [1.3(0.7-2.7)]	32	**0.1**	0.1	7 [1.2(0.6-2.5)]	44	**0.1**	0	3 [0.5(0.2-1.6)]	7	**0.1**	0	0 [0.0-]	0	**0.0**	0.1	4 [0.7(0.3-1.8)]	24	0.1
Marlbourgh	0.8	9 [1.5(0.8-2.9)]	67	**0.2**	0.1	5 [0.8(0.4-2.0)]	21	**0.1**	0	0 [0.0-]	0	**0.0**	0	0 [0.0-]	0	**0.0**	0.2	2 [0.3(0.1-1.3)]	12	0.0
Canterbury	9.2	450 [75.8(69.1-83.1)]	2671	**7.6**	0.9	241 [40.6(35.8-46.0)]	2133	**4.1**	0.3	129 [21.7(18.3-25.8)]	1150	**2.2**	0.7	4 [0.7(0.3-1.8)]	21	**0.1**	1.7	87 [14.6(11.9-18.1)]	597	1.5
West Coast	0.6	100 [16.8(13.8-20.5)]	541	**1.7**	0.1	5 [0.8(0.4-2.0)]	56	**0.1**	0	1 [0.2(0.0-1.2)]	1	**0.0**	0	0 [0.0-]	0	**0.0**	0.1	7 [1.2(0.6-2.5)]	30	0.1
Otago	3.5	81 [13.6(11.0-17.0)]	1285	**1.4**	0.3	25 [4.2(2.8-6.2)]	83	**0.4**	0.1	20 [3.4(2.2-5.2)]	103	**0.3**	0.2	0 [0.0-]	0	**0.0**	0.6	12 [2.0(1.1-3.6)]	23	0.2
Southland	1.6	29 [4.9(3.4-7.0)]	131	**0.5**	0.2	21 [3.5(2.3-5.4)]	94	**0.4**	0	5 [0.8(0.4-2.0)]	26	**0.1**	0	0 [0.0-]	0	**0.0**	0.3	10 [1.7(0.9-3.1)]	29	0.2
Other / Unknown	0	32 [5.4(3.8-7.6)]	383	0.5	0	38 [6.4(4.7-8.8)]	252	0.6	0	40 [6.7(4.9-9.2)]	314	0.7	0	0 [0.0-]	0	0.0	0	7 [1.2(0.6-2.5)]	10	0.1

CI: Confidence Interval. Rates are per 1,000 injury claims. P% = Percentage of population from Census 20061; $(000) expressed in New Zealand dollars.

Total Injury Claims by District and Ethnicity

Accounting for only 3.2% of the population distribution living in Auckland, New Zealand Maori recorded 11.7% of the total injury claims in the Auckland District (see Table 4). This was similar for New Zealand Maori in all other North Island districts. In the South Island, New Zealand Maori recorded more total injury claims in Canterbury (4.1%) but accounted for only 0.9% of the population distribution. This was similar for Pacific Peoples in Auckland (claims: 13.8%; population: 4.2) Wellington (claims: 2.2%; population 0.8%) and Canterbury (claims: 2.2% population 0.3%). All other districts and ethnic groups had a similar percentage distribution for both injury claims and population distribution.

Total Injury Claims by Injury Type and Ethnicity

Soft tissue injuries accounted for 10.6% (±8.5%) of injury claims and 7.9% (±6.7%) of injury entitlement costs for all ethnic groups (see Table 5). Fracture dislocations (claims: 10.0% ±7.7%; costs: 7.9% ±6.0%), concussions (claims: 0.5% ±0.4; costs: 1.3% ±2.4%) and lacerations & wounds (claims: 0.5% ±0.4%; costs: 0.2% ±0.1%) were slightly less. New Zealand Maori recorded more soft tissue injuries (X^2=1,500, df=4), fracture-dislocations (X^2=1,311, df=4) and concussions (X^2=72, df=4) than other ethnic groups.

Total Injury Claims by Injury Site and Ethnicity

More injury claims were made for injuries to the lower than the upper limbs for most (New Zealand European: X^2=6, df=1, p=0.0120; New Zealand Maori: X^2=0, df=1, p=0.7650; Pacific Peoples: X^2=8, df=1, p=0.0042; Asian: X^2=0.6, df=1, p=0.4142) ethnic groups (see Table 6). The knee was the most common injury site recorded for all ethnic groups over the study period. New Zealand Maori recorded more injury claims for the knee than all other ethnic groups (X^2=683, df=4). Although New Zealand Maori recorded more total head and neck injury claims than all other ethnic groups (X^2=303, df=4), New Zealand Europeans recorded more neck (X^2=105, df=1) and face (X^2=63, df=1) injury claims.

TABLE 5: Total injury claims by injury type per 1,000 entitlement claims, 95% Confidence Intervals and costs for ethnic groups from 1999 to 2007.

	NEW ZEALAND EUROPEAN			NEW ZEALAND MAORI			PACIFIC PEOPLES			ASIAN			OTHER/UNKNOWN		
	No	**Rate** *(95% CI)*	**$(000)**	**No**	**Rate** *(95% CI)*	**$(000)**	**No**	**Rate** *(95% CI)*	**$(000)**	**No**	**Rate** *(95% CI)*	**$(000)**	**No**	**Rate** *(95% CI)*	**$(000)**
Soft tissue Injuries	980	164.9 *(154.9-175.6)*	5,377	1110	186.8 *(176.1-198.1)*	7,002	595	100.1 *(92.4-108.5)*	3,396	29	4.9 *(3.4-7.0)*	52	214	36.0 *(31.5-41.2)*	1,174
Fracture / Dislocations	887	149.3 *(139.7-159.4)*	5,220	1032	173.6 *(163.4-184.6)*	6,503	568	95.6 *(88.0-103.8)*	3,375	28	4.7 *(3.3-6.8)*	57	231	38.9 *(34.2-44.2)*	1,776
Other/Unknown	102	17.2 *(13.1-22.5)*	1,618	121	20.4 *(15.9-26.1)*	2,592	75	12.6 *(9.3-17.3)*	999	0	0.0 -	-	23	3.9 *(2.6-5.8)*	3,363
Concussion / Brain injury	41	6.9 *(5.1-9.4)*	86	62	10.4 *(8.1-13.4)*	2,363	17	2.9 *(1.8-4.6)*	44	0	0.0 -	-	31	5.2 *(3.7-7.4)*	239
Lacerations / Wounds	48	8.1 *(6.1-10.7)*	2	42	7.1 *(5.2-9.6)*	111	30	5.0 *(3.5-7.2)*	108	0	0.0 -	-	14	2.4 *(1.4-4.0)*	11
Dental Injuries	20	3.4 *(2.2-5.2)*	18	6	1.0 *(0.5-2.2)*	15	24	4.0 *(2.7-6.0)*	385	0	0.0 -	-	0	0.0 -	-
Deafness	0	0.0-	-	13	2.2 *(1.3-3.8)*	14	0	0 -	-	0	0.0 -	-	0	0.0 -	-
Hernia	8	1.3 *(0.7-2.7)*	4	3	0.5 *(0.2-1.6)*	4	0	0 -	-	0	0.0 -	-	0	0.0 -	-
Corrosive Injuries	0	0.0-	-	3	0.5 *(0.2-1.6)*	3	0	0 -	-	0	0.0 -	-	0	0.0 -	-
Foreign body	0	0.0-	-	3	0.5 *(0.2-1.6)*	1	0	0 -	-	0	0.0 -	-	0	0.0 -	-

CI: Confidence Interval. Rates are perr 1,000 injury claims; $(000) expressed in New Zealand Dollars.

TABLE 6: Total injury claims by injury site per 1,000 entitlement claims, 95% Confidence Intervals and costs for ethnic groups from 1999 to 2007

	NEW ZEALAND EUROPEAN			NEW ZEALAND MAORI			PACIFIC PEOPLES			ASIAN			OTHER/UNKNOWN		
Body Site	**No**	**Rate** *(95% CI)*	**$(000)**	**No**	**Rate** *(95% CI)*	**$(000)**	**No**	**Rate** *(95% CI)*	**$(000)**	**No**	**Rate** *(95% CI)*	**$(000)**	**No**	**Rate** *(95% CI)*	**$(000)**
Head & Neck	**239**	**40.2** ***(35.4-45.7)***	**2,438**	**245**	**41.2** ***(36.4-46.7)***	**4,138**	**126**	**21.2** ***(17.8-25.3)***	**1,153**	**3**	**0.5** ***(0.2-1.6)***	**2**	**88**	**14.8** ***(12.0-18.3)***	**1,288**
Head (except face)	37	6.2 *(4.5-8.6)*	86	73	12.3 *(9.8-15.5)*	3,302	29	4.9 *(3.4-7.0)*	403	0	0.0 -	0	27	4.5 *(3.1-6.6)*	242
Neck, Back of head	85	14.3 *(11.6-17.7)*	2,138	56	9.4 *(7.3-12.2)*	614	35	5.9 *(4.2-8.2)*	274	0	0.0 -	0	23	3.9 *(2.6-5.8)*	980
Face	61	10.3 *(8.0-13.2)*	116	42	7.1 *(5.2-9.6)*	82	38	6.4 *(4.7-8.8)*	443	3	0.5 *(0.2-1.6)*	2	17	2.9 *(1.8-4.6)*	19
Eye	12	2.0 *(1.1-3.6)*	16	27	4.5 *(3.1-6.6)*	48	9	1.5 *(0.8-8.0)*	8	0	0.0 -	0	0	0.0 -	0
Ear	0	0.0 -	0	21	3.5 *(2.3-5.4)*	35	3	0.5 *(0.2-1.6)*	2	0	0.0 -	0	0	0.0 -	0
Nose	44	7.4 *(5.5-10.0)*	82	26	4.4 *(3.0-6.4)*	58	12	2.0 *(1.1-3.6)*	23	0	0.0 -	0	21	3.5 *(2.3-5.4)*	47
Upper Limb	**774**	**130.3** ***(121.4-139.8)***	**3,887**	**939**	**158.1** ***(148.3-168.5)***	**5,259**	**480**	**80.8** ***(73.9-88.4)***	**2,312**	**24**	**4.0** ***(2.7-6.0)***	**44**	**196**	**33.0** ***(28.7-37.9)***	**728**
Shoulder	353	59.4 *(53.5-66.0)*	2,052	403	67.8 *(61.5-74.8)*	3,208	204	34.3 *(29.9-39.4)*	1,113	11	1.9 *(1.0-3.3)*	32	87	14.6 *(11.9-18.1)*	402
Upper & Lower arm	93	15.7 *(12.8-19.2)*	660	127	21.4 *(18.0-25.4)*	640	78	13.1 *(10.5-16.4)*	425	0	0.0 -	0	42	7.1 *(5.2-9.6)*	144
Elbow	37	6.2 *(4.5-8.6)*	266	44	7.4 *(5.5-10.0)*	118	28	4.7 *(3.3-6.8)*	99	0	0.0 -	0	5	0.8 *(0.4-2.0)*	5
Hand/wrist	164	27.6 *(23.7-32.2)*	624	221	37.2 *(32.6-42.4)*	849	116	19.5 *(16.3-23.4)*	474	13	2.2 *(1.3-3.8)*	12	41	6.9 *(5.1-9.4)*	137
Finger/Thumb	127	21.4 *(18.0-25.4)*	285	144	24.2 *(20.6-28.5)*	444	54	9.1 *(7.0-11.9)*	201	0	0.0 -	0	21	3.5 *(2.3-5.4)*	40
Lower Limb	**876**	**147.4** ***(138.0-157.5)***	**4,116**	**952**	**160.2** ***(150.4-170.8)***	**5,091**	**573**	**96.4** ***(88.9-104.7)***	**2,993**	**30**	**5.0** ***(3.5-7.2)***	**63**	**185**	**31.1** ***(27.0-36.0)***	**1,216**
Hip, Upper leg, Thigh	59	9.9 *(7.7-12.8)*	174	42	7.1 *(5.2-9.6)*	139	28	4.7 *(3.3-6.8)*	134	0	0.0 -	0	21	3.5 *(2.3-5.4)*	46
Knee	469	78.9 *(72.1-86.4)*	2,739	484	81.5 *(74.5-89.1)*	3,290	303	51.0 *(45.6-57.1)*	1,826	11	1.9 *(1.0-3.3)*	36	91	15.3 *(12.5-18.8)*	854
Lower Leg	117	19.7 *(16.4-23.6)*	446	139	23.4 *(19.8-27.6)*	579	70	11.8 *(9.3-14.9)*	380	10	1.7 *(0.9-3.1)*	8	30	5.0 *(3.5-7.2)*	79
Ankle	194	32.7 *(28.4-37.6)*	714	241	40.6 *(35.8-46.0)*	996	147	24.7 *(21.0-29.1)*	596	9	1.5 *(0.8-2.9)*	18	37	6.2 *(4.5-8.6)*	234
Foot	34	5.7 *(4.1-8.0)*	42	39	6.6 *(4.8-9.0)*	86	25	4.2 *(2.8-6.2)*	59	0	0.0 -	0	0	0.0 -	0
Toes	3	0.5 *(0.2-1.6)*	1	7	1.2 *(0.6-2.5)*	2	0	0.0 -	0	0	0.0 -	0	6	1.0 *(0.5-2.2)*	3
Chest, Back, Other	**197**	**33.2** ***(28.8-38.1)***	**1,415**	**259**	**43.6** ***(38.6-49.2)***	**2,054**	**130**	**21.9** ***(18.4-26.0)***	**911**	**0**	**0.0 -**	**0**	**48**	**8.1** ***(6.1-10.7)***	**49**
Chest	48	8.1 *(6.1-10.7)*	70	91	15.3 *(12.5-18.8)*	116	25	4.2 *(2.8-6.2)*	33	0	0.0 -	0	21	3.5 *(2.3-5.4)*	31
Back/Spine	68	11.4 *(9.0-14.5)*	1,229	78	13.1 *(10.5-16.4)*	1,931	29	4.9 *(3.4-7.0)*	827	0	0.0 -	0	9	1.5 *(0.8-2.9)*	15
Abdomen/Pelvis	15	2.5 *(1.5-4.2)*	91	9	1.5 *(0.8-2.9)*	5	12	2.0 *(1.1-3.6)*	52	0	0.0 -	0	3	0.5 *(0.2-1.6)*	3
Internal organs	12	2.0 *(1.1-3.6)*	24	0	0.0 -	0	3	0.5 *(0.2-1.6)*	<0.5	0	0.0 -	0	0	0.0 -	0
Multiple Injuries	0	0.0 -	0	4	0.7 *(0.3-1.8)*	2	0	0.0 -	0	0	0.0 -	0	0	0.0 -	0
Unknown	**54**	**9.1** ***(7.0-11.9)***	**516**	**77**	**13.0** ***(10.4-16.2)***	**1,837**	**61**	**10.3** ***(8.0-13.2)***	**808**	**0**	**0.0 -**	**0**	**15**	**2.5** ***(1.5-4.2)***	**81**

CI: confidence Interval. Rates are per 1,000 injury claims. Totals do not equal due to data rounding; $(000) expressed in New Zealand dollars.

TABLE 7: Total injury claims by occupational group per 1,000 entitlement claims, 95% Confidence Intervals and costs for ethnic groups from 1999 to 2007.

			NEW ZEALAND EUROPEAN				NEW ZEALAND MAORI				PACIFIC PEOPLES				ASIAN				OTHER/UNKNOWN			
Occupation	**P%**[2]	**RL%**	**No**	**Rate** *(95% CI)*	**%**	**$(000)**	**No**	**Rate** *(95% CI)*	**%**	**$(000)**	**No**	**Rate** *95% CI)*	**%**	**$(000)**	**No**	**Rate** *(95% CI)*	**%**	**$(000)**	**No**	**Rate** *(95% CI)*	**%**	**$(000)**
Trades Workers	7.9	15.9	376	63.3 *(57.2-70.0)*	6.3	1,583	304	51.2 *(45.7-57.3)*	5.1	1,718	191	32.1 *(27.9-37.0)*	3.2	1,277	5	0.8 *(0.4-2.0)*	0.1	13	66	7.0 *(5.5-8.9)*	1.1	454
Plant & Machinery	5.8	21.3	339	57.1 *(51.3-63.)*	5.7	3,957	589	99.1 *(91.4-107.5)*	9.9	2,671	240	40.4 *(35.6-45.8)*	4.0	1,216	0	0.0 -	0.0	0	98	10.4 *(8.5-12.7)*	1.6	521
Other[1]	5.7	16.5	319	53.7 *(48.1-59.9)*	5.4	1,379	356	59.9 *(54.0-66.5)*	6.0	6,269	198	33.3 *(29.0-38.3)*	3.3	1,456	10	1.7 *(0.9-3.1)*	0.2	39	100	10.6 *(8.7-12.9)*	1.7	659
Elementary[1]		19.6	275	46.3 *(41.1-52.1)*	4.6	1,553	560	94.3 *(86.8-102.4)*	9.4	4,197	242	40.7 *(35.9-46.2)*	4.1	2,137	7	1.2 *(0.6-2.5)*	0.1	32	81	8.6 *(6.9-10.7)*	1.4	1152
Sales & Service	9.4	5.4	125	21.0 *(17.7-25.1)*	2.1	686	99	16.7 *(13.7-20.3)*	1.7	501	75	12.6 *(10.1-15.8)*	1.3	291	3	0.2 *(0.0-1.2)*	0.0	1	21	2.2 *(1.5-3.4)*	0.4	87
Agriculture & Fisheries	11.0	4.9	91	15.3 *(12.5-18.8)*	1.5	573	141	23.7 *(20.1-28.0)*	2.4	711	40	6.7 *(4.9-9.2)*	0.7	196	0	0.0 -	0.0	0	21	2.2 *(1.5-3.4)*	0.4	118
Clerks	12.1	6.0	79	13.3 *(10.7-16.6)*	1.3	646	127	21.4 *(18.0-25.4)*	2.1	1,230	132	22.2 *(18.7-26.4)*	2.2	728	4	0.7 *(0.3-1.8)*	0.1	7	14	1.5 *(0.9-2.5)*	0.2	68
Technicians	12.2	5.0	103	17.3 *(14.3-21.0)*	1.7	663	88	14.8 *(12.0-18.3)*	1.5	535	93	15.7 *(12.8-19.2)*	1.6	413	3	0.3 *(0.1-1.3)*	0.0	5	10	1.1 *(0.6-2.0)*	0.2	132
Professionals	18.9	3.0	71	12.0 *(9.5-15.1)*	1.2	336	55	9.3 *(7.1-12.1)*	0.9	516	45	7.6 *(5.7-10.1)*	0.8	462	3	0.3 *(0.1-1.3)*	0.0	8	7	0.7 *(0.4-1.6)*	0.1	112
Legislators	17.1	2.3	61	10.3 *(8.0-13.2)*	1.0	365	46	7.7 *(5.8-10.3)*	0.8	263	24	4.0 *(2.7-6.0)*	0.4	132	3	0.3 *(0.1-1.3)*	0.0	4	6	0.6 *(0.3-1.4)*	0.1	60

CI: Confidence Interval; Rate reported per 1,000 injury claims; P% = percentage of population from Census 2006[1]*; $(000) expressed in New Zealand dollaras; totals do not equal due to data rounding.*
1 = Employment categopry combined under Statistics New Zealand classifications.
2 = percentages reported from 2006 Census.
RL% = total percentage of total injury claims by occupational group.

TABLE 8: Total injury claims by ethnic group with 95% Confidence Intervals and percentages of total claims from 1999 to 2007. Total population by ethnic group and percentage of population, injury costs and mean cost per claim by ethnic group from 1999 to 2007.

		No	%	No	Rate *(95% CI)*	%	$(000)	%	Mean $
Male	New Zealand European	1,255,257	31.2	1764	313.7 *(299.4-328.7)*	31.4	9,185	22.3	5,207
	New Zealand Maori	274,860	6.8	2205	392.1 *(376.1-408.9)*	39.2	13,526	32.8	6,134
	Pacific Peoples	131,007	3.3	1224	217.7 *(205.8-230.2)*	21.8	6,315	15.3	5,159
	Asian	169,374	4.2	33	5.9 *(4.2-8.3)*	0.6	86	0.2	2,598
	Other/Unknown	304,497	7.6	377	67 *(60.6-74.2)*	6.7	2,257	5.5	5,984
Female	New Zealand European	1,354,332	33.6	75	234.4 *(186.9-293.9)*	23.4	355	22.5	4,722
	New Zealand Maori	290,466	7.2	160	500.0 *(428.2-583.8)*	50.0	746	47.4	4,659
	Pacific Peoples	134,967	3.4	56	175.0 *(134.7-227.4)*	17.5	262	16.6	4,673
	Asian	185,175	4.6	0	0.0 -	0.0	-	0.0	-
	Other/Unknown	282,564	7.0	47	146.9 *(110.4-195.5)*	14.7	211	13.4	4,486

Rates are per 1,000 injury claims; CI: Confidence Intervals; $(000) expressed in New Zealand dollars; Population numbers from 2006 census.

Total Injury Claims by Occupation and Ethnicity

Occupational injury claims varied by ethnic groups. New Zealand Europeans recorded more injuries for trade workers (X^2=551, df=4), New Zealand Maori more injuries for plant and machinery workers (X^2=810, df=4) and Pacific peoples more injuries for elementary occupations (X^2=755, df=4) than other ethnic groups (see Table 7).

Total Injury Claims by Gender and Ethnicity

New Zealand Maori recorded significantly more injury claims for both male (X^2=2,939, df=4) and female (X^2=202, df=4) than all other ethnic groups over the study period (see Table 8). When compared with the population distribution, New Zealand Maori males recorded 6.8% of the population distribution but recorded 39.2% of the injury claims and 32.8% of the injury entitlement costs. This was similar for New Zealand Maori females (pop: 7.2%; claims: 50.0%; costs: 47.4%); Pacific Island males (pop: 3.3%; claims 21.8%; costs 15.3%) and Pacific Island females (pop: 3.4%; claims: 17.5% costs 16.6%).

DISCUSSION

This study identified the number of injury claims, and associated costs, by ethnic group that have occurred from participation in rugby league activities in New Zealand over an eight year period. There have been no studies looking at ethnicity and injuries in rugby league. Despite this there has been some ethnographic research exploring ethnic rugby league players at the professional level of participation in the Northern hemisphere.[29] Seen as a development of personal and social self, ethnic players reportedly participate in contact sports such as rugby league because of the physicality of the game and the ability to be aggressive in a controlled way.[29] Participation in sport by ethnic groups has been described as a positive experience for the players where sports skills are encouraged, and rewarded, amongst their peers, and the wider community, as well as enabling bonding with the players *"whanau, hapu, iwi and friends"*.[33] It is one area where players can demonstrate their masculinity and rowess to both other males and to females.[29] This participation has also been described as an area where a player's identity is tied up with the mythologies that some see rugby league as achieving (working class solidarity) and being able to "*achieve success and play with pakeha as equals*." [3]

When compared to the percentage proportion of population, New Zealand Maori are over-represented in many sporting codes.[33] It has been previously reported that 42% of rugby league participants are New Zealand Maori, 32% European, 22% Pacific and 4% are of other ethnic groups.[3] No current rugby league participation data were available for the period of our study to use as a comparison. The results of our study identified that 39.8% of the total injury claims were recorded as being New Zealand Maori, 28.9% New Zealand European, 19.4% Pacific People and 8.3% other ethnicity. The percentages varied by island and district. The North Island recorded more New Zealand Maori injury claims while the South Island recorded more New Zealand European injury claims. Auckland recorded more Pacific Peoples injury claims (12.5%) despite Pacific Peoples only accounting for 4.6% of the total population in Auckland. There were more New Zealand Maori injury claims in Northland and from Waikato through to Tasman than New Zealand Europeans' claims. New Zealand Europeans recorded more injury claims from Nelson through to Southland than New Zealand Maori. Further research is warranted in identifying whether the ethnic distribution difference as observed in this study for injury claims is also evident in participation numbers.

The incidence of injury claims decreased each year for New Zealand European and New Zealand Maori. In contrast, Pacific Peoples and Asians recorded an increase in the incidence of injury claims over the same reporting period. As reported in another epidemiological study,[21] the incidence of injury claims initially decreased over the 1999-2000 to 2001-02 reporting periods then increased by 25.9% (±19.8%) over the 2002-06 reporting period. It is not known whether the actual participation numbers varied across the study period or if there were other influences over the same reporting period. The implementation of a national rugby league participation database would assist in identifying any changes in ethnic groups and player numbers for future research.

The finding that soft tissue injuries were recorded as the most common injury type for most ethnic groups reflects the nature of the game of rugby league.[5] Soft tissue injuries (sprains, strains, contusions and haematomas) accounted for 46.2% (±3.5%) of total injury types recorded for all ethnic groups. This was similar for fracture dislocations recording 44.6% (±2.7%) of the total injury types. Combined, these injury types accounted for 76.9% (±20.4%) injury entitlement costs for all ethnic groups. This is reflective of the nature of rugby league where tackling, being tackled, acceleration, deceleration and change of direction are key components of rugby league at all levels of participation.[4] Studies reporting injuries in rugby league have identified that these injury types are the most common to occur at all levels of participation.[22] As these injury types are similar between all ethnic groups, injury prevention / reduction programs should be generalisable for all ethnic groups. This would include primary care of these types of injuries through appropriate sideline care by qualified personnel. Further longitudinal research is warranted once these programs are commenced to see what impact on the incidence of injury claims the programs have.

The finding that the lower limb was the most common injury entitlement region may be reflective of the ethnic group differences. Recent research on foot function and morphology of different ethnic groups in rugby league has shown that there are functional differences in foot loading and morphology during gait for different ethnicities.[14] New Zealand Maori and New Zealand Europeans were reported to have a narrower hallux angle and a wider forefoot in regards to total area of the foot in comparison to Pacific Peoples. As shown by this study more injury claims were recorded for the lower limb by New Zealand Maori (17.3%) and New Zealand Europeans (15.9%) than Pacific Peoples (10.4%) and Asians (0.5%). Although no relationship has been proven between the different ethnic groups foot morphology and the incidence of injuries, research is warranted on ascertaining if there is a correlation. Other aspects to be considered are whether there are different stressors on the ankle and knee joints of different ethnicities and how this relates to participation in contact sports such as rugby league.

The incidence and costs of concussions is a concern. King et al.[21] identified that the mean cost of a concussion over the same reporting period was $25,347 (£9,487). The mean cost per concussion over the duration of this study varied by ethnic group. New Zealand European's recorded the lowest mean cost of $2,113 (£803) per concussion while New Zealand Maori recorded the highest mean cost per concussion of $38,118 (£14,488). Explanation of this is beyond the scope of this study but questions raised by this finding are: (1) Is there a reason for the difference in costs associated with concussions by different ethnic groups?; and (2) Do New Zealand Maori have an increased risk of complications from concussions when compared with New Zealand Europeans and Pacific Peoples? The reason for the mean high cost per concussion, and in particular for New Zealand Maori, are areas that warrant further investigation. Comparison between other ethnic groups, outside of those identified in this study, may also be warranted to further explore the area of concussion by ethnicity.

The ethnic groups reported in the ACC data base are limited and data interpretation may influence what ethnic group the injury entitlement claim will be entered in as, especially if two ethnic groups are selected (i.e. New Zealand Maori and New Zealand European). Another limitation in the use of this data is the occupational classification identified. The occupational classifications provided are limited to just ten classifications as identified by the New Zealand Standard Classifications of Occupations.[15] Although there are sub classifications under this standard, these are not recorded in the data input process. The data interpretation of the occupational classification is open for interpretation at the data input stage by the data input operator i.e. a program manager can be classified as professional, technical or administrator whereas they may be a program manager for a trade worker, a computer operator or an elementary occupation.

Despite the limitations of occupational classifications, the current study is similar to another study[13] that reported occupational classification of participants in rugby league. Greenwood[13] identified the small number of 'white-collar' workers and a greater combination of skilled and unskilled workers participating in rugby league activities. Plant and machine operators and assemblers (20.6%), elementary occupations (18.8%) and trade workers (16.8%) were the most commonly reported occupational groups of participants in our study. Professionals, legislators, administrators, managers, technicians and associate professionals recorded only 12% of the total injury claims over the study period. This would suggest, similar to Greenwood,[13] that rugby league participation is primarily undertaken by the 'blue collar' class of occupations in the employment sector.

CONCLUSIONS

Although this study was unable to establish the injury incidence per 1,000 match hours for ethnic groups, it has identified that there are some differences in the injury site and injury type between different ethnic groups. The costs associated with the different ethnic groups as a result of injuries from rugby league participation were identified. Further research is warranted to fully explore the differences between the ethnic groups and what extent these differences are from participation in rugby league activities. Further questions raised by this study are: (1) Despite an increase in the total injury incidence over the period why is there a decrease in injury claims for New Zealand Europeans and New Zealand Maori but an increase in injury claims for Pacific Peoples and Asians?; (2) Are there anthropometric and physiological aspects of different ethnic groups that impact on the incidence of injuries in rugby league participants?

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ARTICLE

Atrial fibrillation in athletes - how to recognise it and how to deal with it

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Atrial fibrillation is the commonest arrhythmia in athletes. It particularly affects those athletes who have undertaken high intensity endurance training over many years. It can affect athletes at all levels, from the elite to the recreational athlete. Guidelines for the management in non-athletes are often irrelevant in competitive athletes, for whom restoration and maintenance of sinus rhythm is the only acceptable outcome. Atrial fibrillation is responsible for significant morbidity and occasional mortality. With appropriate management the athlete can return to sporting participation at the highest level.

This article is based on a literature search plus a New Zealand perspective, given our own personal experiences of dealing with individuals with the condition over the last decade or so.

Atrial fibrillation may be described as a discoordinate contraction of the atria. A more evocative description is that of "a bag of worms", a phrase beloved of teachers of undergraduate medical students. The consequences of atrial fibrillation are twofold. Firstly, there is loss of the atrial booster pump which reduces cardiac output by about 30 per cent. This, therefore, has a profound effect on sporting performance. Secondly, if an athlete remains in atrial fibrillation for over 48 hours there is a significant risk of clot formation in the left atrial appendage. Portions of this clot can break off and embolise. The consequence of the embolus depends on the site in which it lodges, but can be serious, e.g. stroke, heart attack or limb threatening peripheral embolus.

Atrial fibrillation is classified in two ways. A clinical classification divides the condition into paroxysmal (attacks lasting less than seven days) or chronic (continuous) atrial fibrillation. An important other method of classification is by aetiology. Primary atrial fibrillation, i.e. that occurring with no identified risk factors, is sometimes termed lone atrial fibrillation. By contrast, secondary atrial fibrillation results from associated causes as listed in Table 1. The endurance trained athlete exhibits enhanced vagal tone and therefore is more sensitive to small doses of sympathomimetics. Therefore, all athletes presenting with atrial fibrillation need to be closely questioned with regard to their intake of energy drinks plus caffeine or cold and flu tablets, e.g. those containing pseudoephedrine.

Why are trained athletes at increased risk of atrial fibrillation? The proposed mechanism goes something like this. Aerobic training causes volume overload. This volume overload in turn causes four chamber enlargement. Enlargement of the atria results in fibrosis and collagen deposits. These collagen deposits have heterogeneous conduction properties. Prolonged endurance training causes enhanced vagal tone and shortening of the refractory period. A shortened refractory period in turn predisposes to the development of atrial fibrillation.

The symptoms include an irregular pulse. Classically, this is irregularly irregular with varying pulse volume. Some athletes are very astute at describing this phenomenon. Usually there will be palpitations which are an abnormal sensation of the heart beating within the chest. The athlete will describe a dramatically reduced exercise tolerance due to the loss of the atrial booster pump. In the most well documented case, which was televised on nationwide TV during the 2008 trials for the New Zealand rowing team for the Beijing Olympics, Rob Waddell developed a bout of atrial fibrillation and described it graphically as "like rowing through mud". Also the athlete will feel extremely short of

TABLE 1: Aetiology of atrial fibrillation - secondary causes.

ACUTE	CHRONIC
1 Sympathomometics	1 Hypertensive heart disease
a Energy drinks	2 Rheumatic heart disease, especially mitral stenosis
b Caffeine	3 Ischaemic heart disease
c Cold and flu tablets	4 Cardiomyopathy
2 Alcohol	5 Conduction defects, eg, Wolff-Parkinson-White syndrome, sick sinus syndrome
3 Thyrotoxicosis	
4 Myocarditis	
5 Pericarditis	
6 Pulmonary embolism	

breath. These symptoms may be present continuously or intermittently depending on whether the atrial fibrillation is chronic or paroxysmal. It is very rare for athletes to present with manifestations of embolic phenomena.

What are the signs on clinical examination? Firstly, the athlete can be expected to have an irregularly irregular pulse if examined during a bout of atrial fibrillation. There will usually be variable pulse volume. Because of the reduced cardiac output, there may be postural hypotension. Signs of cardiac failure are rare in athletes. Very occasionally there will be evidence of structural heart disease with displacement of the apex beat or a heart murmur. Importantly, between attacks the examination should be entirely unremarkable.

In terms of investigations, the most critical is the electrocardiograph (ECG). This will capture the arrhythmia if the athlete is in atrial fibrillation at the time. If not, then strenuous attempts should be made to capture the arrhythmia via a Holter monitor or an event monitor. Echocardiography is useful to look for thrombus formation in the left atrial appendage and evidence of structural heart disease, e.g. valve abnormalities or thickening of chamber walls. Detailed electrophysiology studies can identify areas of abnormal tissue that harbour electrical wavelets that promote re-entry. Over time a period of atrial fibrillation will induce electrophysiological changes followed by structural changes which facilitate its persistence.

What is the natural history of atrial fibrillation in athletes? Surprisingly, there are very few long term studies and the best documented one is that by Hoogsteen et al[1] who studied 30 male athletes from 1993 to 2002. These athletes had a mean age of 48 years, i.e. they were older than most elite performers. Of the initial 30 athletes, 7 had no further episodes of atrial fibrillation, 15 went on to develop paroxysmal atrial fibrillation and 5 developed continual atrial fibrillation. Three athletes died during the course of the study: one of these was recorded as sudden death; one athlete died of a cerebral bleed and that athlete was on warfarin; the final death was from a cerebral infarct and that athlete was not on warfarin. Athletes who require anticoagulation should not participate in contact, i.e. collision, sports.

Management is enhanced by early recognition by the patient resulting in prompt presentation. If the athlete has been in atrial fibrillation for less than 48 hours then immediate cardioversion to sinus rhythm is indicated. This may be attempted initially chemically with intravenous Flecainide or Amiodarone. If this is unsuccessful then DC cardioversion is required. If the athlete has been in atrial fibrillation for over 48 hours then anticoagulation for six weeks is required before conversion to sinus rhythm can be attempted.

Maintenance of sinus rhythm is critical. Firstly, the athlete should avoid known provocateurs, especially sympathomimetic agents. Secondly, anti-arrhythmic drugs are usually required; Flecainide is the agent usually used in athletes and high doses may be required to obtain adequate blood levels. In recreational athletes Sotalol or Amiodarone has some efficacy in maintaining sinus rhythm. Anti-arrhythmic drugs have negative inotropic effects but these are of minimal importance compared with the negative effects on cardiac output if the athlete remains in atrial fibrillation. If the athlete can be safely maintained in sinus rhythm then there is no compelling need for them to take warfarin.

Increasingly, clinicians are turning to catheter ablation in an attempt to provide a long term cure for the condition without the need to take regular medication. Ablation involves introducing electrode catheters into the heart under fluoroscopic guidance to record electrical signals. The electrode catheter is navigated to a critical site at which ablative energy via a radiofrequency current is delivered to create a localised scar that will disrupt the cause of the arrhythmia[2]. Several New Zealand centres are developing significant expertise in this technique. The procedure may need to be repeated if not all arrhythmogenic foci are ablated. Complications of ablation include a pericardial effusion, which is usually transient and of no consequence. Pulmonary vein stenosis may develop insidiously and is life-threatening. Likewise, an atrio-oesophageal fistula may develop and this can lead to death via catastrophic haemorrhage. Air embolism or cerebrovascular attack is another complication that can be fatal. Fortunately, all of these last three complications are very rare. The rate of important complications relating to the procedure has been put at less than 3 per cent in a multicentre clinical trial.

There are some issues which are worth highlighting. Firstly, the issue of rate versus rhythm control which is emphasised in the New Zealand Guidelines Group publication. This is an irrelevant discussion in high performance athletes as only sinus rhythm is acceptable. However, in recreational athletes it does become a relevant issue. Secondly, there is the issue of adrenergic versus vagal induction of the arrhythmia. Adrenergic induction is associated with exercise and tends to occur in younger patients, whereas vagal induction occurs at rest and tends to be more the preserve of older patients.

In summary, atrial fibrillation is responsible for significant morbidity and occasional mortality. In competitive athletes only sinus rhythm is acceptable. It is important to behave in a physicianly manner and look for any underlying causes for atrial fibrillation. Once identified, the treating doctor should seek expert advice from a cardiologist. Skilled appropriate management should enable the athlete to resume competition at the highest level.

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ARTICLE

Acupuncture - history and the scientific evidence behind its use

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Definition

Acupuncture – derived from the Latin words "acus", meaning needle, and "pungere", meaning to prick i.e. to puncture with a needle – traditionally describes the process of briefly inserting fine needles into various points on the body to either relieve pain or treat various illnesses and conditions[1].

It is now used to refer to a wide range of procedures that stimulate specific anatomical points on the body. The World Health Organisation uses the term broadly to include traditional needle insertion, moxibustion (needling with the application of a heat source to the end of the needle), electro-acupuncture, laser (or photo-) acupuncture, microsystem acupuncture (e.g., on the ear, face, head or scalp) and acupressure (application of pressure to specific points on the body)[2].

History and Origins

There is some debate over the exact origin of acupuncture, though it appears to have originated in China over 2000 years ago and subsequently spread to Japan, Korea, Vietnam and across Asia[3]. Others have hypothesized that non-decorative tattoos as a result of needling and burning seen in precise locations on the bodies of Stone Age mummies indicates that a similar practice may have been used in Central Europe as early as 5000 years ago[4].

By the first century BC, acupuncture appears to have become well established and at around this time the first written record – the Inner Classic of the Yellow Emperor – was produced[5].

Whatever its exact origins, in China, the development of acupuncture occurred at around the same time as the philosophies of Confucianism and Taoism. Unlike Western Medicine (WM) where the science is based on objective observations, measurement and analysis, Traditional Chinese Medicine (TCM) places the emphasis on human sensory awareness and teaches that a practitioner's observation and contemplation are sufficient to understand and explain the human condition[5].

Central to the practice of acupuncture are the concepts of Qi, yin and yang, and the Five Elements. Qi is a philosophical concept that can loosely be described as "vital energy" and flows through meridians in the body. Yin and yang are the basic intuitions of TCM and are complementary opposites that describe everything in nature – yin represents the more material aspects while yang represents the more immaterial; something may therefore have elements of both yin and yang. The Five Elements – wood, water, earth, fire and metal – represent various processes and qualities of the human condition and together with yin and yang are used to describe the state of health or being of an individual[6].

Thus disease or ill-health is thought to arise from an imbalance or disharmony between Qi, yin, yang and the five elements. It is believed that acupuncture works by modulating or dynamically harmonising the flow of Qi and any imbalances that are present, thereby restoring health[6].

Due to the huge number of interactions possible between these concepts, acupuncture treatment is a very individualized therapy and often two people presenting with the same problems will receive differing treatment regimes. It is also common for the regime to change from one session to the next depending on what has apparently been modified since the previous treatment[7].

The majority of acupuncture points as described in TCM are situated on twelve main meridians and two of the eight extra meridians through which Qi is believed to flow[5]. At present there does not appear to be any anatomical basis for these points and there is very little scientific evidence to suggest that they can be objectively detected[8].

Despite still being considered an alternative or complementary therapy, acupuncture is gaining wider acceptance as a legitimate form of therapy in the Western world, particularly among patients for whom conventional science and medicine does not appear to have all the answers. Most Western physicians and practitioners of acupuncture however do not subscribe to the TCM theory regarding acupuncture and instead validate their practice in terms of the discoveries regarding its potential mechanisms of action, myofascial trigger points etc[9]. Thus in WM, acupuncture tends to be used in a more standardized form rather than the highly individualized treatment plans prevalent in TCM.

Since the 1970s, there has been extensive research into acupuncture, its effects and mechanisms. Despite evidence demonstrating that it is effective in a number of conditions[5] there is a persistently high level of scepticism regarding acupuncture in healthcare systems based on WM.

Effects of Acupuncture

Since interest in acupuncture began to rise in the early 1970s there have been hundreds of trials trying to evaluate its efficacy in various conditions. A number of reviews and meta-analyses of these trials have found that the majority produced conflicting or at best equivocal evidence for the efficacy of acupuncture[5]. Nausea and vomiting[10,11,12,13] and dental pain[14] are the two main exceptions where there is substantial evidence supporting its use. At the other end of the spectrum, trials of acupuncture in the treatment of tinnitus[15] and addictions[16] have shown resoundingly that it does not work.

In 1997 the National Institutes of Health Consensus Development Panel on acupuncture in the USA concluded that[17].

"There is clear evidence that needle acupuncture is efficacious for adult postoperative and chemotherapy nausea and vomiting and probably for nausea of pregnancy. Much of the research focuses on various pain problems. There is evidence of efficacy for postoperative dental pain. There are reasonable studies (although sometimes only single studies) showing relief of pain with acupuncture on diverse pain conditions…..However, there are also studies that do not find efficacy for acupuncture in pain. Although many other conditions have received some attention…..the quality or quantity of the research evidence is not sufficient to provide firm evidence of efficacy at this time."

In 2003 the WHO published a review of clinical trials of acupuncture and the following list of diseases, symptoms and conditions for which they felt there was sufficient evidence to conclude that acupuncture is an effective treatment[2].

The report also included lists of a large number of other conditions for which acupuncture may be an effective treatment but where further research is required.

This report should not however be seen as an endorsement of acupuncture as a treatment by the WHO, and there is some concern regarding its objectivity due to the input (and potential bias) of several complementary and alternative medicine practitioners.

Aside from nausea and vomiting and dental pain as outlined above, there is growing evidence to support the efficacy of acupuncture in the treatment of chronic pain and hypertension. In Germany, studies showing the effectiveness of acupuncture in the treatment of chronic pain in osteoarthritis of the knee[18] and in back pain[19] have led to its use being regularly integrated into their management.

Flachskampf *et al* have shown that regular acupuncture can reduce both systolic and diastolic blood pressure to a similar degree as is seen with the use of ramipril and calcium channel antagonists[20].

Mechanisms of Action

A number of mechanisms have been discovered by which acupuncture may exert its effects, particularly with regard to pain, though much still remains to be elucidated.

Early research worked on the premise that the analgesic effect of acupuncture was due to the gate control theory of pain as described by Melzack and Wall[21,22].

A study in the early 1970s suggested that chemical mediators were somehow involved in acupuncture analgesia. CSF from a rabbit that had been subjected to acupuncture was transferred into the third ventricle of a rabbit that had not been given acupuncture and a corresponding analgesic effect was noted in the recipient rabbit[23]. Early research into the identity of these postulated chemical mediators implicated various neurotransmitters, particularly serotonin[24].

In the late 1970s however it was discovered that the analgesic effect of acupuncture could be blocked or reversed by the administration of the opioid antagonist naloxone[25,26]. Han *et al* also discovered that CCK-8, an endogenous opioid antagonist, blocked the analgesic effects of both morphine and acupuncture in rats[27]. Together, these suggested that endogenous opioids were involved in the analgesic effect of acupuncture.

Other research has suggested that acupuncture may mediate inflammatory responses in the body. Sun concluded that acupuncture may inhibit early phase vascular permeability, impair leucocyte adherence to vascular endothelium and suppress exudative reactions[28]. The study also suggested that acupuncture may mediate these processes to a degree that is equivalent to that found with oral aspirin or indomethacin.

Guo *et al*[29] and Gao *et al*[30] have both postulated that acupuncture may stimulate the gene expression of various neuropeptides which then in turn mediate the effects attributable to acupuncture.

Newer brain imaging modalities such as functional MRI and positron emission tomography have allowed researchers to study the physiological and functional responses of the brain in response to

- Acute bacillary dysentery
- Acute epigastric pain
- Adverse reactions to radiotherapy and/or chemotherapy
- Allergic rhinitis (including hay fever)
- Biliary colic
- Correction of malposition of the foetus,
- Depression
- Essential hypertension
- Facial pain (including craniomandibular disorders)
- Headache
- Induction of labour
- Knee pain
- Leucopenia
- Low back pain
- Morning sickness
- Nausea and vomiting
- Neck pain
- Pain in dentistry
- Periarthritis of shoulder
- Postoperative pain
- Primary dysmenorrhoea
- Primary hypotension
- Renal colic
- Rheumatoid arthritis
- Sciatica
- Sprain
- Stroke
- Tennis elbow

acupuncture stimulation of the body. Cho *et al* have shown that stimulation of acupuncture points in the foot that are traditionally related to vision activates areas of the visual cortex in a similar manner as if a light had been shone in the eyes; stimulation of adjacent control points did not elicit the same response[31].

Stimulation of the PC6 acupuncture point is well known to produce anti-emetic effects (though it should be used with caution in the treatment of nausea and vomiting in early pregnancy as it is thought to increase the risk of miscarriage[32]). Yoo *et al* found that stimulation of PC6 consistently activated a number of neural substrates, in particular in the left superior frontal gyrus, the anterior cingulate gyrus, the dorsomedial nucleus of the thalamus and areas of the cerebellum which was not replicated by sham acupuncture at a different, non-acupuncture point[33].

A number of studies have shown activation of pain processing areas (such as the primary somatosensory cortex, the anterior cingulate and prefrontal cortex and the periaqueductal grey matter) in response to acupuncture stimulation[34,35,36].

Perhaps the most controversial (and difficult to study) potential mechanism of action for acupuncture is the placebo effect. The majority of the evidence outlined above is in a laboratory setting and it is not clear if this can be extrapolated into the true clinical setting. The main difficulty that arises in trying to study or quantify the placebo effect is using a suitable placebo. Many studies use sham acupuncture as a placebo, but this has been shown to have consistently similar results to true acupuncture – it may be that needling is what causes the effects and acupuncture points are simply the locations that have been found to be the most effective[8].

Summary

While there is still much scepticism in the Western world regarding acupuncture, there is mounting evidence of its efficacy, particularly in the treatment of nausea and vomiting and pain. Improving technology is allowing increased understanding of the underlying mechanisms involved but as yet there is much that remains unknown. Further research is required but it should be borne in mind that there are many unique difficulties in the study of acupuncture, particularly regarding appropriate controls and placebos.

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Who's the most versatile athlete we've produced?

Over the Christmas holidays one's mind tends to wander - my mind was no exception to this. I was pondering the recent success of some of our New Zealand rowers who have excelled at other sports. In particular Jaime Nielsen, who was on the fringe of the New Zealand Women's Rowing 8, became a medallist in the New Zealand Women's Team Pursuit team at the 2009 World Championships; Hamish Bond competed with distinction in the Round Taupo event and competed for the winning professional cycling team at the nation's premier multistage event, the Tour of Southland; Sonia Waddell, who has recently retired from rowing, finishing 11th in the women's road race at the recent National Championships.

This got me to thinking what criteria one should use to assess our most versatile athlete. For my money, the things to consider would be the skill set required, the energy systems utilised, and whether or not they are able to compete at a high level in both an individual and a team sport.

Over the years there have been some outstanding individuals who have demonstrated great versatility. When I was a boy, the ultimate was to be a double All Black. This term covers those who have played for both the All Blacks and the New Zealand national representative cricket team, now dubbed the Black Caps. The last true double All Black was Brian McKechnie, but I think even he would concede that Jeff Wilson (Goldie), who never quite made the Black Caps to play test cricket, was probably a more talented all-round athlete than himself. Jeff also proved himself a promising basketballer.

I have always been in awe of the fitness and skill set of Michael Jones, who would be one of our greatest All Blacks. I believe he could have competed in the decathlon with distinction, probably at least to Commonwealth Games level. The same probably applies to Bruce Reihana and other All Blacks who have chosen rugby as their career but, in essence, are multitalented individuals. Andrew Mehrtens, for example, was an outstanding young tennis player before going on to become on of our greatest first five-eighths. Likewise, Peter Snell and John Walker are best remembered as outstanding middle distance track runners but were themselves handy tennis players in youth.

Amongst our women it is well known that the best netball players are often superb touch rugby players in the off-season. From the Waikato, where I am based, both Jenny May Coffin and Amigene Metcalfe would exemplify this trend. More recently Anna Scarlett has demonstrated good versatility, switching from netball to beach volleyball.

As a fan of both netball and rowing, it would be interesting to see what our Silver Fern defenders like Casey Williams, Jolene Henry or Leana de Bruin would do in a rowing skiff. Likewise, Laura Langman would probably prove to be quite a good lightweight rower. However, these girls are far too talented at netball to take them away to another sport.

Arguably, the biggest transition I have seen was for Nigel Avery, who transformed himself from a triple jumper to an elite weightlifter in the course of a few years. This obviously necessitated a dramatic alteration in body type, and even more so as weightlifters generally rely on short levers whereas triple jumpers tend to be long limbed. Nigel was amongst the tallest of weightlifters, but his dedication and application got him to bronze medal performances at the Kuala Lumpur Commonwealth Games.

Somewhat earlier, Madonna Harris made a successful transition from elite snow sports performance to cycling.

However, the greatest demonstration of versatility in my view would be that of Rob Waddell, our Olympic gold medal winning rower from the 2000 Olympics. After winning that gold medal plus multiple World Championships he has carried on at an elite level, switching to America's Cup yachting as a grinder. I well recall him telling me that rowing was all about the legs, whereas grinding was all about the arms. It seems to be the way of astute elite athletes that they can sum up the requirements of their discipline in a succinct fashion. In between these careers, he had a short stint at playing rugby for Waikato as a lock and his athleticism and physical presence were impressive. He was arguably the most mobile lock since Ian Jones last took the field.

Let's not forget Rob's wife Sonia, who in her junior years was a 400 metre hurdler competing at the World Junior Championships. Then she converted to rowing as a single sculler and also competed in crew boats. She made two Olympic finals and one silver medal at the World Championships in a quad. More recently, as mentioned above, she has taken up road cycling and one can only speculate as to what her success in that discipline had been like if she had started younger.

Doubtless I have missed some worthy contenders for this title; my selections only reflect athletes I have seen in my 53 years on this planet. I would welcome opinion on the matter from any readers who feel I have overlooked any outstanding competitors whose names are not mentioned. Some of the old timers in our membership may well consider those from the '40s or '50s before sport was televised would fit the bill as our most versatile elite athlete.

For the present, I would stick with my selections.

Chris Milne
Sports Physician and
Avid Sports Fan

2009 - The year in sports and exercise medicine

This column is an attempt to summarise the major issues in sports and exercise medicine as I have seen them over the last year or so. It is by no means comprehensive, but will outline some of the significant issues and developments in our discipline.

The year started off with promotion of a National Cycleway as part of the Government's initiative to promote jobs and also improve the options for physically active tourists who wish to visit here. The concept of an active holiday is relatively new but has been embraced widely, especially by Europeans who place a high value on our varied scenery and clear blue skies. Building on the success of initiatives such as the Central Otago Rail Trail, the concept of a National Cycleway embraces job creation plus a recognition that encouragement of active tourists is a positive thing.

The second issue is an ethical one; 2009 saw the twin controversies of 'Bloodgate' and 'Crashgate'. The first incident arose during a Harlequins rugby match in the UK where the team doctor and physiotherapist appeared to be complicit in an attempt to falsify a blood problem in a player so that player could be substituted by a recognised kicker. There was a New Zealand connection in that the recognised kicker was none other than Nick Evans, who many regard as the player we should have retained in New Zealand as an understudy to Dan Carter. In the event the team coach, Dean Richards, was suspended from all contact with rugby, as was the team physiotherapist, and the team doctor has also been dealt with via the General Medical Council in the UK. This issue sends strong messages to team support staff from all codes that they need to maintain the highest ethical standards.

No less an event was the staged crash in the Singapore Formula 1 Grand Prix by the Renault team. The team boss was found to have been deeply involved in the decision to deliberately crash a car. This is a crash that could have had fatal consequences and the team was rightly punished. It sickens me to hear that a French court has subsequently tried to mitigate the circumstances, as deliberate crashing of a high speed vehicle where the driver's life could be put at risk hardly constitutes what most people would regard as sporting behaviour.

The third issue relates to gender verification. This is a topic that has sunk under the radar in recent years but was brought to world attention when the South African runner, Caster Semenya, won a gold medal in the Women's 800 metres at the World Athletics Championships in Berlin. I have a high regard for South African medicine and, as it turned out, the South African team doctors had alerted the relevant athletics officials to a gender issue. Unfortunately, those officials chose to ignore medical advice and this athlete was subjected to worldwide humiliation as her muscular physique was picked up by sports commentators around the world and the issues were debated at length in the world media. This is hardly the way to discuss a sensitive issue of this nature and, to my mind, the relevant athletics officials should have been sacked. In this instance, it would appear that the medical staff involved did all they could to protect the athlete's rights and integrity, and it is a shame when the officials did not follow through with their obligations.

The fourth issue worthy of comment is that of head injury. The case of Felipe Massa, the Formula 1 driver who was hit in the head by a flying metal object and put out of competition for several weeks, is one of the great head injury comebacks of all time. One would have suspected that his driving days were over, and indeed Michael Schumacher was potentially going to be contacted to drive for the Ferrari team in some of the later races of the season but succumbed to a neck injury himself and was unable to fulfil that role. The wonderful thing is that Massa has made what appears to be a full recovery and his is an object lesson to all people with head injuries that miracles are indeed possible in certain circumstances.

Returning to local issues, one of the developments in the New Zealand Academy of Sport in the past year has been the appointment of fulltime resource people to manage various aspects of high performance sport. These experts have been recruited and are tasked with realising the potential of our best athletes in the lead up to the London 2012 Olympics. The challenge is for clinicians who are only involved with elite sport on a part time basis to ensure that their expertise is available to Academy athletes at short notice to provide the necessary clinical back up. Otherwise, we risk being left off the Academy of Sport's list of providers and one may suspect that elite athlete care would then become the province of a few fulltime doctors. To my mind this would be a grave mistake and, therefore, it is incumbent on all clinicians to make themselves available when the need arises for their services.

Later in the year there were substantial changes to ACC funding borne out of the financial circumstances the Corporation found itself in. This has had the most impact on physiotherapy, where funding for care has been reduced and part charges have been reintroduced. To my mind, the previous service with no part charge was unsustainable in the long term and the physiotherapy profession will adapt, as it has over the years. The practitioners that do well will be those that provide good clinical care and meet outcome measures.

Sports injuries to elite athletes are also major items in the daily news, and injuries to Daniel Carter (achilles tendon) and Shane Bond (multiple body parts) have been in the headlines for various reasons. The important thing is that we, as clinicians, have to provide the best back up we can to such elite athletes and strive to replicate that care as best we can for the weekend warrior.

2010 brings new challenges and, in particular, there are new guidelines for the use of asthma medications. WADA has loosened the rules somewhat and athletes can now use Ventolin (Salbutamol) as a short acting beta agonist, and Serevent (Salmeterol) as a long acting beta agonist merely by applying to Drug Free Sport New Zealand with a declaration of use. Previously, use of these drugs required a positive bronchial provocation test. However, clinicians looking after elite athletes need to be aware that asthma is an inflammatory disease and the prescription of inhaled corticosteroids would be regarded as essential for all athletes in whom a beta agonist was required any more often than a few times a year.

Late in the year the New Zealand Olympic Committee and New Zealand Academy of Sport collaborated to appointment a medical advisory group to provide leadership and direction with regard to sports and exercise medicine issues for our elite athletes. This comprises Drs Dave Gerrard, Steve Targett, Graham Paterson and Peter Burt, and Sharon Kearney, one of our most experienced physiotherapists. I believe this group has the collective wisdom to provide great direction for our elite athletes in the coming years.
So there you have it, this is my summation of the past year in sports and exercise medicine with regard to some significant events and issues. There are many challenges ahead and we are well placed to deal with them.

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BOOK REVIEW

The Sports Medicine Resource Manual

Editors: Peter Seidenberg and Anthony Beutler
Publisher: Saunders Elsevier 2008
Price: NZ$170

Firstly about the title; this is primarily a sports injury resource manual. Do not expect to find anything much about non-injury matters and you will not be disappointed. The only non-injury topics that are covered in any detail include cardiovascular testing, exercise induced asthma testing, ergogenic aids and exercise prescription for various selected populations.

The vast majority of the book is dedicated to injury management and this it does relatively well. One useful distinction made early on in the book is that between victim or culprit. The authors describe this as a pathoanatomic approach to diagnosis. In essence they are referring to the structure being injured, which they describe as the victim, and the forces, tissues or objects causing the injuries which are referred to as the culprits. In other words, it is the distinction between the diagnostic label and the underlying cause as we would recognise it in our daily practice.

This book pays due emphasis to the physical examination. There are eight chapters listing physical examination features and special diagnostic tests for various parts of the body. These are generally pretty comprehensive, although the section on shoulder examination does not mention the belly press test for assessing subscapularis integrity. The lift off test, however, is well covered. Generally the chapters are pretty comprehensive and, in these days of over-reliamce on imaging findings, the emphasis on physical examination is very welcome.

The majority of the book, approximately 60 per cent, is devoted to specific fractures and soft tissue injuries. It is arranged by body part and generally the advice given is similar to that which would be given by experts in this part of the world. There are some points of difference, for example the chapter authors advocate use of a brace or corset for pars injuries, e.g. stress fractures of the lumbar spine. This is not standard practice in Australasia.

There is a section on rehabilitation and this includes a chapter on the practical application of osteopathic manipulation in sports medicine written by two osteopaths. In the USA osteopaths have visiting rights to hospitals, which is rather different to the situation that pertains in New Zealand. The principles of manipulation are well described but the use of these techniques is not confined to osteopaths. Other professional groups including physiotherapists and chiropractors also use such techniques. A major weakness of this chapter is the lack of any reference to the evidence base on which manipulation is grounded.

Other chapters in this section include a description of core stability exercises which appears to be pretty much inline with other presentations I have encountered, and does list the level of evidence upon which various interventions are based. There are chapters on the various physical therapy modalities plus athletic taping and the proper indications and uses of orthopaedic braces.

These are useful references for those wanting further information about these particular topics. The final section of the book is devoted to special tests and procedures. In addition to chapters on cardiovascular testing, testing for maximal aerobic power and exercise induced asthma testing, there is a chapter on gait analysis plus one on assessment and selection of running shoes. This includes a useful table summarising the various midsole technologies used by the various manufacturers in today's running shoes. The authors provide good practical advice that is of relevance both to the novice and experienced runner. The chapter on concussion testing and management is a curious blend of best practice advice from the Prague Consensus Guidelines generated in 2004 including the SCAT card plus a curious hangover from the previous American grading system which the international guidelines were designed to put to rest. I suspect it will be some years yet before American clinicians properly align themselves with the rest of the world.

What are the strengths of this book? Firstly, the emphasis on the evidence base for various interventions in most chapters is very welcome. Secondly, the emphasis on physical examination is to be commended. Thirdly, the sections on rehabilitation and special testing procedures present collections of data that are otherwise difficult to pin down, and are exceptionally useful for the clinician who has to prepare a lecture incorporating any of these components.

The weaknesses of the book are partly a reflection of its exclusive American authorship. Also, the splitting off of the physical examination from the management recommendations for injuries to each body part means that for a complete understanding of any individual area one needs to consult two sections of the book. Therefore, as a general reference book for the average clinician it has significant limitations and would not be a serious threat to Brukner and Khan's text. However, for those clinicians who already have a copy of Brukner and Khan it does provide additional useful information, particularly on the areas of rehabilitation plus special tests and procedures and exercise prescription.

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Hamilton

The Development of Sports Medicine in Twentieth Century Britain

Editors: L A Reynolds and E M Tansey
Publisher: Wellcome Trust, London, 2009

This book is part of a series entitled Wellcome Witnesses to Twentieth Century Medicine and is the transcript of a seminar held by the Wellcome Trust Centre in 2007. It assembled most of the leading participants in the field including leaders such as Sir Roger Bannister, Professor Peter Sperryn (who lectured at Sports Medicine New Zealand Annual Conferences in 1983 and 1994), Professor Charles Galasko, Professor Harry Thomason and Professor Clyde Williams. The meeting was chaired by Professor Domhnall MacAuley, a previous editor of British Journal of Sports Medicine.

Sports medicine began with the early physiological studies of Professor A V Hill, who won the Nobel Prize in 1922 for his work on the physiology of muscles and exercise. Over the first half of the century athletes, doctors, scientists and coaches, all acting in their own interests, had come together and made the athlete's body into a discrete clinical entity.

In 1952 the British Association of Sport and Medicine was formed and the role of Sir Arthur Porritt, a previous Governor-General of New Zealand, is acknowledged as he gave his prestige to the organisation and, indeed, was involved with the inception of SMNZ. Two of the leading lights of the organisation were Dr John Williams, orthopaedic surgeon, and Dr Peter Sperryn, a physical medicine specialist, who both trained at St Mary's Hospital. Williams' first textbook was published in 1962 and updated and co-authored with Peter Sperryn in 1976. I feel privileged to have known both of these men. They were ably assisted by Dr Henry Robson, who edited the British Journal of Sports Medicine from 1963 to 1988.

The first sports medicine clinic set up in the UK was at the Middlesex Hospital in time for the 1948 Olympics which were held in London. Later there was a New Zealand connection via Dr Hugh Burry, ex-All Black, who set up an NHS sports clinic at Guy's Hospital. There was a further New Zealand connection via the successful 1953 Everest expedition led by Sir John Hunt culminating in Hillary and Tensing attaining the summit. The physiologist on that expedition was Dr Griffith Pugh, who also conducted important altitude research prior to the 1968 Olympics in Mexico City.

Another name mentioned was that of Dr Ludwig Guttman, the Director of the Spinal Unit at Stoke Mandeville Hospital, who founded the Paralympic movement.

The Medical Advisory Committee of the International Rugby Board first met in 1978 and its remit was to review all aspects of player safety. It is has continued this role to the present day. There was never much funding allocated for NHS sports medicine clinics; by contrast, these flourished in Europe. Therefore, even in the 1980s one witnessed the scenario of top professional footballers from Great Britain travelling to Europe for their treatment.

Training in sports medicine was available via the BASEM course run by Peter Sperryn and fellow enthusiasts from 1975 at Loughbrough. Peter later edited the British Journal of Sports Medicine from 1988 to 1995.

The London Sports Medicine Institute was set up by a grant from the Greater London Council in 1986 and ran for five years, providing lectures for GPs and a course of its own. Participants from that course could sit a Diploma awarded by the Society of Apothecaries or a Scottish Diploma awarded by the Royal Colleges. The London Sports Medicine Institute became the National Sports Medicine Institute from 1992 to 1997 but its academic programme ceased.

From a New Zealand perspective, the most significant training was available via the London hospital course set up by Dr John King. Our first graduate was Dr Tony Edwards in 1984, followed by Dr John Mayhew, Dr Dene Egglestone and Dr Ruth Highet, and then myself in 1987 followed by Dr Graham Paterson and others at a later date. The value of that course in the 1980s cannot be overestimated. It gave enthusiasts of my generation the opportunity to immerse themselves fully in the field for an entire academic year. It also forged relationships with doctors from other countries who have gone into leadership positions internationally. Finally, it exposed us to world leading thinkers of the ilk of Professor Jerry Morris, who conducted the original London bus driver and conductor studies in the early 1950s.

In the UK specialist recognition was straightforward for those doctors with Fellowship and other specialists, e.g. orthopaedics or rheumatology, but much more difficult for others. In New Zealand fortunately the Medical Council recognised the Australasian Fellowship qualification in 1989, whereas the situation in the UK is still relatively ill-defined.

In 1987 the British Olympic Medical Centre was set up at Northwick Park, with the physiology section under the aegis of Dr Craig Sharp and the clinical medicine section run by Dr Mark Harries. Harries also established a specialist network of some 30 consultants throughout the UK which is analagous to the present New Zealand Academy of Sport network in this country. Following Dr Harries' untimely death a few years ago, Dr Richard Budgett, former Olympic rowing gold medallist, took over the role and he has been appointed Medical Director of the London 2012 Olympics.

I have deliberately written a detailed analysis of this book because there are a lot of similarities between the UK and New Zealand, but also some significant differences. The similarities include the role of enthusiasts, trailblazers who got the initial recognition for the discipline. Sir Arthur Porritt helped the cause in New Zealand as well as the UK. The initial work here was done by Drs Norrie Jefferson, John Heslop and Ted Nye. Later Drs Graeme Campbell, Noel Roydhouse (editor of the New Zealand Journal of Sport Medicine for over 20 years), Matt Marshall and Dave Gerrard all played their part.

One difference would appear to be that there has been significantly less infighting in New Zealand than appears to have been the case in the UK. The hospital systems are similar, but the ability to obtain ACC funding for the treatment of sports injuries has led to the specialty flourishing in New Zealand and also partnerships with ACC in developing resources such as the Sideline Concussion Guideline.

The challenge for the next 100 years, in the UK as well as New Zealand, is for today's junior clinicians to step up and keep running with the torch.

For those readers who wish to read more, online access is available via www.ucl.ac.uk/histmed and then follow the links to publications.

A hard copy of the text is available for approximately US $10 via www.amazon.com, ISBN No. 978 085484 121 9

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European Sport Science Conference Oslo, Norway, 24-27 June 2009

Introduction

In June 2009 I had the good fortune to visit Oslo, Norway to attend the European College of Sports Science conference, thanks to support from the Wintec School of Sport & Exercise Science and the Research office. Too often we attend these conferences and then fail to pass on the new or improved ideas on to others. These summary notes have been passed around many of the sport dietitians/nutritionists and other interested parties, but Dr Chris Milne thought it worth sharing with a wider audience. I have made every attempt to interpret the findings accurately, but be aware I have incorporated some of my subjective opinions into this summary as well!

Willems M, Ponte - University of Chichester - Alanine Supplementation

Carnosine acts as an intramuscular buffer to protons and the supply of beta-alanine increases muscle carnosine content. It has been used in high performance sprint cycling and resistance training programs. Preliminary research suggests this may benefit resistance training programs.

In this study the researchers tested 24 active subjects provided 6.4 grams per day for 30 days. Therefore subjects were required to ingest 16 tablets a day!

The researchers investigated muscle fatigue (by repeated muscle contraction) and found there was no effect on maximal muscle force, but there was an increase in time to failure at 70% maximal effort. This suggests that beta-alanine may be beneficial for the performance of high intensity exercise (or resistance training). The major concern raised at question time was whether the researchers had the supplement tested independently for steroids before using it. The researchers hadn't and therefore we should be mindful that many of these supplements may, in fact, be tainted.

Ben Miller - Protein Metabolism in Endurance Exercise

Ben Miller (formerly of Auckland University) was an invited speaker asked to discuss regulators of protein metabolism for endurance exercise. He presented the current understanding that muscle growth required the stimulation of mTOR, which in turn stimulates muscle growth. He also presented the John Hawley hypothesis on endurance training; that it increases AMP kinase (AMP K) and this stimulates mitochondrial growth. So in fact resistance training and endurance exercise are opposites and one exercise type may not assist the other. However, Ben believes this model is too simplistic and endurance training may also stimulate mTOR. His hypothesis is not yet proven, but makes an interesting idea.

Philip A, University of Dundee: Antioxidants-do they enhance or suppress training induced adaptations

The current understanding of antioxidant function is that exercise increases the production of radical species. Radical species can damage cell protein and lipids, which may result in fatigue, muscle damage and immune dysfunction. Dietary antioxidants may assist the body to remove the radicals before they do damage. Whilst this theory sounds plausible, very little evidence has ever been found to suggest antioxidants ultimately improve performance. We have assumed we just can't detect the improvements. However, recent research suggests that dietary antioxidants may in fact suppress radical signalling that is essential for the adaptations to training that athletes require. Specifically it appears that the radical species are important for AMP K, mitogen activated protein kinases, and mitochondrial growth factors. Therefore, whether antioxidants enhance or interfere with training adaptations remains a controversial issue.

Kevin Tipton, University of Birmingham: Nutritional Strategies to Increase Muscle Mass

Nutritional influences on muscle mass have received a great deal of interest over the past few years. Protein and particular amino acids are thought to be the most important nutrient for muscle building. However, how much, when to consume and in what form is still under scrutiny. Protein prior to and after training assists muscle growth, with only 6 grams of essential amino acids needed, and more than 10 grams add no additional benefits. Excess protein is simply oxidised for energy or excreted in urine.

Whilst it was accepted that the negative health consequences of excess protein have been overstated, it should be remembered that athletes may be consuming protein at the expense of other nutrients, such as carbohydrate. Recent research suggests milk ingestion stimulates muscle anabolism following exercise and this effect is superior to soy protein. Also, whole milk was superior to skim milk, which was superior to whey protein. These results may suggest two things; 1) protein in foods works just as well as that in supplements and 2) animal proteins seem to engender a superior anabolic response following resistance training.

L-arginine

Oral administration of l-arginine is not likely to increase muscle contraction because muscle uptake is tightly regulated and transporters responsible for moving it into the cell are down regulated with supplementation. Take home message.... more is not necessarily better!

Whey Protein/CHO ±HMB Supplementation

The researchers wanted to investigate the impact of adding HMB to whey protein/ CHO on a 12 week resistance training program.

One subject increased fat mass by 4kg, and found no muscle gains at all, however all other subjects increased fat free mass and decreased fat mass. This suggests that not all people respond the same way to a nutritional intervention. It was acknowledged that the subjects were untrained and did complete a very hard resistance training program.

Overall, HMB had NO additional improvement in strength over and above the whey protein/CHO supplement alone.

Magnesium Supplementation in Elite German Athletes

In Germany, magnesium supplementation is the most common form of any supplement an athlete will take (quite a different story in New Zealand), in fact 78% of those surveyed by the German Research Centre of Elite Sport supplemented with magnesium. It was acknowledged by the presenter that magnesium losses in sweat are small, but may become accumulative on repeated exercise sessions.

Low dietary intake was more common in females, however there were no athletes with dietary intakes less than the recommended dietary intake, and this did not include the supplement intake. This suggests the supplement is simply not needed.

Magnesium deficiency was defined as less than 0.6mmol/L and 95% of athletes had adequate serum magnesium, however serum magnesium is a poor indicator of true magnesium intake.

One point that was raised was that exercise needs to be standardised if serum/plasma magnesium is to be determined.

Asker J - University of Birmingham: Nutrition to increase fat oxidation

Asker presented an overview on nutritional strategies that may increase fat oxidation. These are summarised as:

- Fat oxidation appears to be higher following a low GI meal
- Walking and running appear to be more beneficial than cycling (in recreational subjects)
- Fucoxanthin-derived from sea kelp, good results in mice, but quantities needed are too large to consider as an option for humans
- Caffeine-well documented to increase fat oxidation, short term ... probably not sufficient to bring about significant weight loss.
- Green tea extract (ECGC), positive results regarding fat oxidation. Unknown mechanism. Dose used in studies varies from 400-1200mg ECGC per day.
- L-carnitine: the major drawback with carnitine is getting into the cell, given the gradient difference between the cell and blood. However, supplying with glucose to stimulate insulin assists this process. Problem with that is the additional CHO will negate any weight loss effect from the carnitine.

Zinne C - German Sport University, Cologne: Bicarbonate Ingestion

Researchers investigated the use of 0.3mg sodium bicarbonate/kg body weight or 2g placebo taken 90 minutes before a high intensity all out cycling effort. The athletes completed a 10 minute warm up and then 4 x 30 second efforts. The mean power in the first 2 repetitions were similar, however the power on the third and fourth was higher on bicarbonate.

Carr A J - Australian Institute of Sport: Bicarbonate ingestion -Symptoms

Athletes tend to use ingestion protocols that vary widely, which may impact on the ability to achieve the best results. The researchers compared bicarbonate tablets and powder with either 7ml/kg or 14ml/kg taken in over 30 minutes. Both tablets and powder resulted in an increase in bicarbonate in the blood, and levels were highest at 120-150 minutes post ingestion. The symptoms were worse when the smaller volume of fluid was consumed (7ml/kg) with the tablets, and this occurred 90 minutes after consumption. The least symptoms occurred when the bicarbonate was co-ingested with a meal.

Hopkins W & Vandenbogaerde - AUT: Caffeine on Swim Performance

Using fancy statistical mixed linear modelling for monitoring athletic performance, the researchers were able to determine in elite swimmers, a 100mg dose of caffeine enhanced performance by 1.2% in time trials and 1.4% in competition, with each additional 100mg dose reducing the benefit slightly.

Interesting to note that a dose of 100mg for the average weight of a swimmer is below the currently recommended 3mg/kg body weight. This suggests research is still warranted regarding optimal caffeine dosage for elite athletes.

Milasius K - Vlnius Pedagogical University: Tribulus Supplement

Tribulus Terrestris is a popular dietary supplement used by many athletes. It is a herb that has been found to influence hormonal testosterone levels, immunity and endurance capacity. The researchers tested these claims on 32 athletes, putting half of them on 3 capsules (Optimum Nutrition, USA) per day for 20 days, the other half on placebo.

The intervention group displayed a decrease in various immune function parameters, however the concentration of blood testosterone increased during the first 10 days, and then stabilised. Increases in testosterone using this supplement have previously been reported in the literature, but not widely.

Will Hopkins - Statistics

It was an interesting exercise to fly all that way to attend a statistics lecture from Professor Will Hopkins (AUT). He spoke for a few hours on p-values, the importance of presenting research results as the standard deviation, not standard error of the mean and confidence limits. These recommendations provide the reader with an appreciation of the magnitude of the results. This is an important concept for sport nutrition given the sum of information out there, of which a fair proportion may not be presented as it should be.

We went on to discuss more complex topics such as **normality**. His thoughts were that testing for normality prior to analysing results was a waste of time, as it all depends on your sample size as to whether your sample group appear normally distributed. We discussed sample groups that are not **uniform** in their responses and therefore may require a more sophisticated analysis than simply comparing changes in the mean.

Will then touched on his **mixed modelling**, which may be used when each data point is not independent, or when requiring a predictor from a large array of numbers. Mixed modelling differs from linear modelling by allowing numerous co-founding factors. SPSS also does mixed modelling.

Conclusion

Oslo was quite an amazing place given it has been reported as the ugliest city in Norway. The night time light was unnerving as it never really got dark and the locals made use of this by heading out to the cafes at all hours. The cost of food and alcohol was astronomical, but everything else was surprisingly affordable. The old buildings and history surrounding many of these European cities is something we lack here in New Zealand and worth a look.

The ECSS conference had a good mix of internationally recognised speakers without the overwhelming crowds of ACSM conferences (so I'm told), and next year's conference is in Turkey.

Andrea Braakhuis
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PATIENT ADVICE SHEET

LOCAL CORTICOSTEROID INJECTIONS

Introduction

Your doctor has recommended you have a local steroid injection to help treat your painful joint, muscle or tendon problem. This simple guide answers some of the common questions asked about these injections. If you have any further questions you should ask your doctor.

What are steroids?

Steroids are a large group of drugs, with many different properties. There are three main types:

1. ***Corticosteroids;*** powerful anti-inflammatory drugs used to treat asthma, arthritis and other conditions.
2. ***Male sex hormones;*** androgenic anabolic steroids. These are used to treat androgen deficiency and male infertility. Some people use them for "bulking up". They are **BANNED DRUGS** and have no place in injury treatment.
3. ***Female sex hormones;*** eg, estrogens, progestagens. These are used "in the Pill" for contraception and also as Hormone Replacement Therapy (HRT) after menopause.

What do corticosteroid injections do?

They cause a powerful anti-inflammatory action. This should lead to:

1. Removal of debris from the injury site.
2. Reduction of pain and swelling.
3. Greater range of motion.
4. Property alignment of scar tissue as injury repair takes place.
5. A return to sport and other important life activities.

What injuries are they used for?

1. Inflamed joint with excess fluid (effusion).
2. Inflamed bursa (fluid sac around a joint).
3. Inflamed tendons and tendon sheaths.
4. Soft tissues that are being pinched (impingement).
5. Frozen shoulder.

They have been used for these injuries for over 40 years, with very good results in most cases.

BENEFITS

Relieve pain.

Reduce swelling.

Improve healing.

Allow return to full activity.

RISKS (SIDE EFFECTS)

Common (up to 10%)

Dimpling and loss of skin pigment around the injection site. This can be permanent. It is mainly a problem with tennis elbow injections, and those around the Achilles tendon.

Uncommon (up to 5%)

1. Post-injection flare. A few hours after the injection you may get a worsening of pain and swelling. This usually settles in a day of two with ice packs and tablets. Take 3 x 300 mg Dispirin dissolved in half a glass of water every 3-4 hours. Alternatively, use Paracetamol 2 x 500 mg every 4-6 hours.
2. Skin flusing, especially of the face, may occur for up to a few days. This needs no specific treatment and will settle with time.

Rare (less than 1%)

Tendon rupture - avoid heavy exercise of the injected part for at least a week.

Very Rare (much less than 1%)

Infection can very rarely complicate any injection. If the area around the injection site becomes hot and red, contact the doctor who performed the injection for further advice.

In women only; your next period after the injection may be heavier than normal. This is probably due to temporary hormonal changes related to the injection and does not usually require further investigation. It usually occurs in women over the age of 30.

In people with diabetes; you may notice a rise in your blood sugar level for a few days after the injection. Some people may need to use extra insulin.

After your injection:

- Especially if you have had an injection that includes local anaesthetic, be very careful not to stress the area.
- People doing light or office work can continue at work. If you are doing heavy work you should check with your doctor.
- Do **NOT** attempt any heavy lifting, eg, moving a heavy table or doing heavy weights.
- After several days you may return to light training that avoids stressing the injected area.
- At about the one week stage you can plan an ongoing recovery programme in conjunction with your doctor and physiotherapist.
- About one month after the injection you should see your doctor for a follow-up check.
- **REMEMBER**, the injection is not a cure. It works best as part of a total treatment plan with progressively increasing training over several weeks.
- For athletes subject to drug testing, you must complete a Declaration of Use (DOU) form. This can be completed electronically. Go to the website for DrugFree Sport New Zealand - www.drugfreesport.org.nz - and follow the links.

Reference; *A Practical Guide to Joint and Soft Tissue Injections*, James W McNabb, Lippincott Willliams and Williams, Philadelphia, 2005.

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