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Best of British

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Injury surveillance in elite New Zealand track cyclists

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Injuries in a senior amateur rugby union team over two competition seasons resulted in a ratio of 1:5 witnessed to unwitnessed concussions

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How I Treat: Achilles Tendinopathy

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Sports Medicine New Zealand - The first 50 years

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Mecide, cur ate ipsum (Physician, heal thyself) Part I

STUART ARMSTRONG

After my previous editorial on low carbohydrate diet, weight loss and its effects on athletic performance. I was hoping to have a good summer season of running. Unfortunately, in my first race of the season when I was 30 km in, I decided to push myself up a hill to try and catch the leader when I heard a sickening tear in my Achilles. My race was over and it was time to have a good think about rehabilitation of this injury and progressing back to running.

An Ultrasound scan the following day confirmed a 50% tear of my Achilles tendon. For those of you aware of my previous dubious history of a self-administered cortisone injection this was on the contralateral side! But that is a story for another day.

An acute injury like this really brought to the forefront of my mind the acute management of sporting injuries and the evidence around this. We all know the maxim around the management of acute injuries is Rest, Ice, Compression and Elevation.

Rest - Having the benefit of my medical knowledge I was able to stop running immediately and there were no thoughts of pushing on through the pain after hearing that tear. Due to the bush location of my race I did hobble a couple of kilometres to get to an aid station and home. Initially I like to be limited by pain and I used this to dictate the amount of activity. I was able to move around without going into any active plantar flexion and my rest was definitely "relative" in nature. I used crutches for a couple of days, but avoided going in a moon boot. There are a couple of reasons I don't like moon boots, but the main reason is due to the significant muscle wasting you see after being in a moon boot. Previous studies have shown that muscle loss starts within 24 hours of rest or immobilisation and 1 week of bed rest leads to on average a 3.2% loss in muscle cross sectional area.[1] A big factor in recovering from this injury would be building up calf strength and I was keen to minimalise its loss. 1 week after the injury I was able to start walking with active plantar flexion, albeit for a limited duration.

Ice – I'm not a great fan of ice in acute injuries, a controversial statement I know, but there is no great evidence that it does anything useful long term. There is no argument that it can relieve pain and decrease swelling, but my argument is that pain and swelling are there for a reason. The pain is there to stop you using the injured area and the swelling is there to help kick start the healing process. A large number of studies have looked at the benefits of ice in acute muscle injuries and they are summarised in a chapter of the excellent book Evidence based Sports Medicine by MacAuley and Best. In short the studies are of poor quality with no consistent methodology and fail to show any improvement in recovery beyond pain relief and decreased swelling.

Compression – As a young sports medicine registrar I did my research on compression socks and recovery following marathon running.[2] There is a volume of research

on this topic and my personal belief is that compression can aid recovery following extreme exertion in the same way that a gentle warm down and keeping the body moving throughout the day can. In essence, it is useful for the person that finishes a hard training session or race then hops in the car to drive to work and doesn't move for the rest of the day. I look at compression as the time poor person's recovery aid. In the setting of acute injuries there is a relative paucity of good clinical research. The majority of studies are in healthy young males and using compression for prophylaxis. There are not a great number of good studies looking at the acute benefit of compression post injury. There is however good evidence that the use of ankle compression following ankle inversion injury as you start to mobilise is good at preventing further inversion injuries at this vulnerable time.[3]

Elevation – Interestingly there is not a single randomised controlled trial on the use of elevation for the treatment of acute injuries. 4This treatment does make sense to me from the point of view of enforced rest. You are not able to walk around on your lower limb injury if it is elevated. The other rational for elevation is to reduce swelling. Again I'm not sure about this as the swelling is there because of all the inflammatory mediators and factors that are starting the healing process.

In conclusion, although we all are well versed in the maxim of RICE there is not a great deal of evidence for this and although it does help the pain and swelling the question is whether we actually want to be modifying these factors in the very acute stages of an injury. Especially if decreasing pain and swelling lets our injured athlete ignore their injury and cause more trauma.

On a personal note, I am now back running and have built up to a 10 km run this morning. I plan to take you further through the details of my rehabilitation in the next editorial.

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The changing face of sports medicine: Who is the primary care provider for the athlete?

Sports medicine is a multidisciplinary branch of medicine with close cooperation between the different subspecialties. Sports medicine doctors and physicians, physiotherapists, podiatrists, chiropractors, dietitians, massage therapists, psychologists and others work closely together on behalf of their patients.

Until the recent emergence of the sports physician, the sports doctor, usually a general practitioner with some additional training or experience in sports medicine, coordinated the support team.

The sports medicine GP was the first point of contact for the athlete. This "sports doctor" made a provisional diagnosis and where appropriate referred the patient on to professionals within the team. An important task of the sports doctor was also the medical (health) screening of the athlete at regular intervals.

There have been two significant developments in the last decade which have had an impact on this working model. The first was the decision by ACC to give patients the choice to have direct access to the physiotherapist without requiring a doctor's referral. The second was the emergence of the sports physician, the sports medicine specialist.

The transition from the sports medicine GP to the sports physician has been gradual as the first sports physicians were already practicing sports medicine GP's. They are now gradually outnumbered by young doctors who enter the sports physicians training programme straight out of medical school. They miss the unique training, knowledge and practical experience of the GP, which is relevant for any form of primary care.

The sports physicians are positioned as secondary health care providers in line with other medical specialists. Supported by the ACC system they tend to see athletes on a referral basis for a second opinion in regards to injury diagnosis and treatment.

A consequence of the emergence of the sports physician has been the unforeseen marginalisation/decline of the sports medicine GP. There is a general perception that their place has been taken by the sports physician. The gap left in the provision of primary medical care by the sports medicine GP is now filled by the physiotherapist who, supported by the ACC system, has become the first point of contact for the injured athlete.

While the GP is trained in all aspects of primary care the physiotherapist is generally the expert in the more narrow field of treatment of injuries and ailments of the musculoskeletal and nervous system. In my opinion it is doubtful that this new model in sports medicine, whereby the physiotherapist is the first point of contact for the injured athlete, is as good as or better than the previous model.

The physiotherapist will need some serious additional training in regards to primary care, including differential diagnosis, appropriate use and interpretation of radiological imaging and knowledge of the skills offered by other professionals in the support team, to be able to "compete" with the level of care provided by the primary care sports doctor.

The alternative is to 'bring back' the primary care sports medicine practitioner. How this can be done is a matter for wider discussion. I am in the autumn of my career and this letter to the editor is not meant as criticism but more as an observation, based on years of experience. This letter's main aim is to generate further discussion around best practice in primary sports medicine care.

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BEST OF BRITISH - HIGHLIGHTS FROM THE BJSM

CHRIS MILNE

HIGHLIGHTS FROM THE BJSM JANUARY TO JUNE

This instalment of Best of British incorporates articles from the first few months of 2018.

The January issue was published in conjunction with the International Olympic Committee and started with an article provocatively entitled, 'Are Athletes Psychologically Ready for Sport Following a Concussion?'[1] The authors from Montreal and Salt Lake City have noted that there is a paucity of studies relating to psychological readiness to return to sport following a concussion. They have noted that concussed athletes experience psychological concerns regarding return to sport, possibly due to uncertain recovery times, symptoms associated with concussions, such as headaches, nausea and poor concentration, as well as the growing awareness of the potential long term cognitive impairments associated with concussion. It is already known that athletes who are psychologically under prepared when returning to sport report a heightened fear of reinjury. These authors make a plea for greater awareness of the importance of psychological readiness before concussed athletes return to the sporting arena.

Segregation in sport is a controversial issue. Arne Ljungqvist, former chairman of the Medical Commission for the International Olympic Committee, has published a one page summary on this issue.[2] The IOC Medical Commission convened a group of experts to a consensus meeting in Stockholm in October 2003, and there was a follow up meeting held in January 2010 in Miami. However challenges to the rules set down by the International Amateur Athletic Confederation, particularly by a female sprinter from India, named Dutee Chand had thrown further spanners into the mix. The issue is compounded by that of transgender athletes, exemplified by New Zealand weight lifter Laurel Hubbard, who competed in the recent Commonwealth Games in Brisbane. My impression is that there is a significant group of natural born female athletes who resent their competition being open to those athletes who were born male. However in today's climate, these athletes are somewhat reluctant to speak out. Only time will tell how this issue plays out.

Fractures associated with ACL injury need to be taken seriously. Ali Geurmazi and colleagues have provided a succinct article summarising the issues.[3] The most well-known fracture is the segond fracture which is regarded by most experts as pathognomonic of an ACL injury. However there are other less commonly seen fractures which should be treated actively to minimise their long term impact on morbidity.

Paralympic athletes have an increased risk of illness compared with their able bodied cousins. This is because of the frequency of background medical conditions that have led to their paralypmic status in the first place. A study by Derman et al following the Rio summer Paralympic Games found that the rate of illness was lower than that reported for the London 2012 Summit Paralympic Games.[4] The sports with the highest risk were wheelchair fencing, paraswimming and wheelchair basketball. Respiratory system, skin and digestive system

were those most affected by illness.

Issue 2 had some important articles on the athlete's shoulder. Chief amongst these was a review on scapula dyskinesis. Hickey and colleagues reported that scapula dyskinesis increased the risk of future shoulder pain by 43% in asymptomatic athletes.[5] They conducted a meta analysis of five studies including 419 athletes. The results of this meta analysis indicates to me that we should be including an assessment of scapula dyskinesis in our pre participation and medical screening otherwise referred to as the Periodic Health Evaluation. In addition it should be a routine part of evaluation of any athlete with an injured shoulder.

ACL injuries can have a devastating impact on future sporting participation. Courtney Lai and colleagues conducted a meta analysis of 24 studies and analysed the findings.[6] They found that return to sport among the elite athletes following ACL reconstruction was 83% and the graft rupture rate was 5.2%. Most elite athletes took between 6 and 13 months to return to sport, which is less time than has been previously been reported among non elite athletes. My take-home message would be that these data should be shared with our elite athletes who are contemplating ACL reconstruction after sustaining an ACL rupture.

The third issue in February 2018 included a useful editorial regarding running and osteoarthritis headed up 'Running Causes Knee Osteoarthritis: Myth or Understanding?'.[7] The author William Roberts, a senior USA sports physician, concluded the answer to the question, does running cause knee osteoarthritis, has several layers. There is no increased risk in running for fitness or recreation, but there does seem to be a small risk for knee OA in high volume, high intensity runners. Once again this should be shared with our patients.

Can exercise help retain cognitive function in adults older than the age of 50?[8] As an adult over the age of 50, I took a keen interest in this article. Northey and colleagues conducted a meta analysis of 39 studies including 12,820 patients. Analysis showed that physical exercise improved cognitive function. Interventions of aerobic exercise, weight training, multi component training and Tai Chi all had significant benefit, somewhat reassuring for this aging codger, certainly a motivation to keep active.

Dietary supplements are commonly touted as being helpful for osteoarthritis. Liu and colleagues conducted a systematic review and meta analysis on this topic.[9] They found 20 supplements investigated in 69 eligible studies. Of these, only 7 showed clinically important effects for pain reduction at the short term. Substances included collagen hydrolysate, passionfruit peel extract, curcuma extract, boswellia extract, curcumin, pycnogenol and L-carnitine were the most effective. An important omission from this list is turmeric, which is currently being promoted by our erstwhile retired All Black captain Ritchie McCaw, but which fails to feature among the finishers. The authors note that the most widely used supplement e.g. glucosamine and chondroitin do not provide a clinically important effect on osteoarthritis. They note that the current data are of low quality, and recommend further study.

Tennis is a widely played sport and has many health benefits. Babette Pluim and colleagues provide a useful summary of the health benefits of tennis plus a summary infographic.[10] This might be worth putting up at your local

tennis club or in your consulting rooms for perusal by your active patients.

Estimation of body composition in elite athletes is a fraught area. Peter Sonksen, a senior endocrinologist, provides an excellent overview of this area.[11] In particular he considered the effect of growth promotors such as growth hormone, anabolic steroids, beta agonists and insulin. There are some interesting historical photographs included in the article.

Overhead athletes have significant problems with their shoulders. Alexis Wright and colleagues provided a systematic review of exercise prescription for overhead athletes.[12] They found that overall, evidence for exercise interventions in this group is dominated by expert opinion, i.e. only grade D evidence. There was great variability between the exercise approaches suggested by experts and the overall level of evidence was low. The strongest available evidence supported the use of single plane, open chain, upper extremity exercises performed below 90° of abduction, plus closed chain upper extremity exercise. Worth knowing for any experienced physiotherapist working with such an athlete.

Osteoarthritis of the knee is pretty common in our older folk. Kathryn Shaw and colleagues studied the effects of insoles on gait mechanics and people with OA of the knee.[13] They found that lateral wedge insoles produced small reductions in knee adduction angles and external movements plus moderate increases in ankle eversion. The addition of an arch support to a lateral wedge minimised ankle eversion change. They noted a paucity of available date on other insole types and suggested further research, surprise, surprise.

The March issue threw the spotlight on Australian research in sport and exercise medicine and the editorial was headed up unapologetically patriotic.[14] Certainly Australians have punched above their weight in this area. One of the senior researchers is Bill Vicenzino from University of Queensland. He was a senior author in a paper entitled, Patella and Achilles Tendinopathies are Predominantly Peripheral Pain States. They appear to be related to loading of tendons without significant features of central sensitisation. Therefore a relatively mechanistic approach as per the Alfredson concentric and eccentric regime is likely to bring about positive results.

Arthroscopic surgery for degenerative knee arthritis and meniscal tears is a controversial area.[15] Reed Siemieniuk and colleagues convened an expert panel and the results were published in the BMJ in June 2016. They found that among patients with a degenerative medial meniscus tear, knee arthroscopy was no better than exercise alone. The panel made strong recommendations against arthroscopy for degenerative knee disease.

Clinical practice guidelines have been in use for three decades now. Lin and colleagues looked at the overall quality of these guidelines for musculoskeletal pain.[16] They evaluated 34 clinical practice guidelines, mostly for osteoarthritis or low back pain. They found low applicability and a significant compromise of editorial independence. In addition most guidelines were developed by high income countries or in collaborations involving high income countries. Therefore their applicability in low income countries may be rather limited.

Issue 6 published in March highlighted the Sports Medicine New Zealand and the Sport and Exercise Physiotherapy New Zealand combined Ann Cools

roadshow. Ann is an internationally renowned shoulder researcher and is coming to New Zealand for a series of workshops in July. You should try and get to one of these if they are in your vicinity.

Achilles tendinopathy is a term that has been in use for a couple of decades now. The prevailing dogma is that minimal inflammatory features are characteristic of the condition. However Dakin and colleagues from the University of Oxford have published a counter review.[17] They found that tissue and cells derived from tendinopathic and ruptured Achilles tendons show evidence of chronic non resolving inflammation. They further state that the energy storing Achilles shares common cellular and molecular inflammatory mechanisms with functionally distinct rotator cuff positional tendons. They go on to say that strategies that target chronic inflammation are of potential therapeutic benefit for patients with Achilles tendon disease. Certainly a provocative article and well worth reading. It provides some rationale for the use of anti inflammatory tablets or gels in patients with Achilles tendinopathy, in addition to the established concentric and eccentric strengthening regime, or an isometric regime for in season management.

Can protein supplementation build bigger muscles? Morton and colleagues studied this topic and found that dietary protein supplementation significantly enhanced changes in muscle strength and size during prolonged resistance training, i.e. weight training.[18] They found that protein intakes at amounts greater than 1.6g/kg/day do not add any additional benefit.

Extracorporeal shockwave therapy is used in a variety of centres for lower limb conditions. Korakakis and colleagues conducted a systematic review of the topic.[19] They found low level evidence that shockwave therapy was comparable to eccentric training but superior to a wait and see policy at four months in mid portion Achilles tendinopathy. It was noted to be superior to eccentric training at four months in insertional Achilles tendinopathy. It was less effective than a cortisone injection in the short term but superior results in the mid and long term for patients with greater trochanteric pain i.e. gluteus medius insertion pain +/- bursitis. I do not personally use this therapy but occasionally refer patients for it and it is good to see the level of evidence that has been accumulated for its use. More work is needed in this area.

The last couple of articles included an infographic with the intriguing title of How to Maximise Your Golf Performance.[20] I have already sent this to my esteemed colleagues Tony Edwards and Graham Paterson as they take their golf more seriously than I do, which would not be difficult. The final article was entitled Golf Spectating and Health and this included the interesting statistic that the spectators at the 2014 Ryder Cup collectively walked four times around the world.[21] The challenge was to encourage spectators to become more active after the event.

That is all for this issue. Look out for more intriguing data from later issues of BJSM.

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ORIGINAL RESEARCH

Injury surveillance in elite New Zealand track cyclists

Establishing the baseline incidence and prevalence of injury and its effect on training and competition for elite New Zealand cyclists

HELENE BARRON, DUNCAN REID, BRUCE HAMILTON

ABSTRACT

Introduction: Injury surveillance is an essential component of elite sport. Little data is available on injury rates in track cyclists, with the majority of cycling research focussed on road cycling, and suggesting cyclists are at highest risk of overuse knee, back and neck injuries, and acute injuries involving the shoulder/clavicle, lower back and knee.

Aim: To establish the baseline incidence and prevalence of injury, and its effect on training and competition for elite New Zealand track-cyclists.

Study Design: Prospective, longitudinal study

Setting: The "Home of Cycling", Avantidrome, Cambridge

Participants: 33 members of Cycling New Zealand's elite track squad, comprising 17 males (mean 22.71, SD: 4.45), and 16 females (mean 21.5, SD: 4.82).

Interventions: Participants completed two baseline questionnaires detailing current and past injury status, current training volume, and other baseline characteristics, then completed a weekly online self-reporting injury survey for 52 consecutive weeks. Outcome measures were injury incidence and prevalence, with breakdown for gender and squad

Results: Mean compliance for survey completion for the duration of the study was 75.41% (SD 14.8).
64% of participants sustained an injury, with the most common injuries seen in the lower back, hip/buttock region, and the knee. Injury Incidence was 4.9 injuries per 1000 training and competition exposures. For all injuries sustained (53 body parts injured from 44 events), the injury incidence was 5.9 per 1000 exposures. Injury prevalence for the 52 week study was 11%. Point prevalence ranged from one injury per four-week block to seven (mean 3.38, SD 1.80). Further analysis of results reviewed injury location, nature, and effect on training. No significant relationships were found between new injury and squad, gender, previous injury, years in sport, nor between new injuries, injury frequency, or number of treatments.

Conclusions: 64% of participants sustained an injury during the period of the study, with the lower back, hip/buttock region, and knee injured most. However, injury incidence and prevalence was low with rapid return to training and competition. Further research with greater detail around the intensity, nature and load of specific training sessions is required.

Key Words: Track cycling, Injury surveillance, Injury incidence, Self reported injury

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INTRODUCTION

Track cycling is one of the targeted sports for High Performance Sport New Zealand (HPSNZ) and the elite New Zealand track cycling team is currently performing well on the world stage with a tally of 35 World Championship medals in the last 11 years.[1] The goal of the performance health team working with track cyclists, should be to minimise injury risk and optimise performance. However, little published research is available on track cycling injury surveillance, with most research focussed on road cycling, providing minimal specific guidance for key focus areas for the track cycling cohort. Current literature suggests that road cyclists are at higher risk of knee, lower back and shoulder injuries,[2-8] and a summary of these study results is outlined in Table 1. Road, track cycling, and cycle ergometer biomechanics are comparable as the general pedal stroke is consistent,[9] however, track cycling predominantly involves a lower aerodynamic posture, a fixed gear bicycle, and entails a number of different events ranging from 10 second sprints to hour long bunch races[10-13]. When taking into account these factors, and the training and competition load a track cyclist is exposed to (road, track, ergometer, gym/resistance training), it is important to consider that they may have differing injury incidence and prevalence to road cyclists. This study aimed to provide baseline incidence and prevalence of injury, and its effect on training and competition for elite New Zealand track-cyclists.

METHODS

Study Design and Participants

This research was a prospective longitudinal descriptive cohort study of elite New Zealand track cyclists conducted over a consecutive 52-week period during the 2015-2016 track cycling season. Ethical approval was gained from the AUT Ethics Committee (AUTEC) for all components of this research (Reference number 15/108). The type and amount of cycling training and competition undertaken, as well as detail on any injuries which occurred during the 52-week period were collected. All members of the NZ elite track cycling squad were invited to take part in the study. Those who consented to participate completed two separate

Table 1
Cycling injury surveillance studies

Study (Author & Year)	Participants	Study Design	Results
Barrios et al (2015)	Male professional cyclists n=131 Historical group n=65 male riders surveyed between 1983-1995 Contemporary group (currently competing) n=66 interviewed 2003-2009, reporting injuries from 2000-2009.	Retrospective descriptive epidemiologic survey	Injury Profile: HG: 86 injuries cf CG: 141 injuries. Injury/Cyclist ratio: HG: 1.32, CG: 2.13 TI: HG: 39.5%, CG: 53.9% (significant increase, p<0.05) Severe traumatic lesions decreased significantly (p<0.01) HG:49.9%, CG 10.5%. Patellofemoral pain decreased (p<0.01): HG: 28.8%, CG: 6.1% Muscle injuries substantially increased (p<0.01) HG: 13.4%, CG: 44.9% Injury rates: HG: 0.104 per year per cyclist, and 0.003 per 1000km training and competition CG: 0.287 per year per cyclist, and 0.009 per 1000km training and competition Summary: CG double risk of TI cf HG but those lesions less severe. Anatomical region: In both HG and CG >half injuries occurred in the upper extremity or shoulder girdle (HG 52.8%, CG 61.7%) OI: CG more muscle injuries than HG (CG: 44.9%, HG: 13.4%) AIS severity scale: Severe lesions decrease. (p<0.001) from HG: 49.9% to CG: 10.5% OI Location: HG: Knee 63.4%, Muscle 0 Recorded, Spine 13.4%, Other 23.1%; CG: Knee 36.9%, Muscle 21.5%, Spine 29.2%, Other 12.3%
Palmer-Green et al (2014) (Only abstract – not full article – published at time of this research)	British national team cyclists across BMX, Mountain-bike, Track sprint, Road/track endurance n=61 cyclists (16 female, 45 male)	Longitudinal prospective cohort study (surveillance)	95 injuries lasting average of 16 days, 35% of squad with 1 injury per season PREVALENCE: Training injuries 30% (n=77), Competition injuries 11% (n=18) SEVERITY: Training injuries 24 days missed, Competition injuries 14 days missed Overuse injuries: Lower back 29% (n=17), Knee 18% (n=9), Shoulder 35% (n=23) TIME LOSS: Acute injuries: 32 days missed Lumbar spine injuries with weight training in sprinters and endurance road cycling shoulder/clavicle and knee lead to greatest number of training days lost

KEY: HG: Historical group, CG: Contemporary Group, AIS severity scale, BMX Bicycle motocross, TI: Traumatic Injuries, OI: Overuse injury, LBP: Low back pain

Table 1 Continued

Study (Author & Year)	Participants	Study Design	Results
Clarsen et al (2015)	5 sports monitored including: n=98 cyclists (from 5 professional teams - 84 males, 14 females)	Prospective Cohort study (using a panel design)	Cyclists – 92% response rate Prevalence: All overuse injuries (with 95% C.I.) Knee 23 (17-28) Lower back 16 (12-20) Shoulder 7 (4-10) Thigh 8 (7-9) Substantial overuse injuries (with 95% C.I.) Knee 8 (7-9) Lower back 6 (4-7) Shoulder 1 (0-1) Thigh 4 (3-5)
Steffen et al (2012)	Athletes competing at Beijing and Vancouver Olympic games – of which n=518 cyclists	Injury surveillance – prospective cohort study	518 cyclists 30 injuries – 22% of all Olympic injuries 5.7% of participating athletes
Clarsen et al (2010)	Professional male road cyclists from 7 professional teams n=109	Descriptive epidemiological cohort study (cross sectional, retrospective)	94 injuries in 63 athletes, 45% in back, 23% knee TIME LOSS INJURIES: 23: Knee 57%, Lower back 22%, Lower leg 13% PREVALENCE: LBP 58% with 19% seeking medical treatment, Anterior knee pain 36% with 19% seeking medical treatment MISSED/MODIFY COMPETITION: Knee 9%, Lower back 6% MISSED/MODIFY TRAINING: Knee 27% TIME LOSS INJURY: Knee 57%, Lower back 17%, Lower leg or Achilles 13% 1 career ending lower back injury Average time loss of 13.5 days per injury (excl. career ending back injury)

KEY: HG: Historical group, CG: Contemporary Group, AIS severity scale, BMX Bicycle motocross, TI: Traumatic Injuries, OI: Overuse injury, LBP: Low back pain

baseline surveys followed by 52 consecutive weekly injury surveillance questionnaires. Any athlete who withdrew from the squad between the time of selection and the beginning of the research project, who lost their place in the squad, or who had no phone or computer access was excluded from the study.

Injury Surveillance Tools

Cyclists completed two initial surveys, one to provide baseline characteristics including age, self-reported years in sport, and current and previous injuries, and the second (in the form of the validated 15 Oslo Sports Trauma Research Centre (OSTRC) overuse injury questionnaire) to capture the presence of any overuse symptoms at baseline. The OSTRC overuse injury questionnaire has three sections relating to the knee, the lower back, and the shoulder which are areas described in the current literature as commonly injured in cycling.[2-8] This tool has also been utilised in other cycling injury surveillance studies[4,5] and therefore its use here makes comparison of studies more straight forward.

Injury surveillance for this study was achieved utilising the Programme for Injury and Illness Surveillance (PILLS) tool which was created and introduced by HPSNZ. This survey is completed using an online application, allowing for a consistent method of gathering self-reported athlete data, and was completed weekly for 52 consecutive weeks.

Outcome Measures

Variables recorded were injury occurrence, cause, nature of the injury (acute, overuse and recurrent injuries as defined by Finch & Cook[17]), number of training or competition sessions missed or modified due to injury, and the number of treatments received.

Injury incidence was defined as the number of new injuries per 1000 training and competition exposures.[18-19]

Injury prevalence was calculated by dividing the number of athletes reporting injuries with the number of completed questionnaires received, and was recorded as a percentage.[16,20]

RESULTS

Thirty three of the 41 (81%) eligible athletes consented to participate. Mean compliance for weekly survey completion for the year was 75.41%

(SD 14.8). Baseline descriptive data from the 33 subjects is outlined in Tables 2 and 3. One female subject was significantly shorter than her team mates (154.40cm - >2SD) but all other descriptive results indicated no significant outliers, with normal distribution of remaining data. There was homogeneity of variance as assessed by Levene's Test for Equality of Variances, therefore, an independent t-test was run on the data. No significant difference was found between males and females in terms of age, but there was significant difference in height and weight ($p<0.0001$). Chi-square tests found no statistically significant differences between cycling disciplines (sprint versus endurance) in male or female subjects ($X2 =2.6$, $p=0.1$).

Table 2
Subject demographics at enrolment

Variable	Total (SD n=33	Male (SD) n=17	Female (SD) n=16	p-value
Mean age (years)	22.12 (4.6)	22.71 (4.5)	21.50 (4.8)	0.46
Mean height (cm)	175.57 (7.4)	180.59 (4.5)	170.24 (6.0)	0.0001
Mean weight (kg)	74.2 (11.8)	81.67 (10.4)	66.26 (7.2)	0.0001

Note SD = standard deviation. * = significant at p=0.05

Table 3
Participant Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
Average training hours per week	33	13.00	35.00	21.6	6.4
Years in cycling (at any level)	33	2.00	22.00	8.3	4.1

Table 4
Injury count, and breakdown for squad ad gender

	One body part injured	Two or more body parts injured
Injury count	**39**	**5**
Sprint (n=13)	22	1
Endurance (n=20)	22	4
Male (n=17)	**24**	**2**
Female (n=16)	**20**	**3**
Men's endurance (n= 8)	11	2
Women's endurance (n=12)	11	2
Men's sprint (n=9)	13	0
Women's sprint (n=4)	9	1

Initial OSTRC overuse survey results indicated that 15% of participants reported shoulder symptoms at baseline, 39% reported Lower back symptoms, and 21% reported knee symptoms (illustrated in Figure 1).

During the study five participants were excluded as they were decarded from the NZ cycling squad. This occurred for all five athletes at weeks 40-41 which was when the final team was named for World Championships and a new squad selected for the 2016-17 season. Because outcome measures for this study are recorded as ratios or percentages, exclusion of these participants from week 40 on did not skew the data, but did reduce the total possible data points able to be recorded.

Twenty one of the 33 subjects (64%) sustained at least one injury during the period of inclusion in the study. Four of these participants reported injuring multiple body sites at the one time, with one participant reporting two multi-site incidents during the 52-week period. Thirteeen subjects sustained multiple injuries over the year, and 12 subjects reported no incidence of injury for the period of data collection. Table 4 shows the injury count recorded, with a breakdown of the events based on squad and gender.

Fifty three body parts were injured in 44 recorded events. Six of the eight (75%) men's endurance athletes sustained at least one injury, with seven of 12 (58%) of the women's endurance squad, five of the nine (56%) men's sprint squad and three of four (75%) of the women's sprint squad sustaining an injury.

Injuries per cycling squad by body region are displayed in Figure 2. Figure 3 displays these results when further condensed into four main body regions, with proportional breakdowns

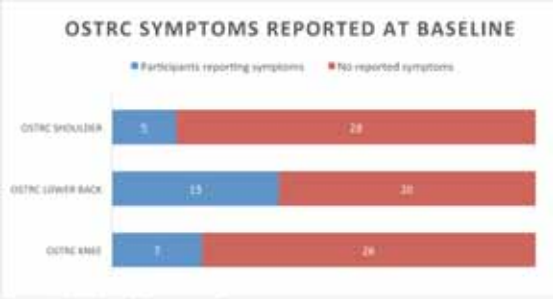

Figure 1. OSTRC initial survey results

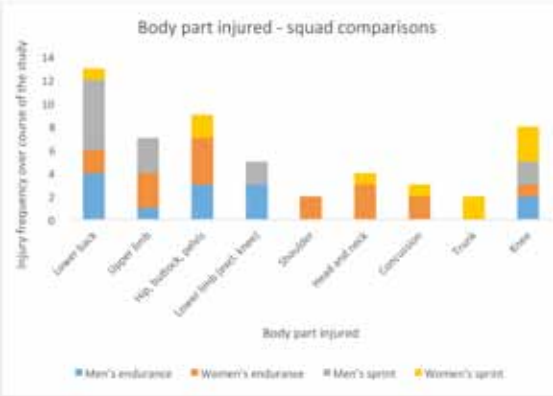

Figure 2. Injury count per body part

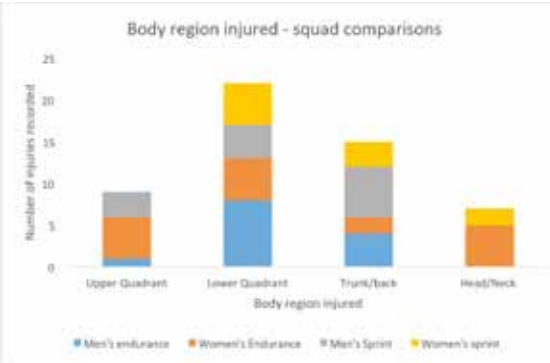

Figure 3. Number of injuries recorded – condensed body part, squad comparisons

for squad and gender in Figures 4 and 5. Thirty-six of the 44 (82%) were recorded as being acute, eight recurrent (18%) with no overuse injuries reported using the PILLS app.

A record was made of all treatments received by each participant at the High Performance Sport Clinics as well as all self-reported treatments. This tally included all physiotherapy and massage treatments, and maintenance therapies or performance optimisation treatments including those not recorded against a specific injury in this study, and allowed an outcome measure of total number of treatments per athlete, which was then compared with injury count and is displayed in Table 5.

Eleven of the injury-incurring incidents were sustained in sports specific training, 20 in the gym, six in competition and seven defined as other (with a mean of 11 and SD 6.38).

Subjects reported 8962 planned training exposures, at a mean of 689 exposures per four-week block of surveys (SD 142). Of these 8962 planned sessions, 60 sessions (0.67%) were missed and a further 84 (0.94%) modified due to injury. This totalled 144 training exposures affected by injury, or 1.6%, with a mean of 11.1 (SD 7) trainings missed or modified per four week block of surveys (Figure 6). Also outlined in Figure 6 are the months of the year, highlighting major competitions, and the international track World Cup and Championship season – October through March.

Injury Incidence and Prevalence

Injury Incidence in this study was described as the number of new injuries per 1000 training and competition

exposures. Therefore, with 44 injury events over 8962 exposures, the injury incidence was 4.9 per 1000 exposures. For all injuries sustained (53 injuries from 44 events) over 8962 training and competition exposures, the injury incidence was 5.9 per 1000 exposures. Injury prevalence for the course of the study was 11%. Point prevalence through the year ranged from one injury per four week block to a maximum of seven (mean 3.38, SD 1.80). Season total prevalence for the study was 112 injuries per 1000 surveys completed.

Pearson's chi square tests were performed to determine any relationship between squad, gender, or previous injury, years in sport, and new injuries or injury frequency. No significant relationships were found.

DISCUSSION

The purpose of this study was to provide an injury profile in track cyclists over a full training and competition year.

This appears to be the first study that has investigated injury surveillance in the elite track cycling cohort in isolation. Palmer-Green[8] reviewed injury surveillance in track cyclists, but only as part of a larger cohort of the British National Cycling team which included Road, BMX and Mountain Bike athletes which makes drawing comparisons to the current research a challenge.

A total of 81% of eligible participants completed the study. Participants used the Program for Injury and Illness Surveillance (PILLS) self-reported survey on a smart phone application to record their injury and training data over a 52-week period. During the course of the study 64% of the participants reported one or more injuries. This was further

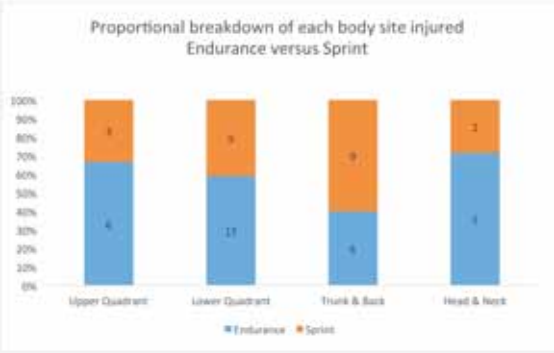

Figure 4. Proportional breakdown of each body site injured - Endurance versus Sprint

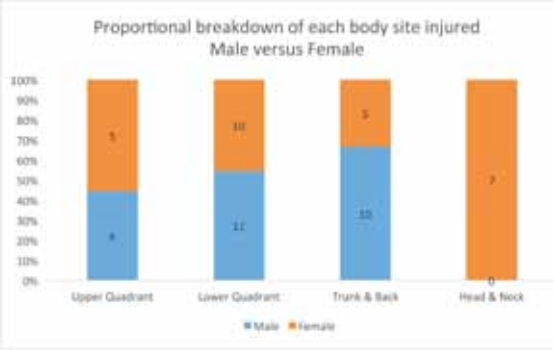

Figure 5. Proportional breakdown of each body site injured – Male versus Female

Figure 6. Training exposures vs injury prevalence

Table 5: Treatments received and injuries recorded.

Participant Code	TREATMENTS - Physio, Massage Injrury, Massage, Maintenance and Performance Therapies	Injury Tally
ME1	69	2
ME2	47	2
ME3	63	3
ME4	34	1
ME5	83	1
ME6	62	0
ME7	96	3
ME8	33	0
WE9	47	0
WE11	68	1
WE12	62	2
WE14	60	0
WE15	51	3
WE16	60	1
WE17	37	2
WE18	58	0
WE19	9	0
WE20	14	1
WE21	24	1
WE22	10	0
MS23	92	1
MS24	101	2
MS25	87	4
MS26	28	0
MS27	84	0
MS28	71	0
MS29	31	2
MS30	18	0
MS31	53	4
WS32	113	4
WS33	71	0
WS34	64	3
WS35	63	2

broken down into squads, where 75% of the men's endurance squad sustained at least one injury, 58% of the women's endurance squad, 56% of the men's sprint squad and 75% of the women's sprint squad with no significant difference in injury rate per squad ($p>0.05$). This equated to 1.6 injuries per athlete, which is comparable to the study by Clarsen et al (2010) study of professional road cyclists which was indicating an injury rate of 1.5 injuries per athlete.[4] The injury incidence was 4.9 per 1000 training exposures, and for all injuries sustained the injury incidence was 5.9 per 1000.

Of the injuries recorded, 25% involved the lower back, 17% Hip/buttock/pelvis, with the knee and upper limb 15% and 13% of recorded injuries respectively. Injuries were also sustained involving the lower limb (excluding the knee), shoulder, trunk, head/neck and concussion. Other cycling specific studies have assessed road cyclists specifically male professional cyclists 3-5 and demonstrated that the knee, lower back, shoulder, lower leg and thigh were the predominant regions injured consistent with the current study.

Previous studies by Barrios and Clarsen investigating injury in road cycling report similar body regions injured in their surveillance studies, although they were all predominantly reported as overuse injuries rather than acute onset events.[3-5] Injury incidence was higher in this study than previous road cycling studies, however the previous research only looked at cycling training and competition injuries whereas the current research recorded all injuries sustained. This may account for the lower rate in the previous studies given that the largest proportion of injuries reported in the current research took place in the gym.

With respect to the location of the injuries sustained, 45% (20/44) of the injury-incurring incidents were sustained in the gym, 25% (11/44) were sustained in sports specific training, 14% (6/44) in competition and 16% (7/44) other (with a mean of 11 and SD 6.38). Previous road cycling studies have only looked at injuries sustained in bike training or competition[3-5] so there is no comparable data to this current research. It is not clear from the current training exposure data what proportion of time was spent between the different types of training exposure (gym, sport specific training or competition), as the PILLS app does not record this information.

Over the 52 weeks of the current research, 60 training sessions (0.67%) were missed and a further 84 (0.94%) modified due to injury. This totalled 144 of 8962 planned training exposures affected by injury, or 1.6%. Studies investigting the impact of injury on performance in athletics,[21] found that if an athlete completed 80% or more of their training then they were seven times more likely to achieve their performance goal.[21] Whilst Raysmith and Drew's study was in an athletic cohort and may

not be directly comparable to cycling, it is still assessing elite athletes, and can be used as reference point. Therefore this study reflects not only a low incidence but also a low impact of injury in terms of effect on training and competition in this cohort.

Injuries in the current research were classified as acute, overuse or recurrent and no self-reported overuse injuries were recorded. This is inconsistent with other cycling studies and so further analysis was completed on the treatment notes corresponding to those self-reported injuries. From this, it was determined that 16 of the self-reported acute injuries were in fact overuse – indicating 46% of injuries were acute, 36% were overuse, and 18% were recurrent.

Barrios (2015) and Clarsen (2010, 2014)'s studies reported predominantly overuse injuries,[3-5] and therefore in comparison with this study, suggests that although both road and track cyclists are at risk of overuse injuries, track cyclists are at greater risk of acute injury than road cyclists.

Initial baseline survey using the OSTRC overuse injury surveillance questionnaire indicated that 15% of participants had shoulder symptoms at the initiation of the research, 39% had Lower back symptoms, and 21% reported knee symptoms. It is unclear whether these symptoms continued during the study and weren't reported or whether they had resolved. This should be an area for future research, either in the wording of the questions used in the injury surveillance tool used, or in the classification methods and tools used by the researcher, or both. In the PILLS injury surveillance tool utilised in this study the questions in the survey ask for a date upon which the injury occurred which did not allow for description of a steadily building event. Another area of investigation to consider is the impact of the variety of training that a track cyclist is exposed to (track training and competition, road cycling, ergometer training, strength and conditioning gym load) and is this protective for injuries, particularly overuse.

No relationships or associations were found between injury incidence and the age, gender or squad discipline of the participants, or in relation to previous injury history. Also investigated but found to be of no significant result were the relationships between age, gender, or squad discipline and the nature, location (geographical location of injury occurrence, and body part location), severity, duration of the injuries, and number of treatments received. When training exposures and injury prevalence were compared across the year, it indicated a possible link between training load and injury prevalence, with an increase in training exposures coinciding with an increase in injury prevalence, and vice versa. There was no statistical relationship found between these variables in this study, but this should be investigated further in future research. The lack of statistical significance with all of these variables was likely due to small sample size and also the low frequency of injuries sustained during the period of the study. Continuing to capture injury data with this cohort over a longer period of time may allow for better assessment of relationships and risk factors.

To improve the applicability of these study results, detailed load measurements should be recorded in any future injury surveillance research. Recent studies suggest that changes in training load may affect injury risk, where training is measured as a combination of exposure (training duration) and either internal (for example rate of perceived exertion) or external (for example distance travelled) load.[21] Hulin et al determined that in the cricket bowling population, an increase in acute (7 day) workload of >1.5 ratio (or >150% increase) compared to their chronic (28 day rolling average) load resulted in a greater than two-fold increase in risk of injury in the following week, and those with a workload ratio increase >2 (200%) had a relative risk of 4.5 for subsequent injury compared with those with a .5-.99 acute: chronic ratio.[21-22] Although this is not specifically in the cycling cohort it is a notable change in risk based on change in training load. Further detail around the nature of load (Rate of Perceived Exertion, and training duration as possible measures) is required

for improved applicability of the results.[22-24]

CONCLUSIONS

The results of this study provide the first descriptive injury profile for the elite New Zealand track cycling cohort. This will aid those working with track cyclists to assess specific injury prevention methods with the aim to enhance performance by reducing training and competition time lost to injury.

Sixty-four percent of participants sustained an injury over the course of the study. The greatest number of injuries were seen in the lower back, the hip/buttock/pelvis region and the knee. Injury incidence and prevalence was low with rapid return to training and competition. The injury surveillance (PILLS) app used in this study did not directly capture overuse injuries, but on review of clinician treatment notes, this number was not high (36%) in comparison to previous road cycling studies.

Future Research

Ongoing data collection in subsequent years is required in order to increase sample size to determine if any relationships exist between the participants and the injuries they sustain.

Future research of this nature should also look at improving detail around training exposures and load which will improve the accuracy and therefore applicability of the study results.

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Injuries in a senior amateur rugby union team over two competition seasons resulted in a ratio of 1:5 witnessed to unwitnessed concussions

DOUG KING, PATRIA A HUME, CONOR GISSANE, TREVOR CLARK, CLOE CUMMINS

ABSTRACT

Aim: To determine the type, site and rate of injuries in men's senior amateur rugby union matches, with focus on concussion.

Methods: A prospective observational cohort study was conducted on a men's senior amateur rugby union team (n=36 players in 2012 and 35 players in 2013) in New Zealand. Types, sites, and frequency of injuries were recorded by a sports medic. Concussions (witnessed or unwitnessed) were only recorded if they were formally diagnosed by a health practitioner. Assessment of SCAT scores for baseline and post-concussive events was conducted. Unwitnessed concussions were determined using changes >3 seconds for pre- to post-match King-Devick test scores with associated changes pre- to post-match SCAT.

Results: 203 injuries were recorded (236.6; 95%CI: 206.2 to 271.5 per 1,000 match hr) over the study of 71 players. An injury was sustained on average every 17 minutes of a match. Most concussions were unwitnessed (RR: 23.9; 95%CI: 11.3 to 50.5; p<0.0001) during play but were identified initially post-match using the SCAT tool. Therefore, whilst a witnessed concussion occurred once every five matches a concussion occurred every match on average. The head/neck region was most frequently injured (88.6; 95%CI: 70.8 to 110.9 per 1,000 match hr), followed by the upper limb (74.6; 95%CI: 58.4 to 95.3 per 1,000 match hr), lower limb (50.1; 95%CI: 37.2 to 67.6 per 1,000 match hr) and chest/back/abdomen (23.3; 95%CI: 15.0 to 36.1 per 1,000 match hr). The ball carrier most frequently sustained concussion injuries.

Discussion: Amateur New Zealand senior men's rugby union is a contact sport where an injury occurs on average every 17 minutes. Most injuries were transient sprains/strains. However, of concern was a concussion occurring on average once a match, most of which were unwitnessed. The ratio of witnessed to unwitnessed concussions was 1:5.3. Major injuries occurred at a rate of 35.0 (95% CI: 24.5 to 50.0) per 1,000 match hr.

Conclusion: The witnessed to unwitnessed concussion ratio of ~1:5 found in this study indicates that concussion is a largely "hidden" injury. Given that concussion injuries were not seen to occur during play, with diagnosis of concussion made only upon players presenting after the game for assessment with the King-Devick tool, it is important for pre-season baseline testing and post-game testing of cognitive function to be

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INTRODUCTION

Reportedly the most popular contact team sport played in more than 200 countries,[1-3] rugby union is a field-based sport contested between two opposing teams of 15 players comprised of eight forward-playing positions and seven back-playing positions.[4] Played at professional, amateur and junior levels of participation by males and females, rugby union is played on a field measuring a maximum of 100 m by 70 m with two in-goal areas, typically of 10 m depth (end-zones). The objective of rugby union is to score as many points as possible by attacking the opposition team's defensive line with the ball, forcefully running towards, and into, the defensive line. The ball can only be passed backwards between players and only the player with the ball can be tackled by the defending team. There is an abundance of tactical kicking and chasing the ball and attempting to run through gaps in the opposition's defence.[4] The team without the ball can use physical contact to tackle the opposition player with the ball to limit advancement of the ball towards their try line and force errors to regain possession of the ball.[4] As a result, the team assumes both defensive and attacking roles multiple times within the same match. The game involves multiple aggressive contact situations (rucks and mauls) where attacking players control the ball and defending players must stop the advancement of the ball. Given rugby is physically demanding, involving both high-intensity (sprinting, tackling, rucking, mauling) and low-intensity (jogging and walking) activities, the risk of an injury is ever-present.[5]

Studies using different definitions of injury have resulted in varied injury rates for rugby being reported.[6] Some studies7-10 have utilised a match time loss only11 injury definition, whilst other studies[7,12] have utilised an all-encompassing injury definition.[13] Consequently, varied injury rates have been reported.[6] At the professional level of competition injury incidence has varied between 912 and 21814 per 1,000 match hours of competition. Injury incidence has been reported as lower in competitions within the Pacific Nations (58.9 per 1,000 match hr),[8] women's world cup (38.6 per 1,000 match hr),[9] U20 international level (55.0 to 64.0 per 1,000 match hr),[15] junior world rugby trophy (31.6 per 1,000 match hr),[10] youth (26.7 per 1,000 match hr),[12] 17 year olds (49.3 per 1,000 match hr),[7] players aged 9-11 years. (6.0 per 1,000 match hr)[7] and players aged 6-15 years (15.5 per 1,000 match hr).[16] Despite the aforementioned injury rates, there is however, a noticeable paucity of injury incidence data for rugby union at the senior premier amateur level.

AIM

This study determined the incidence of injuries over two seasons of competition matches for a senior amateur rugby union team in New Zealand.

METHODS

Study Design

A prospective observational cohort study was conducted during the 2012 and 2013 competition seasons for a premier club level amateur rugby union team in New Zealand.

Ethical Approval

The Auckland University of Technology Ethics Committee approved all procedures involved in this study (AUTEC 12/156) and all players participating in the study gave written informed consent prior to participating.

Player Characteristics and Playing Position

All players were considered amateur due to receiving no remuneration for participating in match activities. The matches were played under the rules and regulations of the New Zealand Rugby Union. Players were grouped into four positional groups:[17] Front row forwards (player numbers 1, 3, 4 and 5), back row forwards (2, 6, 7 and 8), inside backs (9, 10, 12, 13) and outside backs (11, 14, 15). The hooker (player No 2) was included in the back-row forwards based on their roving style of play, whilst the scrum half (player No 9) was included in the inside backs due to being the link between the forwards and the backs.

Injury Reporting

Over the study, all injuries sustained throughout a match were recorded. The team medic was a registered comprehensive nurse with tertiary sports medicine qualifications and accredited in injury prevention, assessment, and management. Injury data were collected from all matches in which the team participated, which included preseason fixtures and all competition matches including the final series. All injuries were recorded on a standardised injury report form regardless of severity.[18,19]

Injury Definition

The definition of injury utilised for this study was "Any physical complaint, which was caused by a transfer of energy that exceeded the body's ability to maintain its structural and/or functional integrity that was sustained by a player during a rugby match or rugby training, irrespective of the need for medical attention or time-loss from rugby activities".[20]

Injuries were classified anatomically according to the player position at the time of the injury occurring, the injury site, the nature of the injury, and the causative mechanisms.[18,20] All injuries were recorded, including multiple sites and types of injuries that were sustained. For injuries identified post-match (ie, unwitnessed concussions), players were asked to estimate the activity that caused the injury and time (match period of play) this occurred. All injuries that occurred during match participation were recorded regardless of severity. However, injuries were also classified according to the number of matches missed as a result of the injury.[20] Transient (0-3 days missed), Mild (4-7 days missed), Moderate (8-28 days missed), or Major (28+ day missed).[20]

King-Devick (K-D) test

Based on the time to perform rapid number naming, the K-D test takes less than two minutes to administer.[21,22] The K-D test involved the players reading aloud a series of random single-digit numbers from left to right. The K-D test included one practice (demonstration) card and three test cards varied in format on either a moisture-proof 6x8 inch spiral bound physical test or as an application on a IPad platform. Players were asked to read the numbers from left to right across the card as quickly as they could without making any errors using standardised instructions. The time was kept for each test card, and the K-D summary score for the entire test was based on the cumulative time taken to read all three test cards. The number of errors made in reading the test cards was recorded. Baseline K-D times for all participants were established either preseason or when participants joined the team after the season had commenced. The best time (fastest) of the two trials without errors became the established baseline K-D test time.[21] When head trauma was suspected the K-D test was utilised as a screening tool to assess for possible concussive injury as part of a series of concussion assessments. The K-D test has not been recommended for use as a standalone diagnostic tool[23,24] and the K-D should be utilised in conjunction with other concussion assessment tools as a sideline screening tool.[24,25] The test was administered once using the same instructions, and the time and errors were recorded then compared to the subject's baseline. Worsening of time and/or errors identified on the sideline or post-match K-D test have been associated with concussive injury,[1,21,22,25-28] and players with any changes from their baseline scores were referred for further medical assessment. K-D test performance has been previously shown to be unaffected in various noise levels and testing environments.[29] The K-D has been reported to have significant correlations ($p<0.0001$) with the visual motor speed (VMS), reaction time (RT), verbal memory (VEM) and visual memory (VIS) of the Immediate Post-concussion Assessment Cognitive Test (ImPACT®)[30] computerised concussion evaluation system. Worsening of the K-D times from baseline[31] have been associated with changes in the Standardised Assessment of Concussion (SAC) ($r=-0.37$; $p<0.0001$) and total symptoms ($r=0.24$; $p=0.0002$). The K-D has also been reported to have an high test-retest reliability (ICC's between 0.86 and of 0.97) in a variety of adolescent and adult

athletic populations.[21,32-35] The K-D tests utilised were v2.2.0 (http://www.kingdevicktest.com) on an IPad2. The IPad2 version enables the use of the K-D test with three different test platforms and these were varied over the duration of the study. The baseline was assessed with platform 1 and the post-match tests were conducted with either platform two or platform three randomly alternated.

Concussion Definition and Assessment

The definition of a concussion utilised for this study was "Any disturbance in brain function caused by a direct or indirect force to the head. It results in a variety of non-specific symptoms and often does not involve loss of consciousness. Concussion should be suspected in the presence of any one or more of the following: (a) Symptoms (such as headache), or (b) Physical signs (such as unsteadiness), or (c) Impaired brain function (eg, confusion) or (d) Abnormal behaviour."[36]

Concussions were classified as witnessed (a concussive injury identified during match activities resulting in removal from match activities) and unwitnessed (changes >3 seconds for pre to post-match King-Devick (K-D) test scores with associated changes pre- to post-match SCAT3 (Sport Concussion Assessment Tool 3)).

An unwitnessed concussion was defined for the purpose of this study as "Any disturbance in brain function caused by a direct, or indirect, force to the head that does not result in any immediate observable symptoms, physical signs, impaired brain function or abnormal behaviour but has a delay in the post-match K-D score of >3 seconds and has associated changes in the post-match SCAT3".[37] The concussion management procedures utilised while conducting this study have been previously reported.[28,37-39] The results of the K-D and the SCAT3 assessments for concussions identified in this cohort have been reported previously.[1,37]

Exposure Determination

Over the duration of the competition, 43 matches were studied. The match exposure was calculated on the basis of 15 players (8 forwards and 7 backs) per team exposed for 80 minutes per team match. The overall match injury exposure was 859.8 hr: 458.6 hr for forwards (229.3 hr for front-row forwards; 229.3 hr for back row forwards) and 401.2 hr for backs (229.3 hr for inside backs; 172.0 hr for outside backs).

Injury Incidence Calculations

The incidence of injury was reported as injuries per 1,000 match hours with 95% confidence intervals (CI). The expected injury frequency was calculated as previously published[40,41] (see Table 1).

Statistical Analyses

Independent t-tests were used to assess differences in baseline data and a one-sample chi-squared ($\chi 2$) test was used to determine whether the observed injury frequency was significantly different from the expected injury frequency. To compare between injury rates, risk ratios (RRs) were used. Data were reported as means with 95% confidence intervals (CI).[42] Results were considered significant at $p<0.05$.

RESULTS

Sample

A total of 71 male players (2012 n=36, 2013 n=35) participated in the study at the senior amateur domestic competition with a mean age of 23.1 ±3.1 years. There were 17 players enrolled in both seasons (2012-2013) while 54 players completed one year of the study.

Incidence of injury

Over the duration of study there were 203 injuries recorded (see Table 1). On average there was an injury occurring every 17 minutes (15-20 min) per match. There were more injuries (p=0.3736) recorded in 2012 than 2013.

Player Position

There were differences in injury incidence by playing position. Front-row forwards recorded more injuries in 2012 than 2013 (RR: 1.9; 95%CI: 1.0 to 3.4; p=0.0468) (see Table 2). There were fewer injuries to outside backs (209.8; 95%CI: 151.4 to 290.9 per 1,000 match hr) than back-row

Table 1: Characteristics of participants of a senior amateur rugby union team in New Zealand by mean (±standard deviation) for age, matches played, injury observed and expected, injury rates, total number of injuries, match minutes per injury for injuries per year, total injuries observed and concussive injuries per 1,000 match hr with 95% confidence intervals.

		2012	2013	Total
Total Injuries	Number of players enrolled	36	35	71
	Age, years ±SD	22.8 ±3.4	23.3 ±6.1	23.1 ±3.1
	Matches played [preseason; match] (match hr)	24 [5,19] (479)	19 [1,18] (379)	43 [6,37] (858)
	Match exposure hours	478.8	379.1	857.9
	Injuries Observed	107	96	203
	Injuries Expected	111	92	203
	Injury rates per 1000 playing hours (95% CI)	223.5 (184.9 to 270.1	253.3 (184.9 to 309.4)	236.6 (206.2 to 271.5)
	Hours per injury (95% CI)	4.5 (3.7 to 5.4)	4.2 (3.4 to 5.1)	4.3 (3.8 to 5.0)
	Total number of injuries per game (95% CI)	4.5 (3.7 to 5.4)	4.8 (3.9 to 5.9)	4.6 (4.0 to 5.3)
	Player appearances per injury (95%CI)	3.4 (2.8 to 4.1)	3.1 (2.6 to 3.8)	3.3 (2.8 to 3.7)
	Match minutes played per injury (95%CI)	17.9 (14.8 to 21.7)	16.7 (13.6 to 20.4)	17.3 (15.1 to 19.9)
Total Concussion	Concussions identified [witnessed, unwitnessed]	22 [5,17]	22 [2, 20]	44 [7, 37]
	Concussions expected	25 [4, 21]	19 [3,16]	44 [7, 37]
	Concussion injury rates per 1000 playing hours (95% CI)	45.9 (30.3 to 69.8)	58.0 (38.2 to 88.1)	51.3 (38.2 to 68.9)
	Hours per injury (95% CI)	21.8 (14.3 to 33.1)	17.2 (11.3 to 26.2)	19.5 (14.5 to 26.2)
	Total number of concussions per game (95% CI)	0.9 (0.6 to 1.4)	1.2 (0.8 to 1.8)	1.0 (0.8 to 1.4)
	Player appearances per concussion (95%CI)	16.4 (10.8 to 24.9)	13.0 (8.5 to 19.7)	14.7 (10.9 to 19.7)
	Match minutes played per concussion (95%CI)	87.3 (57.5 to 132.5)	69.1 (45.5 to 104.9)	78.2 (58.2 to 105.1)
Witnessed Concussion	Witnessed concussions identified	5	2	7
	Witnessed concussions expected	3.9	3.1	7
	Concussion injury rates per 1000 playing hours (95% CI)	10.4 (4.3 to 25.1)	5.3 (1.3 to 21.1)	8.2 (3.9 to 17.1)
	Hours per injury (95% CI)	95.8 (39.9 to 230.1)	189.5 (47.4 to 757.8)	122.6 (58.4 to 257.1)
	Total number of concussions per game (95% CI)	0.2 (0.1 to 0.5)	0.1 (0.0 to 0.4)	0.2 (0.1 to 0.3)
	Player appearances per concussion (95%CI)	72.0 (30.0 to 173.0)	142.5 (35.6 to 569.8)	92.1 (43.9 to 193.3)
	Match minutes played per concussion (95%CI)	384.0 (159.8 to 922.6)	760.0 (190.1 to 3,038.9)	491.4 (234.2 to 1,030.8)
Unwitnessed Concussion	Unwitnessed concussions identified	17	20	37[a]
	Unwitnessed concussions expected	20.7	16.3	37
	Unwitnessed concussion injury rates per 1000 playing hours (95% CI)	35.5 (22.1 to 57.1)	52.8 (34.0 to 81.8)	43.1 (31.3 to 59.5)
	Hours per injury (95% CI)	28.2 (17.5 to 45.3)	19.0 (12.2 to 29.4)	23.2 (16.8 to 32.0)
	Total number of concussions per game (95% CI)	0.7 (0.4 to 1.1)	1.1 (0.7 to 1.6)	0.9 (0.6 to 1.2)
	Player appearances per unwitnessed concussion (95%CI)	21.2 (13.2 to 34.1)	14.3 (9.2 to 22.1)	17.4 (12.6 to 24.1)
	Match minutes played per unwitnessed concussion (95%CI)	112.9 (70.2 to 181.7)	76.0 (49.0 to 117.8)	93.0 (67.4 to 128.3)

CI: = confidence interval; Significant difference ($p<0.05$) than (a) = witnessed concussions.

forwards (RR: 1.7; 95%CI: 1.1 to 2.5; p=0.0111), inside backs (RR: 1.6; 95%CI: 1.1 to 2.5; p=0.0183) and front-row forwards (1.3; 95%CI: 0.9 to 2.0; p=0.2273) over the study.

Injury Sites

The head/neck region was the most commonly recorded injury site over the study (88.6; 95%CI: 70.8 to 110.9 per 1,000 match hr). There were more injuries recorded to the head/neck region of the body than the lower limb (RR: 1.8; 95%CI: 1.2 to 2.5; p=0.0025) and chest/back/abdomen (RR: 3.8; 95%CI: 2.3 to 6.2; p<0.0001) over the study.

Injury Types

Sprains/strains (96.8; 95%CI: 78.0 to 120.0 per 1,000 match hr) were most frequent over the two years of the study. There were more concussions over the study than fractures (RR: 2.9; 95%CI: 1.7 to 5.2; p=0.0002) and wounds (RR: 4.9; 95%CI: 2.4 to 10.0; p<0.0001).

Concussions

There were more concussive events witnessed (p=0.2568) in 2012 than 2013, noting that there were 22 concussion injuries in each year. On average there was a concussion occurring once every match. There were more unwitnessed (n=37) than witnessed (n=7) concussions (RR: 23.9; 95%CI: 11.3 to 50.5; p<0.0001) over the study with a witnessed concussion occurring once every five matches on average. The witnessed to unwitnessed concussion ratio was 1:5.3.

Inside backs recorded more total concussions than front-row forwards (RR: 2.5; 95%CI: 1.0 to 6.4; p=0.0495) (see Table 2). More total concussions were recorded to the ball-carrier (21.0; 95%CI: 13.2 to 33.3 per 1,000 match hr) than contact with the ground (RR: 4.5; 95%CI: 1.5 to 13.2; p=0.0028) over the study and this was similar for players identified with an unwitnessed concussion (RR: 5.3; 95%CI: 1.6 to 18.2; p=0.0029). There were more unwitnessed concussions reported to have occurred in the fourth than the first quarter of matches (RR: 2.5; 95%CI: 1.0 to 6.4; p=0.0495).

Injury Severity

Transient injuries (136.4; 95%CI: 113.8 to 163.5 per 1,000 match hr) were the most common injury severity recorded.

Injury Mechanism

The tackle (170.2; 95%CI: 144.7 to 200.2 per 1,000 match hr) was the most common injury mechanism recorded over the study (see Table 2). Although there were more tackle related injuries in 2013 than 2012 (RR: 1.8 95%CI: 1.3 to 2.4; p=0.0006), there were fewer ruck/maul related injuries in 2013 than 2012 (RR: 4.2 95% CI: 1.8 to 10.0; p=0.0004). The ruck/maul accounted for more injuries than the scrum (RR: 6.3; 95%CI: 2.7 to 14.9; p<0.0001) and lineout (RR: 38.0; 95%CI: 5.2 to 276.2; p<0.0001) over the study.
The ball carrier was most commonly concussed, with the injury mechanisms resulting in concussion injuries being the tackle and collision with the ground.

DISCUSSION

Injury Risk Profile

Rugby union is an aggressive contact sport designed to test both team and individual ability to attack the opposition and defend one's try line. It is therefore, not surprising that rugby union carries a significant injury risk profile. Previous studies have attempted to quantify this risk for different age groups, gender and levels of competition.[2,7-10,12,15,16] However, to date there remains limited data on the risk profile of senior men's amateur rugby union match activities.

Although a consensus statement has been established[20] for the definition of an injury and data collection procedures in rugby union, this is more focused on data capture at the professional level of participation by full-time medical staff.43 Professional teams typically have medical support such as physiotherapists and sports medicine specialists and they can easily manage injuries that do not result in match time loss, however, these services are not always available to amateur level teams.[44] The inclusion of transient, or non-time

loss injury data enables a true, global picture of the incidence of injury in sports as 70 to 92% of all injuries sustained fall into the transient injury category.[13]

Previous studies have reported an injury rate ranging from 912 to 21814 per 1,000 match hours of competition. In this study we report an injury rate of 236.6 (95% CI: 206.2 to 271.5) per 1,000

Table 2: Incidence of injuries for a senior amateur rugby union team in New Zealand over two completion seasons by player position, injury site, and injury type, injury cause, injury severity and injury period by number of injuries, incidence per 1,000 match hr with 95% confidence intervals.

	2012		2013		Total	
	N=	Rate (95% CI)	n=	Rate (95% CI)	n=	Rate (95% CI)
Player position						
Front Row Forwards	33[bf]	258.5 (183.7 to 363.6)	14[ad]	138.5 (82.0 to 233.9)	47	205.5 (154.4 to 273.5)
Back Row Forwards	26[b]	203.6 (138.6 to 299.1)	35[ac]	346.3 (248.6 to 482.3)	61[f]	266.7 (207.5 to 342.7)
Inside Backs	34[f]	266.3 (190.3 to 372.7)	25	247.3 (167.1 to 366.0)	59[f]	257.9 (199.8 to 332.9)
Outside Backs	14[bce]	146.2 (86.6 to 246.9)	22[a]	290.2 (191.1 to 440.7)	36[de]	209.8 (151.4 to 290.9)
Forwards	59	231.0 (179.0 to 298.2)	49	242.4 (183.2 to 320.7)	108	236.1 (195.5 to 285.1)
Backs	48	214.8 (161.9 to 285.1)	47	265.7 (199.6 to 353.6)	95	237.3 (194.1 to 290.2)
Injury site						
Head/Neck	39[j]	81.5 (59.5 to 111.5)	37[ij]	97.6 (70.7 to 134.7)	76[ij]	88.6 (70.8 to 110.9)
Upper Limb	34[j]	71.0 (50.7 to 99.4)	30[j]	79.1 (55.3 to 113.2)	64ij	74.6 (58.4 to 95.3)
Lower Limb	25[j]	52.2 (35.3 to 77.3)	18[g]	47.5 (29.9 to 75.4)	43[ghj]	50.1 (37.2 to 67.6)
Chest/Back/Abdomen	9[ghi]	18.8 (9.8 to 36.1)	11[gh]	29.0 (16.1 to 52.4)	20[ghi]	23.3 (15.0 to 36.1)
Injury type1						
Sprains/Strains	46[lmnopq]	96.1 (72.0 to 128.3)	37[nopq]	97.6 (70.7 to 134.7)	83[lmnopq]	96.8 (78.0 to 120.0)
Contusion	25[knopq]	52.2 (35.3 to 77.3)	23[nopq]	60.7 (40.3 to 91.3)	48[knopq]	56.0 (42.2 to 74.2)
Concussions	22[klnopq]	45.9 (30.3 to 69.8)	22[nopq]	58.0 (38.2 to 88.1)	44[knopq]	51.3 (38.2 to 68.9)
Fractures	7[klmnpq]	14.6 (7.0 to 30.7)	8[lmopq]	21.1 (10.6 to 42.2)	15[klmpq]	17.5 (10.5 to 29.0)
Wounds	6[klmnpq]	12.5 (5.6 to 27.9)	3[lmn]	7.9 (2.6 to 24.5)	9[klm]	10.5 (5.5 to 20.2)
Dislocations	3[klmnoq]	6.3 (2.0 to 19.4)	2[lmn]	5.3 (1.3 to 21.1)	5[klmn]	5.8 (2.4 to 14.0)
Other*	2[klmnop]	4.2 (1.0 to 16.7)	2[lmn]	5.3 (1.3 to 21.1)	4[klmn]	4.7 (1.8 to 12.4)
Injury cause						
Tackle	61[suvwx]	127.4 (99.1 to 163.7)	85[ruvw]	224.2 (181.3 to 277.4)	146[uvwx]	170.2 (144.7 to 200.2)
Ruck/Maul	32[stvwx]	66.8 (47.3 to 94.5)	6[r]	15.8 (7.1 to 35.2)	38[tvwx]	44.3 (32.2 to 60.9)
Other**	10[tux]	20.9 (11.2 to 38.8)	2[t]	5.3 (1.3 to 21.1)	12[tux]	14.0 (7.9 to 24.6)
Scrum	3[tu]	6.3 (2.0 to 19.4)	3[t]	7.9 (2.6 to 24.5)	6[tu]	7.0 (3.1 to 15.6)
Lineout	1[tuv]	2.1 (0.3 to 14.8)	0	0 -	1[tuv]	1.2 (0.2 to 8.3)
Injury severity						
Transient	63[z12]	131.6 (102.8 to 168.4)	54[z12]	97.6 (70.7 to 134.7)	117[z12]	136.4 (113.8 to 163.5)
Mild	12[y]	25.1 (14.2 to 44.1)	19[y2]	50.1 (32.0 to 78.6)	30[y]	35.0 (24.5 to 50.0)
Moderate	17[y]	35.5 (22.1 to 57.1)	17[h2]	44.8 (27.9 to 72.1)	26[y]	30.3 (20.6 to 44.5)
Major	15[y]	31.3 (18.9 to 52.0)	6[z1]	15.8 (7.1 to 35.2)	30[y]	35.0 (24.5 to 50.0)

Table Continued from Page 28

Injury period						
1st period of match play	19[4]	158.7 (101.2 to 248.9)	11[456]	116.1 (64.3 to 209.6)	30[456]	139.9 (97.8 to 200.1)
2nd period of match play	36[3]	300.8 (216.9 to 416.9)	28[3]	295.5 (204.0 to 427.9)	64[3]	298.4 (233.6 to 381.3)
3rd period of match play	30	250.6 (175.2 to 358.5)	28[3]	295.5 (204.0 to 427.9)	58[3]	270.4 (209.1 to 349.8)
4th period of match play	22	183.8 (121.0 to 279.1)	29[3]	306.0 (212.7 to 440.4)	51[3]	237.8 (180.7 to 312.9)
1st half of match play	55	229.7 (176.4 to 299.2)	39	205.8 (150.3 to281.6)	94	219.2 (179.0 to 268.3)
2nd half of match play	52	217.2 (165.5 to 285.1)	57	300.8 (232.0 to389.9)	109	254.1 (210.6 to 306.6)

CI: = confidence interval; * = infection, foreign body; ** = Fall, Slip, Twist, Overuse; 1 = will not equal total amount as multiple injury types recorded; Significant difference (p<0.05) than (a) = 2012; (b) = 2013; (c) = Front Row Forwards; (d) = Back Row Forwards; (e) = Inside Backs; (f) = Outside Backs; (g) = Head/neck; (h) = Upper Limb; (i) = Lower Limb; (j) = Chest/Back/Abdomen; (k) = Sprains/Strains; (l) = Contusion; (m) = Concussions; (n) = Fractures; (o) = Wounds; (p) = Dislocations; (q) = Other; (r) = 2012; (s) = 2013; (t) = tackle; (u) = Ruck/maul; (v) = Other; (w) = Scrum; (x) = Lineout; (y) = Transient; (z) = Mild; (1) = Moderate; (2) = Major; (3) = 1st period of match play; (4) = 2nd period of match play; (5) = 3rd period of match play; (6) = 4th period of match play

Table 3: Incidence of concussions in a senior amateur rugby union team in New Zealand over two competition seasons by player position and injury period per 1,000 match hr with 95% confidence intervals.

	Total Concussions		Witnessed Concussions		Unwitnessed Concussions*	
	n=	Rate (95% CI)	n=	Rate (95% CI)	n=	Rate (95% CI)
Player Position						
Front Row Forwards	6[b]	26.2 (11.8 to 58.4)	1	4.4 (0.6 to 31.0)	5	21.9 (9.1 to 52.5)
Back Row Forwards	14	61.2 (36.2 to 103.3)	3	13.1 (4.2 to 40.7)	11	48.1 (26.6 to 86.8)
Inside Backs	15[a]	65.6 (39.5 to 108.8)	2	8.7 (2.2 to 35.0)	13	56.8 (33.0 to 97.9)
Outside Backs	9	52.5 (27.3 to 100.8)	1	5.8 (0.8 to 41.4)	8	46.6 (23.3 to 93.2)
Forwards	20	43.7 (28.2 to 67.8)	4	8.7 (3.3 to 23.3)	16	5.0 (21.4 to 57.1)
Backs	24	60.0 (40.2 to 89.4)	3	7.5 (2.4 to 23.2)	21	52.5 (34.2 to 80.5)
Injury Cause						
Ball carrier	18[d]	21.0 (13.2 to 33.3)	2	2.3 (0.6 to 9.3)	16[d]	18.7 (11.4 to 30.4)
Tackler	11	12.8 (7.1 to 23.2)	4	4.7 (1.8 to 12.4	7	8.2 (3.9 to 17.1)
Unknown	11	12.8 (7.1 to 23.2)	0	0.0 -	11	12.8 (7.1 to 23.2)
Contact with ground	4[c]	4.7 (1.8 to 12.4)	1	1.2 (0.2 to 8.3)	3[c]	3.5 (1.1 to 10.8)
Injury Period						
1st period of match play	6[f]	28.2 (12.7 to 62.7)	0	0.0 -	6[f]	28.2 (12.7 to 62.7)
2nd period of match play	14	65.8 (39.0 to 111.1)	3	14.1 (4.5 to 43.7)	11	51.7 (28.6 to 93.3)
3rd period of match play	9	42.3(22.0 to 81.3)	4	18.8 (7.1 to 50.1)	5	23.5 (9.8 to 56.4)
4th period of match play	15[e]	70.5 (42.5 to 116.9)	0	0.0-	15[e]	70.5 (42.5 to 116.9)
1st half of match play	20	46.3 (29.9 to 71.7)	3	6.9 (2.2 to 21.5)	17	39.3 (24.5 to 63.3)
2nd half of match play	24	55.5 (37.2 to 82.9)	4	9.3 (3.5 to 24.7)	20	46.3 (29.9 to 71.7)

* = injury cause and injury period based on player recall after concussion identified with K-D and SCAT scores post-match; Significant difference (p<0.05) than (a) = Front Row Forwards; (b) = Inside Backs; (c) = Ball Carrier; (d) = Contact with ground; (e) = 1st period of match play; (f) = 4th period of match play.

match hr., suggesting that this level of competition carries the highest reported risk of injury. Amateur players may be less fit, less trained and less optimised for the rigors of competitive rugby. This competition also has an eclectic array of ages, talents, fitness and skill levels as evidenced by the high turnover of players with 71 players included in this team over two seasons.

Witnessed to Unwitnessed Concussion Ratio

In recent times an increased understanding of the effects of concussive and subconcussive injuries has raised awareness of this condition. These injuries represent a spectrum of disorders from temporary to permanent neurological injury. In this study, a concussion was recorded, on average, once every match resulting in an incidence of 51.3 (95% CI: 38.2 to 68.9) per 1,000 match hr. Of concern was that most of the recorded concussions were unwitnessed (RR: 23.9; 95%CI: 11.3 to 50.5; $p<0.0001$), making it difficult for match officials and medical staff to intervene. Furthermore, whilst most injuries at this level of competition occur to the head and neck region (88.6; 95%CI: 70.8 to 110.9 per 1,000 match hr), most are minor, again making it difficult for officials to discern which impact is the one predisposing to concussion. The witnessed to unwitnessed concussion ratio of ~1:5 found in this study of a senior male amateur rugby union team in New Zealand over two competition seasons indicates that concussion is a largely "hidden" injury. Given the concussion injury was not seen to occur during play, with diagnosis of concussion made only upon players presenting after the game for assessment with the King Devick, it is important for pre-season baseline testing, and post-game testing of cognitive function to be undertaken.

Musculoskeletal Injury

Limb trauma occurs at an accumulative rate of 124.7 per 1,000 match hr., with the upper limb being more commonly injured (74.6; 95%CI: 58.4 to 95.3 per 1,000 match hr) than the lower limb (50.1; 95%CI: 37.2 to 67.6 per 1,000 match hr). This is in conflict with previous studies reporting rugby union match injuries where the lower limb was more commonly injured than the upper limb.[2,45] This finding may be related to the changing style of rugby match play that has occurred since these previous studies[2,45] were undertaken. Further studies are encouraged to identify whether the upper limb is becoming more commonly injured than the lower limb. However, most injuries are sprains and strains (96.8; 95%CI: 78.0 to 120.0 per 1,000 match hr) or contusions (56.0; 95%CI: 42.2 to 74.2 per 1,000 match hr), which is not uncommon for contact sports such as rugby union. More concerning injuries occurred, on average, every second game (35.0; 95%CI: 24.5 to 50.0 per 1,000 match hr), including fractures (17.5; 95%CI: 10.5 to 29.0 per 1,000 match hr), wounds (10.5; 95%CI: 5.5 to 20.2 per 1,000 match hr) and dislocations (5.8; 95%CI: 2.4 to 14.0 per 1,000 match hr). These injuries typically require urgent medical attention, yet at this level it is rare to have a medical professional present with club members providing some form of first aid on the sideline.

Player Position

Backrow forwards recorded the highest injury incidence (266.7; 95%CI: 207.5 to 342.7 per 1,000 match hr) when compared by player positional group. This may be related to the role that they undertake during match participation with an open roaming position, a high workload, and a high tackle rate.[46] This correlates with the tackle being the most common cause of injury (170.2; 95%CI: 144.7 to 200.2 per 1,000 match hr). Interestingly, inside backs had the highest concussion rate (65.6; 95%CI: 39.5 to 108.8 per 1,000 match hr) and the ball carrier (21.0; 95%CI: 13.2 to 33.3 per 1,000 match hr) recorded more concussions (12.8; 95%CI: 7.1 to 23.2 per 1,000 match hr) than the tackler.

Study Limitations

A limitation of the methodology is the players retrospective reporting of time and events of concussion in the unwitnessed concussive events. By definition, a player who has been concussed

has had a "disturbance in brain function", thus the accuracy of their recall may be limited. While this study affords a comprehensive view of a single New Zealand amateur team, extrapolation of results to all senior men's amateur rugby in New Zealand or in other nations would be questionable.

CONCLUSION

Amateur senior men's rugby union has a significant injury risk profile with major injuries occurring at a rate of 35.0 (95% CI: 24.5 to 50.0) per 1,000 match hr. An injury occurs on average every 17 minutes, most of which are transient sprains/strains. However, the head/neck region is the most commonly injured site with a concussion occurring on average once a match, most of which are unwitnessed. The witnessed to unwitnessed concussion ratio of ~1:5 found in this study indicates that concussion is a largely "hidden" injury. Given the concussion injury was not seen to occur during play, with diagnosis of concussion made only upon players presenting after the game for assessment with the King-Devick tool, it is important for pre-season baseline testing and post-game testing of cognitive function to be undertaken.

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Achilles Tendinopathy

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We all know how common Achilles tendinopathy is, particularly in our middle and older age running athletes, but also in the 'out of shape' often overweight individual who does a bit more than they're used to or has an acute precipitating event. When I began my sports medicine specialist training in the mid-2000s, the Alfredson eccentric strengthening exercises seemed to be being applied universally, from my perspective with mixed outcomes. That was also when autologous blood injections were becoming popular, and I performed my research on this (a fairly poor quality RCT that was the forerunner to Kevin Bell's much improved study on the same). I have maintained an interest in the management of this condition, and will outline my current approach here.

Confirm the diagnosis

My first point would be to be sure of what you are dealing with. I think many of my colleagues have had the experience of a so-called Achilles tendinopathy in fact being primarily related to posterior ankle impingement, a calcaneal bone stress injury, a lumbar disc causing nerve root irritation, or other less common pathologies. This is a mainly clinical diagnosis, and relying on imaging of the Achilles is complicated by the high rate of asymptomatic Achilles that look abnormal on ultrasound.

Load management – unsexy but pivotal

I am a fan of the Purdham-Cook continuum model of tendinopathy[1] (although in practice I find the 'dysrepair' phase difficult to identify). I believe that having a conceptual basis is crucial to then frame the principles of treatment. Identifying the stage of tendinopathy is the first step, and in my clinic the most common presentation by far is the mixed reactive and degenerative type, ie, a subclinical tendinosis that becomes symptomatic from either overload or an acute event, and then becomes trapped in the 'injury → failed healing response → reinjury' loop. Related to this, determining the current load capacity tells me where we need to start. Someone who is getting pain with simple ADLs needs to be further offloaded, such as a relatively brief period of time in a moonboot or the use of heel raises, or simply to modify their weightbearing activity levels if this is practical. Some of course just need to be told to stop running for a decent period of time and work on a more controlled loading program. Educating patients on the realistic time frames for tendon recovery is useful early on. During the time of relative offloading some form of controlled strengthening is definitely preferable, usually in the form of isometric inner range holds (either double- or single-leg heel raise depending on pain levels) to build up muscle strength and provide low level tendon load. After about 4 weeks they are reassessed regarding whether they are ready to progress to some concentric exercises and then eventually a predominant eccentric and finally plyometric component. Of course throughout this time their tolerance for non-rehab weightbearing activities should be expected to progressively increase.

I often reflect that there is nothing 'medical' about this advice to date, but it seems that perhaps I have the benefit of spending the requisite time initially with the patient to go through this and then the authority of the specialist to perhaps underline and reinforce much of what they have been told prior to seeing me, usually with subsequent compliance.

Objective functional measures

It can be difficult for patients to remember what their condition was like 3+ months ago and

thus what progress they have made. I therefore find it useful to have them complete a baseline VISA-A (Victorian Institute of Sport – Achilles[2]) questionnaire at their first appointment, and then at subsequent follow-up visits to allow tracking of an objective measure over time. They can do this in the waiting room so does not add to the consultation time. I acknowledge that the VISA-A has limitations, particularly for the less 'dynamic' patients, but some of the measures (e.g. duration of morning stiffness) are relevant across the board.

Mid vs insertional

I feel as if a bit too much is often made of the distinction between mid and insertional pathology. Of course I acknowledge the theory of entheseal compression and agree with avoidance of excessive loading in a stretched position, but that is how I treat the mid tendon early on as well. And with the Haglund's deformities, these have usually been present and asymptomatic for years before presentation so again taking a load management approach initially seems appropriate, rather than referring everyone off to the surgeon straight away.

Biomechanical factors

I have a low threshold for recommending the involvement of one of our local sports podiatrists if I feel that there is a significant biomechanical and/or footwear contribution to the patient's presentation. If the patient's time or resources preclude this then I can provide a rudimentary service but my preference is to engage the experts in the area. Of course evaluating the contribution of the kinetic chain and addressing any proximal weakness in particular is often helpful.

Fancy adjuncts

As I said above, I conducted some relatively early research on autologous blood injections, and reviewed the literature then on the range of so-called adjunctive treatments including the likes of other injectables, shock wave therapy, and nitrate patches. Since then of course PRP has come on the scene, and I have monitored the evidence (or lack thereof!) that has come out and not been impressed enough to jump onboard at this stage (this was used as the example of a literature search in by Ardern et al[3]). I appreciate that a number of my colleagues offer some of these options, and acknowledge that some patients are undoubtedly looking for something additional and that this meets this apparent need. Given that in my experience I have found that the short-term efficacy of such treatments seems to correlate positively with the enthusiasm and optimism of the person explaining and delivering it (and therefore the expectations of the patient), I feel as if I am not the best person to be offering it. While it is no longer common practice to inject corticosteroids into or around the Achilles tendon, it can sometimes be tempting to arrange an ultrasound-guided injection of a prominent retrocalcaneal bursa that appears to be contributing to an enthesopathy. Having had a patient rupture their Achilles 3 weeks after this I am now even more gun shy, even with the precaution of having the patient go into a moonboot for 2-3 weeks post-injection.

The curly ones

There remains ongoing debate about the potential role of plantaris compression, but from a pragmatic perspective if a patient is responding atypically to load management and imaging reveals a plantaris then I would have a lower threshold for obtaining a foot and ankle orthopaedic opinion. Sometimes adhesions without a plantaris present can present similarly and while brisement is not part of my current practice, perhaps it should be. I keep my radar on for presentations associated with systemic inflammatory or metabolic disease. It is fairly well recognised that the recently post-menopausal woman is at higher risk, and there also seems to be a difficult to quantify association with lipid metabolism but also confusingly statins in some patients.

And finally, I try to find some time in every consultation to initiate or reinforce a more general wellness concept, usually related to physical activity for those patients I feel would benefit from this. I see this a little like the smoking cessation approach (but in reverse) where a number of small nudges and encouragement over time is more likely to lead to eventual sustained behaviour change.

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From a podiatry perspective, too, the Purdam Cook model is useful in clinical decision making. In addition, updated input from Ebony Rio recognises increasing inclusion of neural factors in different areas of tendinopathy management.

By the time I see tendinopathy clients they have often already been enrolled in an exercise regime. Frequently they have stopped the programme part way through.

In my 20 years of podiatry practice, the request for biomechanical input for achilles tendinopathy has reduced. This is undoubtedly associated with the newer information we have around the causative mechanisms of this sort of condition, and its aetiology being primarily biological. Nevertheless, it is important to assess whether there is a biomechanical component – and how large that component is – before discussing options with the client.

During my client consultation I reinstate and reinforce the need for a fundamental exercise base to be well established (and often reintroduced). The staging paradigm presents a good basis to help form this around. I see quite a few "acute on chronic injuries" of this type and patient education is very important to achieve successful resolution. I run through the basic pathophysiology of this condition with the patient so they have a good understanding of the benefit of the exercises and what we are trying to achieve.

A key point is to integrate plyometric exercises as a final step before full transition back into sports.

I have observed a big difference in responses between compression and mid-section achilles tendinopathy. The reduction of compression is a good adjunct to treating people with chronically painful insertional achilles tendinopathy. I find that heel raises are often better suited to this variation of the condition.

Footwear selection and footwear components are very important, typically including different pitch heights for rugby boots, cross training shoes etc. In decompressive loading for the compression retrocalcaneal insertional tendinopathies we use FS6 socks and integrate a small offloading device external to this, stitched into the sock (see figures). This prevents it from moving in the shoe and provides real-time relief where footwear compression can be a problem. We have found good results using this for retrocalcaneal contusions also.

In summary, while there are still biomechanical issues to be addressed in some cases, training loads and exercise regimes are the mainstays of treatment. How we educate our clients, to build their understanding, buy-in and follow through on their treatment regime, is vital.

Figure 1: Location of pain and marking.

Figure 2: Transferring markings to the sock.

Figure 3: Padding trimmed to be stitched.

Figure 4: Padding stitched and re-usable in shoes.

HISTORICAL - PART I

Sports Medicine New Zealand The first 50 years

DAVID GERRARD AND CHRIS MILNE

Early Beginnings and Founding Figureheads

In Dunedin on 11 February 1963, a small gathering of like-minded individuals convened the first formal meeting of the New Zealand Federation of Sports Medicine (NZFSM). This fledgling group was the forerunner of Sports Medicine New Zealand (SMNZ), heralding the formation of a special interest group based on collaborative interests in sport science and clinical medicine at the University of Otago. The meeting took place in the Red Lecture Theatre, in the Scott Building of the Otago Medical School and a commemorative plaque inside the entrance of the Scott Building records this landmark occasion. The prime objective of the meeting was to elect foundation officers of a fledgling organisation to be modeled on its counterpart, the British Association of Sport and Medicine (BASM). The attendees shared a common interest in the medical and scientific aspects of sport and physical activity.

From left, Sandy Macalister, Jack Kilpatrick, Norrie Jefferson and John Heslop, Dunedin 1963

It seemed proper at that time for Dunedin to provide the forum for this seminal event given that its campus hosted New Zealand's only schools of medicine, physiotherapy, dentistry and physical education. There are no official records to confirm the full list of attendees that evening in 1963, but a commemorative plaque records the election of the following to the first NZFSM Executive Committee; Dr Norrie Jefferson (President), Dr Jack Kilpatrick (Hon Secretary) and Dr John Heslop and Professor Phillip Smithells, as members of the inaugural leadership group. Of those "founding fathers" Professor Philip Smithells, was Dean of the University of Otago School of Physical Education, and the only non-medical member. Doctors Norrie Jefferson, Jack Kilpatrick and John Heslop, graduates of the Otago Medical School, were consultant clinicians at Dunedin Public Hospital in the Departments of Radiology, Medicine and Surgery respectively. But before long, and reflective of the local expertise on the Otago campus, this group was expanded to include oral surgeon, Professor Sandy Macalister and physician Dr Ted Nye. These men contributed clinical and practical expertise together with an active involvement in rugby, cricket, swimming, hockey, fencing and athletics at a provincial, national and international level.

However the idea to formally establish a sports medicine group in New Zealand had been germinating for some years prior to 1963. From personal communications,(N. Jefferson 1996) the

topic of sport and medicine had been a regular conversational piece for a pair of young New Zealand Army Medical Officers serving in Japan during the latter stages of the Second World War. Dr Mayne Smeeton (Auckland) and Dr Norrie Jefferson (Dunedin) found themselves working at the New Zealand Field Hospital at Kiwa on the Inland Sea, not far from Hiroshima. Beyond their clinical medical interests these two shared a love of sport and Dr Jefferson, in recalling the background to these early discussions, reflected that it was, "… when relaxing in the Officers' Mess in the evenings, that the subject of sports medicine would usually come up for discussion." (personal communication 1995). Mayne Smeeton had been the NZ Universities high jump champion while Norrie Jefferson, by his own admission was a "modest harrier" with the Leith Club in Dunedin.

But it would be some years later, following another chance meeting between two Otago-trained doctors in post-War England, that the likelihood of a sports medicine interest group in New Zealand really took shape. Once again, Norrie Jefferson was a central figure. Having completed his tour of military duty, Norrie travelled to St Mary's Hospital, London to gain clinical experience in the burgeoning area of medical imaging in what was to be his ultimate specialty as a diagnostic radiologist. And it was there that he made the serendipitous connection with another Kiwi expatriate, Sir Arthur Porritt, an eminent, highly respected general surgeon who was also on staff at St Mary's. Porritt was already celebrated for his remarkable and diverse achievements and his move to England in 1923 as a Rhodes scholar from Otago University had seen him stay on after his time at Oxford to complete surgical training. Porritt was an Olympic Bronze medallist athlete for New Zealand in the 100 metres at Paris in 1924, the event immortalised by the movie "Chariots of Fire". He had also served as New Zealand's Chef-de Mission at the 1936 Berlin Olympics where as mentor to Jack Lovelock (another Otago medical graduate) he witnessed a memorable gold for New Zealand in the 1500m. And as a member of the International Olympic Committee he also bore first hand witness to the spectacle and intrigue of what was to become known as the "Nazi Olympics" of 1936. Porritt had also been co-founder, in 1952, with another outstanding medical knight Sir Adolphe Abrahams, of the British Association of Sport and Medicine (BASM).

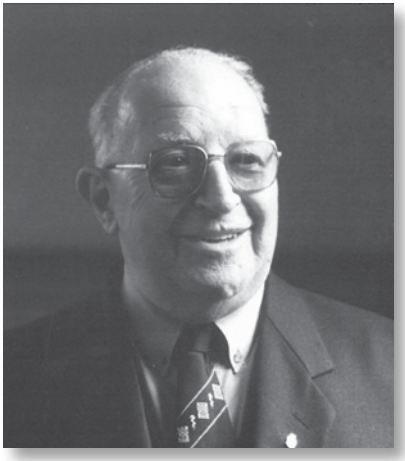
Arthur Porritt, 1924, Paris

It was therefore opportune and most appropriate for Norrie Jefferson to engage Arthur Porritt in discussions on the emerging status of sports medicine in the Antipodes. Stimulated by valuable insights, Norrie Jefferson was already shaping plans for the establishment of a sports medicine interest group in New Zealand and in 1950, having fulfilled his obligations to postgraduate training he returned to New Zealand. Accepting a consultant radiologist position at Dunedin Hospital Norrie was delighted to find that Mayne Smeeton had also been appointed to a Dunedin Hospital anaesthetic post. Serendipity aside, the pair resumed their earlier discussion, more informed now by the input from Arthur Porritt. At the same time, two other Dunedin Hospital doctors, John Heslop and Jack Kilpatrick also declared an interest in sport and medicine. They brought their active involvement in athletics, cricket and swimming into the mix. Despite the fact that these four clinicians provided a focus of interest in sports medicine, the formalization of a national interest group remained dormant for a number of years. Meanwhile on the Otago campus, the national School of Physical

Education was expanding, and an interest in the physiology of exercise and health was being kindled. Professor Phillip Smithells the first Dean of Physical Education expressed a desire to bring sport science to the table. This collegiality, founded in the early 60's continued to blossom and today has emerged as the specialised area of sports science. At the same time, a budding young oral surgeon, A. D. "Sandy" Macalister, from a University background in rugby and athletics, joined the academic staff of the Otago Dental School and the professional ranks of sports medicine were swelled. Sandy Macalister was to become a leading figurehead in New Zealand sports medicine and as Professor of Oral Surgery at the University of Otago Dental School, chaired the National Executive during a period of extensive change.

The next significant stage of development for sports medicine locally, occurred in February 1961 when the New Zealand Branch of the British Medical Association (BMA) held an important meeting in Auckland chaired by Dr W.E. Henley. When it was announced that this Branch meeting would be highlighted by an address from Sir Arthur Porritt, Norrie Jefferson sought permission for a meeting of persons interested in sport and medicine to be held during a break in the main agenda. At this ad hoc meeting, Sir Arthur addressed the work of the British Association of Sport and Medicine, highlighting its advantages not only to the athletes of England but to the public at large. His powerful advocacy for sports medicine set the scene for profitable debate, after which it was determined by the attendees, "That it seemed to be desirable to form a NZ Society of Sport and Medicine." (Jefferson 1969) Subsequently a Dunedin-based steering committee was established to investigate this proposal, collect information from kindred international organisations and to report back to the New Zealand Medical Association at a future Annual Meeting.

Thereafter followed the 1963 meeting of interested parties in Dunedin and the New Zealand Federation of Sports Medicine was finally established after a prolonged and somewhat convoluted gestation. What followed in the subsequent few years remains a little unclear and no official records of executive meetings are available. However it seems very likely that the establishment of a professional organisation representing the joint interests of medicine and sport was the catalyst to the appointment of Dr Renton Grigor (Auckland) as the first doctor to accompany a New Zealand Olympic Team. That appointment was made in 1964 on the occasion of the Tokyo Olympics and heralded the beginnings of a longstanding relationship between the NZFSM and the New Zealand Olympic and Commonwealth Games Association (NZOCGA). Today this relationship has become the basis for appointments of medical staff to every Olympic and Commonwealth Games team. Active membership of SMNZ has become a pre-requisite for such appointments.



Dr Norrie Jefferson

OBE KStJ MBChB (Otago) DMRD FRACR FSMNZ

Inaugural President
Sports Medicine New Zealand

In 1963, Norrie Jefferson, a consultant radiologist at Dunedin Hospital, chaired a meeting of like-minded clinicians who formed the New Zealand Federation of Sports Medicine (NZFSM). With colleague doctors Jack Kilpatrick and John Heslop and Dental Professor Sandy Macalister, these men constituted the first national executive committee, soon joined by Professor Phillip Smithells, Dean of Physical Education.

Dr Norrie Jefferson, the son of a Methodist minister, attended Wellington College before entering the Otago Medical School. He studied overseas at St Mary's Hospital London, gaining specialist qualifications in diagnostic radiology immediately after WW2. At St Mary's there was also a serendipitous meeting between Norrie Jefferson and a staff surgeon named Arthur Porritt. Dr Porritt, also an Otago medical alumnus, was a general surgeon, who, as a Rhodes scholar at Oxford, competed for New Zealand at the 1924 Paris Olympics, winning bronze in the 100 metres, immortalised by the movie "Chariots of Fire". Much earlier Arthur Porritt had been instrumental in establishing the British Association of Sport and Medicine. Norrie Jefferson returned to New Zealand in 1950 stimulated by his discussions with Arthur Porritt, however the birth of sports medicine locally was still some time away.

Norrie Jefferson was a modest athlete who joined the Leith Harrier Club in Dunedin in 1936. He became President of Otago Athletics in 1955 and President of Athletics New Zealand in 1960. His life-long interest in track and field was acknowledged when was made Patron of Athletics Otago and later accorded Life Membership. During this period Norrie gave considerable support to the coach Arthur Lydiard, whose stable of athletes including Sir Murray Halberg and Sir Peter Snell was rewriting world middle-distance records. Norrie provided Arthur Lydiard with medical guidance that enabled the establishment of the 1970s "jogging phenomenon". The premise, that sustained aerobic activity had value in cardiac rehabilitation, was fundamental to the establishment of the contemporary concept of exercise prescription. Norrie managed the athletics section of the 1962 Empire Games team to Perth and travelled extensively with New Zealand Paraplegic Teams to Jamaica (1966), Israel (1970) and Germany (1972). In 1979 he was awarded an OBE for services to disability sport. Public recognition of his extraordinary services to the wider community came when he was made a Knight of the Order of St John, reflective of many years of outstanding service to that organisation.

Described as the "Father of New Zealand Sports Medicine" Dr Norrie Jefferson guided the early development of sports medicine in New Zealand, facilitating dialogue that raised the profile of sport, exercise and medicine from clinical interest to specialist medical practice. As Foundation Chairman, Dr Norrie Jefferson maintained an interest in the activities of SMNZ until health limited his physical capacity to attend meetings. His presence at the 2008 Annual Conference in Dunedin was appropriately acknowledged and he delighted in astute, active dialogue with conference attendees, reflecting on the contribution of medicine to professional sport and public health awareness. Norrie Jefferson died peacefully in Dunedin in 2013 in his hundredth year. His vision was fundamental to the genesis of contemporary sports medicine culture, and his founding philosophy no less applicable today.