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**Best of British**

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# Lost in knowledge translation

CHRIS WHATMAN

A buzz term in research circles at the moment is knowledge translation (KT). While overseas last year I attended a seminar dedicated to KT and integrated knowledge translation (iKT) – an extension of knowledge translation that involved applying the principles of KT to the entire research process. I was astounded by the statement that on average it takes 17 years for research to impact on practice in health care! I wonder if this is the same in the field of sports medicine. It does seem there is substantive evidence in many areas of health care that there is a big gap between what we know and what we do.

Some brief reading around KT identified it certainly is an issue worthy of attention with entire books dedicated to the topic. It has been reported that only around 55% of American adults receive the recommended care from health services.[1] Perhaps one of the issues in this field is the apparently more than 100 terms used when discussing the process of putting research knowledge into action and the many theories and frameworks proposed for achieving knowledge translation which can make it confusing.[2] I did find a definition, which may or may not be useful, the Canadian Institutes of Health Research (CIHR) defines KT as "a dynamic and iterative process that includes the synthesis, dissemination, exchange and ethically sound application of knowledge to improve health, provide more effective health services and products and strengthen the healthcare system", sounds reasonable to me. Authors have made the point that the common theme across all the various terms used in this space is that it goes beyond the simple dissemination of knowledge to the actual use of knowledge – again makes sense. As I noted earlier iKT is the application of KT principles in the entire research process. Basically get the key stake holders (often referred to as the knowledge users) involved in the design of the research questions, decisions regarding methodology, data collection and interpretation of findings. It is thought the research will then more likely target solutions to issues that are a priority for stake holders and thus it is more likely to influence decision making.

Evidence based medicine has been promoted as crucial to achieving better outcomes for patients and it is obviously closely linked to KT. Interestingly it has been suggested that the breakdown of KT may not be in the process of translating the knowledge but in the initial production of the knowledge itself. The knowledge produced by researchers fails to answer the questions that are important to clinicians – there's nothing for the clinicians to "use Monday morning". Focusing energy on improved dissemination of research findings is a wasted effort if the initial questions addressed in the research are not important to the clinicians. Again greater clinician participation in the research at the outset is advocated to improve this. Thus both need to be worked on, iKT is needed to make sure end users are involved in the design of research projects at the outset and better dissemination is needed at the conclusion of projects. Evidence suggests this early engagement is a critical factor in predicting whether research will be used or not.[3]

A major challenge in this space for clinicians and other decision makers in healthcare is the sheer volume of research being produced, over 1000 articles are indexed in Medline daily! Not only is this impossible for clinicians to keep up with but access, time to read and a lack of skills to critically appraise the research findings are often issues. Additionally the content of published papers often doesn't provide sufficient detail in the methods to allow clinicians to use the findings from the study. Researchers need to always keep this in mind and summarise findings in practical terms that are meaningful to clinicians.

A nice example of KT in practice comes from colleagues in Canada where injuries and particularly concussion have become an increasing concern, especially in leagues where body checking (BC) was permitted. Research conducted in collaboration with various hockey associations identified three to four fold increased risk of injury in leagues where BC was permitted. These findings were used to inform policy change in USA hockey where BC is now delayed until players are older. The authors concluded that the collaborative process involved in KT can maximise the impact of the research on reducing the risk of injury in youth sports.

I am aware of various initiatives in New Zealand where research into many aspects of sports injury prevention is being undertaken with a similar collaborative approach, likely ticking many of the KT boxes. There is likely a lack of specific knowledge regarding the underpinning principles and theory of KT but I see it in action everywhere! There are great partnerships with the likes of ACC and many end user sports where researchers and postgraduate students work in the sport and conduct research at the same time. The research questions are driven by the sport (the end knowledge users), the design and collection of the data is a collaborative effort and there are often members from the sport involved in interpretations and implementation of the findings. There is always room for improvement however and we are continually looking for ways to better engage decision makers across the sport and healthcare sector to ensure we are addressing the questions important to them and reflecting on whether or not the research influenced future practice. This is obviously an important issue and iKT should be a priority for all those involved in sport medicine research.

## References


1 McGlynn E, The quality of healthcare delivered to adults in the USA. *New England Journal of Medicine,* 2003. **348**:2635-2645.

2 McKibbon K, C Lokker and N Wilczynski, A cross sectional study of the number and frequency of terms used to refer to knowledge translation in a body of health literature in 2006: a tower of babel? *Implementation Science*, 2010. **5**(16).

3 Cargo M and S Mercer, The value and challenges of participatory research: strengthening its practice. . *Annual Review of Public Health*, 2008. **29**:325-50.

# BEST OF BRITISH - HIGHLIGHTS FROM THE BJSM

CHRIS MILNE

This Best of British column reviews issues from BJSM seen from the latter half of 2017.

In the July issue, there was a useful editorial by Bruno Saragiotto and colleagues entitled "Dispelling the myth that chronic pain is unresponsive to treatment".[1] These authors point out that although there is a lot of emphasis on the biopsychosocial aspects of pain, there seems to be an under appreciation of how important pain intensity is to the average patient. The authors pointed out that this may be due to the growing awareness of the serious harms associated with opioid analgesia. However, they consider that a more constructive and more accurate approach would be to point to the analgesic effects of non-opioid treatment both drug and non-drug, and look to optimise these effects. I would tend to agree.


The next article of interest was entitled "No evidence for the use of stem cell therapy for tendon disorders: a systematic review".[2] Haiko Pas and colleagues performed a systematic review on the topic and found four published and three unpublished trials with a total of 79 patients. They felt that all trials were of high risk of bias and only Level 4 evidence was available. At this stage, they conclude that the use of stem cell therapy for tendon disorders in clinical practice, is currently not advised. Blood-flow restriction training as a clinical rehabilitation tool is something that is being increasingly mentioned in the literature. This involves use of a pressure device to restrict blood-flow to a limb and provide low load resistance training to see if muscle hypertrophy and strength gains can be obtained. The loading can be as low as 30% of one repetition max. These authors found 20 studies and concluded that low load blood-flow restriction training was more effective and tolerable than simple low load training. They emphasise the need for an individualised approach to training prescription to minimise patient risk and increase effectiveness.

The high incidence of injury in rugby union has been a concern for many years. High training volumes are thought to play a role and Shane Ball and colleagues conducted a systematic review on the topic.[3] The authors found 15 studies eligible for inclusion. Overall, injury incidents ranged from 3.3-218 injuries per 1000 player hours in matches and 0.6-6.1 injuries per 1000 player hours in training respectively. Collision events were responsible for the greatest injury incidence and the lower limb was the most common location of injury. These data are consistent with previous data from professional rugby. The other factor that is frequently mentioned in connection with rugby injuries is that players in the professional game have got bigger and faster particularly in the last two decades and from first principle, these factors are likely to increase the severity of injury.

The second July issue started with an editorial entitled "Management of concussion in disability sport: a different ball game?".[4] Liam Richard West and colleagues argue that there are no specific guidelines for athletes with disability with regard to management of their concussion. However, they also mention that there is a paucity of evidence for producing such guidelines and therefore recommend further research and education regarding the diagnosis of concussion in disability sports. Pending that additional evidence, the current Berlin Guidelines should be applied for management of concussion in all athletes.

Sudden cardiac death in young athletes is both rare and dramatic. As a consequence, there has been much study and literature on the topic. Dr Rui Providência and colleagues argue that it may be time to loosen the restrictions on athletes with cardiac disorders competing in sport.[5] Traditionally, doctors have adopted a paternalistic approach and these authors recommend involving the athletes in the decision-making process. They also point out that there are additional complexities for team physicians employed by sporting clubs. Nevertheless, the same empowering approach and obligations apply in this situation. Most clinicians in New Zealand would adopt an inclusive informed consent process, but this article is worthy of study for those of us who have to advise athletes with cardiac disorders on their sport participation.

Exercise has long been touted as a useful therapy for people with inflammatory rheumatic diseases. Sveaas and colleagues examined the effect of cardiorespiratory and strength exercises on disease activity in such patients.[6] They found 26 trials with a total of 1286 participants. They found a small beneficial effect on disease activity scores, with these being reduced by an average of 0.19, and also benefits with regard to joint damage being slowed. There was moderate quality evidence for a small beneficial effect on the ESR dropping by an average of 20% but no effect on C-reactive protein. Beneficial effects were also seen for symptoms.

Rib stress fractures are one of the commonest injuries in rowers and canoeists. Traditional diagnosis has relied on clinical evaluation plus a bone scan or MRI scan. X-rays are useless in the condition. However, a Canadian group found promising results from the use of ultrasound. These Canadian authors Roston et al, found ultrasound to have a sensitivity of 83% and specificity of 76% in detection of metatarsal stress fractures.[7] Unfortunately, no data specific to the ribs is available. These authors showed cortical disruption at the fracture site in several rowers with rib stress injuries. Certainly, this is a modality that has promise for the future.

Issue 15 in August, could be termed the controversy issue. It contained several provocative articles including one by Malhotra on the role of saturated fat in coronary heart disease. These authors argue that coronary heart disease is a chronic inflammatory condition, the risk of which can be effectively reduced via healthy lifestyle interventions. They believe that the role of ideal cholesterol has been exaggerated. Certainly, this issue would be debated by many. The advice in the article to undertake physical activity for at least 150 minutes per week is certainly

something that would have widespread agreement.

Another controversial issue is that of a call to ban the tackle in school rugby in the UK. This issue was aired by BJSM in 2016 and Professor Allyson Pollock & colleagues have provided a rebuttal of an article published by Ross Tucker & colleagues. Viewing this controversy from the opposite side of the world is quite illuminating.[9] Certainly, rugby has such a central role in New Zealand sport and I do not think their arguments would gain much traction here. From what I understand, New Zealand Rugby has been relatively pro-active in starting children off with tag and progressively exposing them to the tackle version. Also, our better summer weather leads to a lot more touch rugby being played in New Zealand, and this provides young athletes with the opportunity to practice their evasion skills, which hopefully will prove useful as they mature into senior players and enable the All Blacks to retain their top ranking in world rugby.

Later in the same issue, is an article by Ken Quarrie and colleagues entitled "Facts and values: on the acceptability of risks in children's sport using the example of rugby – a narrative review".[10] These authors conclude that based on the evidence currently available, the risks to children playing rugby do not appear to be inordinately high compared with those in a range of other childhood sports and activities but better comparative information is urgently needed. This is a perspective I would support.

Acute Achilles tendon ruptures are a relatively common sports injury, and yet we still see cases where the early diagnosis is missed. The article by Dishan Singh recommends consideration of Simmonds triad.[11] This includes looking for the angle of declination of the foot when the person is lying prone on the examination couch, palpating for a gap and then performing a squeeze test. Collectively, two of these three tests were found to be positive in all ruptures suggesting a sensitivity of 100% for this triad. It is worth alerting all clinicians to this paper as sometimes the diagnosis can be tricky.

The August issue opened with an excellent editorial entitled "Misinterpretations of the 'p value': a brief prima for academic sports medicine".[12] We are all exhorted to read literature with a critical eye and the p value has assumed Holy Grail status in recent news. These authors conclude that the p value is a probability under the assumption that a treatment is ineffective. Translation of this probability into a categorical decision is questionable. I would also include the issue of Hills causality criteria. You can Google these, but in essence if a conclusion is biologically plausible, and there is a dose response effect plus a ceiling effect, then it makes it much more likely to be true. A ceiling effect also applies. I would argue that if all of Hills causality criteria apply to a particular study, then possibly the p value could be relaxed to 0.1 rather than the traditionally applied 0.05. Likewise, if the proposition is biologically implausible and there is no dose response observed, then the p value should probably be more exacting eg, 0.01. MRI scans are being increasingly used in musculoskeletal medicine. An article by Adam Weir and colleagues consider the question of MRI investigation for groin pain in athletes.[13] They ask the question "Is radiological terminology clarifying or confusing?". They make the critical point that clinicians need to interpret any radiological findings in the light of their clinical examination. Clearly there is a need for collaboration between clinicians and radiologists to provide the best diagnostic information for the athlete. This can then guide treatment.


Calf muscle strains are common in most running based sport and Brady Green and Tania Pizzari conducted a systematic review of risk factors for injury.[14] They found 10 studies representing 5397 athletes with 518 calf injuries. They found that increasing age and previous calf strain injury were the most predictive of future calf injury. Certainly, as an experienced clinician, I have seen many of these over the years and the most problematic occur in middle aged men who are compulsive runners and probably have developed a bit of soft tissue tightness as they enter middle age. Taking time to evaluate the different contributors to their calf muscle strain and implementing appropriate biomechanical support, can go a long way towards preventing recurrence particularly in this sub-group.

Patellofemoral osteoarthritis is an important source of symptoms in knee OA and is strongly associated with disability. Harvi Hart and colleagues examined the prevalence of radiographic and MRI defined patellofemoral OA.[15] They found 85 studies to evaluate. In their studies, half the people with knee pain or radiographic OA have patellofemoral involvement. Management should be non-surgical in the first instance. If non-invasive means have been fully instituted and the symptoms remain significant, a cortisone injection can provide significant short term relief for a few months if there is a significant life event eg, a major overseas trip in a middle aged or older person. This has been my experience over the last two decades.

Issue 17 in September could well be called the pain issue. It contained two major articles on analgesic use in the athlete. The first of these was the IOC consensus statement on pain management in elite athletes.[16] The article is based on an IOC consensus meeting in November 2016 and runs to 13 pages. It is a comprehensive review of the role of analgesics in pain management in athletes and includes guiding principle plus specifics of medication and management based on pain severity and anticipated return to play. The authors make the point that there is no sound rationality for long term use of NSAIDs in pain management for elite athletes. They also advise against use of opioid medication to treat sub-acute or chronic pain. Certainly, the risks of dependence on opioids are well documented and are a major cause of ill health in the USA. Fortunately, the incidence of this problem is much less in New Zealand. Following that article, there was a narrative review on pain in elite athletes written by Brian Hainline and colleagues.[17] They distinguish between nociceptive pain, neuropathic pain and neoplastic pain. There is a very useful table linking the history and physical examination findings to the likely contributing pain types.

Later in the same issue, was a summary of the sports injury and illness data from the Rio 2016 Olympic Summer Games.[18] Soligard and colleagues studied 11,274 athletes from 207 countries that were competing at the Olympics. Overall, 8% of athletes incurred at least one injury during the Olympic Games and 5% became ill. The figures were slightly lower than the Olympic Summer Games of 2008 and 2012. Most risky sports for injury were BMX cycling, boxing and mountain biking plus Tai Kwan Do.


Later in the same issue, was an article entitled "Neurological tests improve after Olympic-style boxing bouts: a pre-tournament and post-tournament study at the 2016 Womens World Boxing Championships".[19] This statement in the title would tend to go against what one would expect. I am unaware of any data suggesting that being smacked in the head improves your brain function however the authors point out that many of the tests tasks were novel for the boxers so there may have been a practice effect that helped their performance in these tests after they had competed. Certainly, this would be a rational explanation for the results they found.

Hyperandrogenism in female athletes is much in the news these days. Eklund and colleagues studied the serum androgen profile and the physical performance in female Olympic athletes.[20] They compared the data from 106 Swedish female Olympic athletes with age and BMI match sedentary controls. The athletes demonstrated significantly higher levels of the pre-cursor androgens DHEA and 5-DIOL plus higher bone mineral density and greater lean muscle mass. Their data suggests that endogenous androgens are associated with a more anabolic body composition and enhanced performance in female athletes.

The very next article looked at serum androgen levels and their relation to performance in track and field events in both male and female athletes.[21] They found that female athletes with high free testosterone levels had a significant competitive advantage over those with low free testosterone levels in the 400 metres, 400 metre hurdles, 800 metres, hammer throw and pole vault. By contrast, for the male athletes, those with the highest free testosterone concentration did not out-perform those with lower free testosterone concentration in any of the events.

Issue 18 was published in collaboration with the Swiss Sports Medicine Society. The opening editorial by Rodenberg and Holden examined cognition enhancing drugs (nootropics).[22] These include methylphenidate, (better known as Ritalin), modafinil and dextroamphetamine. They contend that drugs that influence cognition have at least some potential to enhance sports performance possibly by improving reaction time as well as possibly via boosting confidence and intensifying aggression. They make the point that athletes are subject to drug testing, but coaches and team officials are not. Certainly, an area for debate and discussion.

GPS technology has been applied to many areas in sport. John Orchard documents its use to measure daily and weekly movement patterns in exercise medicine patients.[23] This technology means that patients are now in a position to self-monitor their entire daily and weekly workloads. He contends that step counting benefits the injured as much as those who are fit. He anticipates that the fitness tracker and mobile app industry will likely soon transition workload recommendations from elite athlete injury research to how members of the general public rehabilitate and manage chronic diseases with exercise.

Shoulder impingement is a very common problem in the community. Steuri and colleagues studied the effectiveness of conservative interventions including exercise, manual therapy and medical management.[24] They analysed 200 articles and found that corticosteroids were superior to control and NSAIDs had a small advantage over placebo. Exercise was superior to doing nothing and specific exercise was superior to non-specific exercise. Manual therapy plus exercise was superior to exercise alone. Laser was superior to sham laser and extracorporeal shockwave therapy also had a positive effect on pain. Overall, they found the quality of evidence to be pretty low, and therefore stated that clinicians should apply the evidence cautiously when making clinical decisions.

Mental health issues are gaining increasing prominence in the sports medicine literature. Gorczynski and colleagues evaluated a range of symptoms in high performance athletes and non-athletes, conducting a comparative meta analysis.[25] They looked at five articles reporting data from 1545 high performance athletes and 1811 non-athletes. They found that both male and female high performance athletes were no more likely than their sedentary counterparts to report mild or more severe depressive symptoms. They noted that male high performance athletes were only half as likely to report mild or more severe depressive symptoms than female high performance athletes. This would accord with the data we are aware of in New Zealand and praise should be given to Sir John Kirwan for his role in improving awareness of depressive illness in men in particular and hopefully reducing the stigma associated with mental health issues.

My pick for the most valuable article in this three month period would be that by Ken Quarrie & colleagues on the acceptability of risks in children's sport using the example of rugby.[10] There is an excellent quote by Richie McCaw – "If you look too closely at the game it's silly really when you bash the hell out of each other. But it's fun and if you took the risk out of everything you wouldn't do anything in your life."

I think this pretty much sums up the way most New Zealanders would feel about our national sport.

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## REFERENCES

1 Saragiotto BT, Maher CG, Traeger AC, et al Dispelling the myth that chronic pain is unresponsive to treatment *Br J Sports Med* 2017; **51**:986-988.

2 Pas HIMFL, Moen MH, Haisma HJ, et al No evidence for the use of stem cell therapy for tendon disorders: a systematic review *Br J Sports Med* 2017; **51**:996-1002.

3 Ball S, Halaki M, Orr R Training volume and soft tissue injury in professional and non-professional rugby union players: a systematic review *Br J Sports Med* 2017; **51**:1012-1020.

4 West LR, Griffin S, Weiler R, et al Management of concussion in disability sport: a different ball game? *Br J Sports Med* 2017; **51**:1050-1051.

5 Providência R, Teixeira C, Segal O, et al Is it time to loosen the restrictions on athletes with cardiac disorders competing in sport? *Br J Sports Med* 2017; **51**:1056-1057.

6 Sveaas SH, Smedslund G, Hagen KB, et al Effect of cardiorespiratory and strength exercises on disease activity in patients with inflammatory rheumatic diseases: a systematic review and meta-analysis *Br J Sports Med* 2017; **51**:1065-1072.

7 Roston AT, Wilkinson M, Forster BB Imaging of rib stress fractures in elite rowers: the promise of ultrasound? *Br J Sports Med* 2017; **51**:1093-1097.

8 Malhotra A, Redberg RF, Meier P Saturated fat does not clog the arteries: coronary heart disease is a chronic inflammatory condition, the risk of which can be effectively reduced from healthy lifestyle interventions *Br J Sports Med* 2017; **51**:1111-1112.

9 Pollock AM, White AJ, Kirkwood G Evidence in support of the call to ban the tackle and harmful contact in school rugby: a response to World Rugby *Br J Sports Med* 2017; **51**:1113-1117.

10 Quarrie KL, Brooks JHM, Burger N, et al Facts and values: on the acceptability of risks in children's sport using the example of rugby — a narrative review *Br J Sports Med* 2017; **51**:1134-1139.

11 Singh D Acute Achilles tendon rupture *Br J Sports Med* 2017; **51**:1158-1160.

12 Stovitz SD, Verhagen E, Shrier I Misinterpretations of the 'p value': a brief primer for academic sports medicine *Br J Sports Med* 2017; **51**:1176-1177.

13 Weir A, Robinson P, Hogan B, et al MRI investigation for groin pain in athletes: is radiological terminology clarifying or confusing? *Br J Sports Med* 2017; **51**:1185-1186.

14 Green B, Pizzari T Calf muscle strain injuries in sport: a systematic review of risk factors for injury *Br J Sports Med* 2017; **51**:1189-1194.

15 Hart HF, Stefanik JJ, Wyndow N, et al The prevalence of radiographic and MRI-defined patellofemoral osteoarthritis and structural pathology: a systematic review and meta-analysis *Br J Sports Med* 2017; **51**:1195-1208.

16 Hainline B, Derman W, Vernec A, et al International Olympic Committee consensus statement on pain management in elite athletes *Br J Sports Med* 2017; **51**:1245-1258.

17 Hainline B, Turner JA, Caneiro JP, et al Pain in elite athletes—neurophysiological, biomechanical and psychosocial considerations: a narrative review *Br J Sports Med* 2017; **51**:1259-1264.

18 Soligard T, Steffen K, Palmer D, et al Sports injury and illness incidence in the Rio de Janeiro 2016 Olympic Summer Games: A prospective study of 11274 athletes from 207 countries *Br J Sports Med* 2017; **51**:1265-1271.

19 Howell DR, Meehan WP, Loosemore MP, et al Neurological tests improve after Olympic-style boxing bouts: a pretournament and post-tournament study in the 2016 Women's World Boxing Championships *Br J Sports Med* 2017; **51**:1279-1284.

20 Eklund E, Berglund B, Labrie F, et al Serum androgen profile and physical performance in women Olympic athletes *Br J Sports Med* 2017; **51**:1301-1308.

21 Bermon S, Garnier P Serum androgen levels and their relation to performance in track and field: mass spectrometry results from 2127 observations in male and female elite athletes *Br J Sports Med* 2017; **51**:1309-1314.

22 Rodenberg RM, Holden JT Cognition enhancing drugs ('nootropics'): time to include coaches and team executives in doping tests? *Br J Sports Med* 2017; **51**:1316.

23 Orchard JW Using technology to measure daily and weekly movement patterns in exercise medicine patients Br J Sports Med 2017;51:1317-1318.

24 Steuri R, Sattelmayer M, Elsig S, et al Effectiveness of conservative interventions including exercise, manual therapy and medical management in adults with shoulder impingement: a systematic review and meta-analysis of RCTs *Br J Sports Med* 2017; **51**:1340-1347.

25 Gorczynski PF, Coyle M, Gibson K Depressive symptoms in high-performance athletes and non-athletes: a comparative meta-analysis *Br J Sports Med* 2017; **51**:1348-1354.

# Sport-related drinking motives of professional rugby league and rugby union players

MICHEAL LUCK, GEOFF DICKSON, MICHAEL NAYLOR


## ABSTRACT

### Aim

This study investigated 1) the validity and reliability of the Athlete Drinking Scale (ADS) in the context of professional rugby union and rugby league players in Australasia; 2) differences between the Sport-Related Drinking Motives (SRDM) of professional rugby union and rugby league players in Australasia.

### Method

A modified version of the ADS was developed to suit the professional sport context and completed by a sample of professional rugby union and league players (n = 193) from Australia and New Zealand. The results of confirmatory factor analysis (CFA) suggested a three factor, nine-item scale was most appropriate to measure the construct.

### Results

Compared to rugby league players, rugby union players reported higher levels of both positive reinforcement and team/group drinking motives. The respondents in this study identified SRDM of similar magnitude to samples in alternate settings reported elsewhere.

### Discussion

The revised ADS has fewer items than the original ADS. Athletes in different sports are likely to have differing levels of sport-related drinking motivations.



### Keywords

drinking motives, alcohol, sport, rugby league, rugby union



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## INTRODUCTION

In recent years, rugby union and rugby league athletes in Australasia have experienced relentless public and media scrutiny of their alcohol consumption, binge drinking and what might best be described as alcohol-fuelled incidents.[1-3] Alcohol negatively affects athletic performance.[4] Alcoholism is also prevalent amongst professional footballers.[5] Together, these provide a compelling case to better understand the sport-related drinking motives (SRDM) of professional footballers.

Drinking motives explain a significant amount of variance in drinking behaviours in athletes including peak drinking, consequences, personal problems and social problems.[6] The Athlete Drinking Scale (ADS)[7] is central to SRDM research. The ADS comprises three motive subscales. Positive Reinforcement (PR) occurs when athletes drink alcohol to enhance positive feelings. Team/Group (TG) refers to efforts to strengthen or enhance a player's standing in a team or group setting. Finally, when an athlete uses alcohol to cope with unfavourable on-field results or performances, this is referred to as Sport-Related Coping (SRC).

Most SRDM knowledge is derived from studies of amateur athletes competing in the United States of America's National Collegiate Athletic Association (NCAA)[6–8] and American high school sport.[9] A notable exception is research conducted on a cross-section of New Zealand athletes.[10] No research has yet explored the SRDM of professional athletes. There are a number of unique characteristics differentiating professional footballers from high-school and college students. Professional athletes are obviously paid, and are typically older. Emotional maturity, self-image and judgement evolve until the brain's prefrontal cortex is fully developed. Longitudinal neuroimaging studies demonstrate that the adolescent brain continues to mature well beyond teenage years.[11]

Therefore, determining the utility of the ADS among professional athletes before assuming its applicability to this population is worthwhile. The specific objectives of the research are to: 1) Test the validity and reliability of the ADS in the context of professional rugby union and rugby league players in Australasia; 2) Identify differences between the SRDM of professional rugby union and rugby league players in Australasia. We hypothesise that the factor structure of the ADS for professional athletes will be consistent with student-athlete research. We also hypothesise that, given the similarities between the NRL and Super Rugby environments, there will be no differences in the sport related drinking motives between NRL and Super Rugby players.

## METHODS

### Participants

Participants were professional athletes recruited from all 16 National Rugby League (NRL) teams and from New Zealand's five Super Rugby teams. The NRL is the world's elite rugby league competition.

The premiership is contested by sixteen teams, fifteen in Australia with one in New Zealand. The NRL and its clubs receive significant revenue from sponsorships and broadcasting agreements. The majority of players are fulltime professionals. Super Rugby is the pre-eminent professional men's rugby union football competition in the Southern Hemisphere and Japan.[12] Like the NRL, the Super Rugby competition is highly commercialised and the players well remunerated.

### Procedures

The Rugby League Professionals Association (RLPA) and the New Zealand Rugby Players Association (NZRPA) facilitated access to each group of players. The RLPA initially contacted all NRL players who had supplied an email (n = 342) and followed up three weeks later with a reminder. In the end, 110 rugby league questionnaires were submitted of which 105 had no missing data and were therefore carried forward to the analysis. The NZRPA advised the researchers to develop a hard copy of the questionnaire that they would distribute during NZPRA-team meetings. Of the 84 submitted, 74 questionnaires were carried forward to the analysis because they had no missing data. Therefore, the total sample was 179. The survey was anonymised.

### Measures

The first section of the survey queried basic demographics, and playing career data. The second section of the questionnaire featured a modified version of the 19-item ADS.[7] Consistent with previous research,[8,13] respondents indicated their level of statement agreement on a scale ranging from 1 (strongly disagree) to 6 (strongly agree). The ADS contains three subscales: Positive reinforcement (9 items), team/group (7 items) and sport-related coping (3 items). It was thought that rigorous psychometric testing could result in a shorter version of the ADS for use among professional athletes – particularly for measuring PR and TG. A panel, with expertise in scale development and professional sport, reviewed the original ADS items. The panel recommended the alteration of five items for context appropriateness. The modified ADS items are listed in Table 1. The Auckland University of Technology Ethics Committee provided ethical approval.

**Table 1:** The Athlete Drinking Scale (ADS)

| | Original SRDM Items | Modified SRDM Items |
|---|---|---|
| PR1 | I enjoy the feeling of getting drunk | |
| PR2 | I drink to have a good time with my teammates | |
| PR3 | After a game/match/meet, it is important for me to go out and celebrate with alcohol | After a game, it is important for me to go out and celebrate with alcohol |
| PR4 | Because I work so hard at my sport, I should be able to drink to have a good time | |
| PR5 | I drink to celebrate athletic victories | I drink to celebrate a victory |
| PR6 | I get a rush out of becoming drunk | |
| PR7 | If I've performed well, I feel like I can go out and drink a little more than usual | |
| PR8 | Winning or performing well is a good reason to go out and drink | |
| PR9 | I drink because I believe in a "work hard, play hard" lifestyle | |
| TG1 | I drink to "fit in" with my teammates | |
| TG2 | When drinking alcohol with teammates, it becomes a competition | |
| TG3 | I feel pressure from my teammates to drink alcohol | |
| TG4 | Alcohol use is an important part of the athletic culture at this institution | Alcohol is an important part of my team's culture |
| TG5 | I drink because it's part of the culture of being an athlete | |
| TG6 | I drink because it helps our team develop cohesion | |
| TG7 | I drink because my teammates expect me to drink with them | |
| SRC1 | I drink to help me deal with poor performances | |
| SRC2 | I drink to deal with sport-related stress | I drink to deal with football related stress |
| SRC3 | I tend to drink more when I'm not performing well athletically | I tend to drink more when I'm not performing well on the field |

### Analysis

Multivariate normality was examined to inform the decision of which estimator to utilise for ADS factor analysis. Using MPlus statistical software, we conducted Confirmatory Factor Analysis (CFA) to assess the reliability and validity of the scale. We used the Tucker-Lewis Index (TLI), Comparative Fit Index (CFI), Standardised Root Mean Square Residual (SRMR) and Root Mean Square Error of Approximation (RMSEA) – to provide a thorough assessment of model fit.[14] For the TLI and CFI, a cut off of .95 was used. The cut-off values for SRMR and RMSEA were .08 and .06 respectively.[15] In addition to examining fit indices, other aspects of the CFA were interpreted. Variables with cross-loadings greater than .50 were considered for removal from the analysis.[15] Additional item and construct level analysis is needed in the form of the construct reliability (CR) statistic as well as average variance extracted (AVE). For CR, a value above .70 is preferred,[16] while for AVE, a value greater than or equal to .50 is best.[17] Potential relationships between the independent variable (ie, sport) and SRDM dimensions were explored using analysis of variance (ANOVA) and multivariate analysis of variance (MANOVA). Pillai's Trace was used throughout the MANOVA analysis. For the group difference testing, significance of effect was interpreted at $p < .05$. Effect sizes were calculated for noteworthy relationships.

## RESULTS

The assumption of multivariate normality among SRDM items was violated, with the skewness (532.37) and kurtosis (926.29) values both significant ($p < .05$). This was not unexpected and we therefore took the traditional approach of using maximum likelihood parameter estimates with standard errors and a mean-adjusted chi-squared statistic (MLM).[14] The results of the initial CFA were problematic.

The data did not fit the hypothesised model well. ($\chi 2 = 300.99$, df = 149 $p < .01$). The RMSEA was high at 0.10, CFI was low at .81, TLI was low at .78, and SRMR was high at 0.09. To identify the sources of poor model fit, further item and construct analyses were needed. The AVE scores for all three dimensions were below .50. The CR for PR (0.89) and TG (0.83) were acceptable, but low for SRC (0.36). The high correlations between PR and TG (0.80), and TG and SRC (0.69) suggested that items were overlapping within these constructs. Prior to the second CFA, seven items were discarded for various reasons in line with the objective of refining and shortening the ADS for use in this alternative context. Items PR9 (0.42), TG1 (0.48), TG3 (0.57) and SRC3 (0.02) were removed because of low factor loadings. Items PR9 and TG7 were removed because they cross-loaded. Items PR7 and PR2 were removed for being highly correlated with other PR items. All suspect items were carefully examined to ensure that their removal did not compromise factor meaning.[14]

A second model was estimated and resulted in improved fit indices ($\chi 2 = 65.10$ df = 51, $p = 0.9$). The RMSEA score of .05 and the SRMR score of .06 were both acceptable. CFI was .97 and TLI was .96, both indicating model fit. Compared to the initial CFA, AVE values were improved considerably (PR = 0.53, TG = 0.49, SRC = 0.54), although the TG factor AVE score is technically below the benchmark of .50. The two largest correlations decreased after model re-specification. PR and TG went down to 0.75, while TG and SRC decreased to 0.56. After this step, a further three items were removed for the specification of the third and final model. Items PR1 and PR6 were removed principally in the interest of creating a scale with fewer items. It was determined that the underlying meaning of the PR factor was not compromised by their removal and that inherent meaning was captured within the remaining items. Similarly, the removal of TG2 increased model fit without compromising the TG factor.

The fit indices from a third CFA provided evidence of further model fit improvement ($\chi 2 = 29.95$ df = 24, $p = 0.19$). In addition, the RMSEA (.05), SRMR (.05), CFI (.98) and TLI (.97) scores also indicated excellent model fit. All three AVE scores are above the 0.5 benchmark, and the CR statistics were above .70. The correlation between PR and TG rose slightly (to 0.81), as did the correlation between TG and SRC (0.53). Overall, these results are an indication that the refined ADS was valid and reliable and that the three factors were interpreted sufficiently differently by the footballers (ie, discriminant validity).

Composite variables were created based on items retained through the results of CFA. A one-way, independent MANOVA assessed any SRDM differences between league and union respondents on the three ADS dimensions. Mean scores are presented in Table 2.

There was a significant effect for league/union on SRDM, $F(3,188) = 4.88$, $p < .05$. ANOVA identified a statistically significant difference between the league and union respondents for the PR, $F(1, 190) = 10.40$, $p < .01$) and TG composite variables $F(1, 190) = 7.36$, $p < .01$). In both instances, the PR and TG motives were higher for rugby union players than rugby league players. The effect sizes for the PR composite variable (0.46) and for TG (-.39) are near the medium range (Cohen, 1998). There was no significant difference for the SRC composite variable $F(1, 190) = 0.00$, $p = .98$).

**Table 2:** Mean Scores for ADS Composite Variables by Sport

| SRDM | Sport | n | Mean | Std Deviation |
|---|---|---|---|---|
| Positive Reinforcement | League | 109 | 3.02 | 1.14 |
| | Union | 83 | *3.55 | 1.15 |
| Team / Group | League | 109 | 2.28 | 0.99 |
| | Union | 83 | *2.68 | 1.06 |
| Sport-Related Coping | League | 109 | 1.88 | 1.13 |
| | Union | 83 | 1.88 | 1.04 |

*Statistically Significant Difference *(p < .01)*

## DISCUSSION

This study investigated the sport-related drinking motives of professional rugby union and league players. The first objective was to test the validity and reliability of the ADS in the context of professional rugby league and rugby union in Australasia. The second objective was to identify SRDM differences between professional rugby union players and rugby league players.

The findings contribute to our understanding and measurement of SRDM in a number of ways. First, the original ADS is not well suited for professional sport settings. Five items required re-wording to make the ADS more relevant to respondents. The result of careful re-wording and psychometric testing, was a valid and reliable scale incorporating the original three factors, but with a reduced number of items. The next contribution is that the PR motives can differ between type of sport. More specifically, the PR motives are higher for rugby union players when compared to rugby league players. One possible explanation is that rugby league players have means other than alcohol to celebrate or acquire positive reinforcement. Another explanation is that rugby league players do not need to win or perform well to justify alcohol consumption. An additional contribution to our understanding of SRDM is that team/group motives for union players are higher than league players. Rugby union has always been characterised by its amateur foundations,[18] its camaraderie,[19] and after match sociability.[20] Rugby is no longer amateur at the elite level, but "its traditions, passion, pride and 'camaraderie' are elements even the most instrumental technically rational coaches would never completely expunge from their coaching rationale".[19]

## CONCLUSION

This research provides novel and important insight into the measurement of SRDM amongst professional athletes. The revised ADS has sound psychometric properties and can be used to assess SRDM across at least two types of professional team sport – rugby league and union. The revised ADS has fewer items than the original ADS. Athletes in different sports are likely to have differing levels of sport-related drinking motivations.

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## REFERENCES

1 Smith A, Stavros C, Westberg K, et al., Alcohol-related player behavioral transgressions: Incidences, fan media responses, and a harm-reduction alternative. *Int Rev Sociol Sport*, 2014; **49**(3–4):400–416.

2 O'Brien K, Kolt G, Martens M, et al., Alcohol-related aggression and antisocial behaviour in sportspeople/athletes. *J Sci Med Sport*, 2012; **15**(4):292–297.

3 O'Brien K, Miller P, Kolt G, et al., Alcohol industry and non-alcohol industry sponsorship of sportspeople and drinking. *Alcohol and Alcoholism*. 2011; **46**(2): 210–213.

4 Prentice C, Stannard S, Barnes M, Effects of heavy episodic drinking on physical performance in club level rugby union players. *J Sci Med Sport*. 2015; **18**(3):268–271.

5 Jones C. Alcoholism and recovery: A case study of a former professional footballer. *Int Rev Sociol Sport*. 2014; **3–4**:485–505.

6 Martens M, Labrie J, Hummer J, et al., Understanding sport-related drinking motives in college athletes: Psychometric analyses of the Athlete Drinking Scale. *Addict Behav*. 2008; **33**(7):974–977.

7 Martens M, Watson J, Royland E, et al., Development of the athlete drinking scale. *Psychol Addict Behav*. 2005; **19**(2):158–164.

8 Martens M, Martin J, College athletes' drinking motives and competitive seasonal status: Additional examination of the athlete drinking scale. *Addict Res Theory*. 2010; **18**(1):23–32.

9 Herring T, Zamboanga B, Olthuis J, et al., Utility of the Athlete Drinking Scale for assessing drinking motives among high school athletes. *Addict Behav*. 2016; **60**:18–23.

10 O'Brien KS, Ali A, Cotter JD, et al., Hazardous drinking in New Zealand sportspeople: Level of sporting participation and drinking motives. *Alcohol and Alcoholism*. 2007; **42**(4):376–82.

11 Johnson S, Blum R, Giedd J. Adolescent maturity and the brain: The promise and pitfalls of neuroscience research in adolescent health policy. *J Adolesc Heal*. 2009; **45**(3):216–21.

12 Meiklejohn T, Dickson G, Ferkins L. The formation of interorganisational cliques in New Zealand rugby. *Sport Manag Rev*. 2015; **19**(3):266–78.

13 Martens M, Pedersen E, Smith A, et al., Predictors of alcohol-related outcomes in college athletes: The roles of trait urgency and drinking motives. *Addict Behav*. 2011; **36**(5):456–64.

14 Kline R. Principals and practice of structural equation modelling. New York: Guildford; 2005.

15 Hu L, Bentler P. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. Struct Equ Model A Multidiscip J. 1999;6(1):1–55.

16 Fornell C, Larcker D. Evaluating structural equation models with unobservable variables and measurement error. *J Mark Res*. 1981; **18**(1):39–50).

17 Bagozzi R, Yi Y. On the Evaluation of Structural Equation Models. *J Acad Mark Sci*. 1988; **16**(1):79–94.

18 O'Brien D, Slack T. The emergence of a professional logic in English Rugby Union: The role of isomorphic and diffusion processes. *J Sport Manag*. 2004; **18**(1):13–39.

19 Horton P. Rugby union football in Australian society: An unintended consequence of intended actions. *Sport Soc*. 2009; **12**(7):967–85.

20 Donnelly P, Young K. Reproduction and transformation of cultural forms in sport: A contextual analysis of Rugby. *Int Rev Sociol Sport*. 1985; **20**(1–2):21–37.

ORIGINAL RESEARCH

# Injuries in New Zealand amateur rugby league matches by positional groups

CLOE CUMMINS, DOUG KING, TREVOR CLARK


## ABSTRACT

### Aim

To investigate the incidence, site, nature, and severity of injuries of a division 1 amateur rugby league team in New Zealand by positional groups.

### Study Design

Observational prospective cohort study.

### Subjects

A total of 145 amateur rugby league players of a premier level division 1 amateur rugby league team participated in this study over two consecutive competition seasons.

### Outcome Measures

For each match-play injury it was recorded; how the injury occurred; how many games, if any were subsequently missed; and the type, site and severity of injury. Injuries were reported as rate per 1,000 hours, also broken down into severity according to the number of games missed.

### Results

Over the duration of the study there were 377 injuries recorded with a total injury rate of 419.3 per 1,000 match hr. There was a significant difference observed that hit-up forwards recorded more lower limb injuries than adjustables (RR: 1.6 [95% CI: 1.0 to 2.6] p=0.044) but not significantly more than outside backs (RR: 1.3 [95% CI: 0.9 to 2.1] p=0.210). Hit-up forwards recorded more injuries as the ball-carrier than the tackler, whilst outside backs and adjustables recorded more injuries as the tackler than the ball carrier.

### Conclusions

This study has identified that there are positional differences in the rate, site and type of injuries that occur during amateur rugby league match-play. The study further identifies that rugby league match-play is associated with high injury rates and that additional injury prevention strategies designed to reduce the incidence of injury in rugby league are required.



### Keywords

amateur, rugby league, New Zealand, injury incidence



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## INTRODUCTION

Played internationally, rugby league is an intermittent contact-based team sport where players are required to undertake repeated bouts of high-intensity activity (sprinting and high-speed running) interspersed with bouts of lower intensity activity (standing, walking and jogging).[10] Additionally players frequently engage in physically demanding contact and wrestle bouts.[12] Indeed, during a rugby league match forwards are reported to be involved in an average of 59 physical collisions (42 tackles, 17 hit ups) whilst backs are involved in an average of 40 physical collisions (24 tackles, 16 hit ups).[1] As a result of these physical collisions there is a risk of musculoskeletal injury occurring during match-play.[22] Understanding the injury risks are important for those playing the game as well as for those who provide health care for these participants, both in terms of injury prevention and injury management. Knowledge of these risks can be utilised in the production and promotion of injury prevention strategies that may assist in reducing the risk of injury for participation in contact sports such as rugby league.

Several studies have documented the rate of injuries that occur at the amateur, semi-professional and professional levels of participation.[21] These injury rates vary according to the level of participation with elite players typically but not always having a higher incidence of injury than non-elite players.[22] When injuries that result in missed matches are considered, the injury rate varies from 27 to 68 per 1,000 match hr;[22] however, when all injuries (including transient injuries) are considered, the injury rate is reported to be as high as 823 per 1,000 match hr.[22] Despite this knowledge no study to date has attempted to describe the difference in injury risk between amateur rugby league playing positions throughout match-play. Therefore, the purpose of this study was to record and document the injuries sustained within a premier level division 1 amateur rugby league team and to compare these by positional groups.

## METHODS

A prospective observational cohort study was undertaken to document the incidence of injuries occurring in an amateur rugby league team over two consecutive domestic seasons. All procedures in the study were approved (AUTEC 08/44) and conducted in accordance with principles of the Declaration of Helsinki. The competition seasons were undertaken from March through to August each year. The team participated in the premier level of a division 1 regional domestic competition. All registered players were considered amateur as they derived their main source of income

from other means and did not receive match payments. A total of 145 amateur rugby league players (mean± S.D. age, 26.3±9.1 yr.; height, 1.79±0.04 m; mass, 95.3±10.9 kg) participated in this study. Data were collected from 54 hit-up forwards (prop, n=28; second row, n=26), 35 adjustables (hooker, n=9; halfback, n=10, five-eighth, n=7; loose forward, n=9) and 56 outside backs (centre, n=27; wing, n=16; fullback, n=13).

Over the duration of both seasons, all match injuries from the team being observed were recorded on a standardised injury reporting form regardless of severity.[17] The same team medic was with the team throughout the duration of the study. The team medic, a registered comprehensive nurse (with tertiary sports medicine qualifications and accredited in injury prevention, assessment, and management) assessed and recorded all the injuries that occurred. Only injuries that were identified by a qualified health professional were recorded. Injury data was collected from all matches the team participated in, which included preseason fixtures and all competition matches, including the final series. Injuries that resulted in more than one injury type were recorded as one injury event. Player position was recorded as the position that the player was undertaking at the time the injury occurred.[16] The injury definition utilised in this study has been previously defined,[19] with injuries classified according to the site, nature, and severity of the injury recorded.[18]

### Statistical Analysis

Over the study period, 52 player matches were completed and all were 80 minutes (1.33 hours) in duration with an overall injury risk exposure of 899.1 match hr. Injury rates were calculated as previously described.[14] To compare multiple counts, 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. Data were reported as rates per 1,000 match hr. with 95% confidence intervals (CI's). To compare between injury rates, risk ratios (RR's) were used. The RR's were assumed to be significant at $p<0.05$.

## RESULTS

As seen in Table 1 over the duration of the study there were 377 (2008: 163 and 2009:

**Table 1.** Total and missed match injury statistics for New Zealand amateur rugby league participants by positional groups per season for injuries observed, injuries expected, injury rate, exposure hours, player appearances, per game and per game minutes played by rates per 1000 match hours with 95% confidence interval.

| | 2008 | | | | 2009 | | | | TOTAL | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | OSB | ADJ | HUF | Total | OSB | ADJ | HUF | Total | OSB | ADJ | HUF | Total |
| **TOTAL INJURIES** | | | | | | | | | | | | |
| Injuries Observed | 47 | 48[2] | 68 | 163 | 61 | 77[1] | 76 | 214 | 108 | 125[b] | 144[a] | 377 |
| Injuries Expected | 49.8 | 57.7 | 66.5 | 174.0 | 58.2 | 67.3 | 77.5 | 203.0 | 108 | 125 | 144 | 377 |
| Injury rates per 1000 | 294.5 | 375.9 | 532.6 | 392.8 | 327.6 | 516.9 | 510.2 | 442.0 | 312.3 | 451.9 | 520.5 | 419.3 |
| match hr. (95% CI) | (221.3-391.9) | (283.3-498.9) | (419.9-675.5) | (336.9-458.0) | (254.9-421.1) | (413.4-646.3) | (407.5-638.8) | (386.6-505.4) | (258.6-377.1) | (379.2-538.4) | (442.1-612.9) | (379.1-463.9) |
| No of games played | 24 | 24 | 24 | 24 | 28 | 28 | 28 | 28 | 52 | 52 | 52 | 52 |
| Exposure hrs | 159.6 | 127.7 | 127.7 | 415.0 | 186.2 | 149.0 | 149.0 | 484.1 | 345.8 | 276.6 | 276.6 | 899.1 |
| Hrs per injury (95% CI) | 3.4 | 2.7 | 1.9 | 2.5 | 3.1 | 1.9 | 2.0 | 2.3 | 3.2 | 2.2 | 1.9 | 2.4 |
| | (2.6-4.5) | (2.0-3.5) | (1.5-2.4) | (2.2-3.0) | (2.4-3.9) | (1.5-2.4) | (1.6-2.5) | (2.0-2.6) | (2.7-3.9) | (1.9-2.6) | (1.6-2.3) | (2.2-2.6) |
| Total No. Injuries per | 2.0 | 2.0 | 2.8 | 6.8 | 2.2 | 2.8 | 2.7 | 7.6 | 2.1 | 2.4 | 2.8 | 7.3 |
| game (95% CI) | (1.5-2.6) | (1.5-2.7) | (2.2-3.6) | (5.8-7.9) | (1.7-2.8) | (2.2-3.4) | (2.2-3.4) | (6.7-8.7) | (1.7-2.5) | (2.0-2.9) | (2.4-3.3) | (6.6-8.0) |
| Player appearances | 6.6 | 6.5 | 4.6 | 1.9 | 6.0 | 4.7 | 4.8 | 1.7 | 6.3 | 5.4 | 4.7 | 1.8 |
| per injury (95%CI) | (5.0-8.8) | (4.9-8.6) | (3.6-5.8) | (1.6-2.2) | (4.6-7.7) | (3.8-5.9) | (3.8-6.0) | (1.5-1.9) | (5.2-7.6) | (4.5-6.4) | (4.0-5.5) | (1.6-2.0) |
| Game minutes played | 40.9 | 40.0 | 28.2 | 11.8 | 36.7 | 29.1 | 29.5 | 10.5 | 38.5 | 33.3 | 28.9 | 11.0 |
| per injury (95%CI) | (30.7-54.4) | (30.1-53.1) | (22.3-35.8) | (10.1-13.7) | (28.6-47.2) | (23.3-36.4) | (23.5-36.9) | (9.2-12.0) | (31.9-46.5) | (27.9-39.7) | (24.5-34.0) | (10.0-12.2) |
| **MISSED MATCH INJURIES** | | | | | | | | | | | | |
| MM Injuries Observed | 4[2] | 3[2] | 5[2] | 12.0 | 14[1] | 13[1] | 17[1] | 44 | 18 | 16 | 22 | 56 |
| MM Injuries Expected | 8.3 | 7.4 | 10.2 | 25.8 | 9.7 | 8.6 | 11.8 | 30.2 | 18 | 16 | 22 | 56 |
| MM Injury rates per | 9.6 | 7.2 | 12.0 | 28.9 | 28.9 | 26.9 | 35.1 | 90.9 | 52.1 | 57.8 | 79.5 | 62.3 |
| 1000 match hr (95% CI) | (3.6-25.7) | (2.3-22.4) | (5.0-28.9) | (16.4-50.9) | (17.1-48.8) | (15.6-46.2) | (21.8-56.5) | (67.6-50.9) | (32.8-82.6) | (35.4-94.4) | (52.4-120.8) | (47.9-80.9) |
| No of games played | 24 | 24 | 24 | 24 | 28 | 28 | 28 | 28 | 52 | 52 | 52 | 52 |
| Exposure hrs | 159.6 | 127.7 | 127.7 | 415.0 | 186.2 | 149.0 | 149.0 | 484.1 | 345.8 | 276.6 | 176.6 | 899.1 |
| Hrs per MM injury | 39.9 | 42.6 | 25.5 | 34.6 | 13.3 | 11.5 | 8.8 | 11.0 | 19.2 | 17.3 | 12.6 | 16.1 |
| (95% CI) | (15.0-106.3) | (13.7-132.0) | (10.6-61.4) | (19.6-60.9) | (7.9-22.5) | (6.7-19.7) | (5.4-14.1) | (8.2-14.8) | (12.1-30.5) | (10.6-28.2) | (8.3-19.1) | (12.4-20.9) |
| Total No. MM Injuries | 0.2 | 0.1 | 0.2 | 0.5 | 0.5 | 0.5 | 0.6 | 1.6 | 0.3 | 0.3 | 0.4 | 1.1 |
| per game (95% CI) | (0.1-0.4) | (0.0-0.4) | (0.1-0.5) | (0.3-0.9) | (0.3-0.8) | (0.3-0.8) | (0.4-1.0) | (1.2-2.1) | (0.2-0.5) | (0.2-0.5) | (0.3-0.6) | (0.8-1.4) |
| Player appearances | 78.0 | 104.0 | 62.4 | 26.0 | 26.0 | 28.0 | 21.4 | 8.3 | 37.6 | 42.3 | 30.7 | 12.1 |
| per MM injury (95%CI) | (29.3-207.8) | (33.5-322.5) | (26.0-149.9) | (14.8-45.8) | (15.4-43.9) | (16.3-48.2) | (13.3-34.4) | (6.2-11.1) | (23.7-59.6) | (25.9-69.0) | (20.2-46.7) | (9.3-15.7) |
| Game minutes played | 480.0 | 640.0 | 384.0 | 160 | 160.0 | 172.3 | 131.8 | 50.9 | 231.1 | 260.0 | 189.1 | 74.3 |
| per MM injury (95%CI) | (180.1-1278.9) | (206.4-1984.4) | (159.8-922.6) | (90.9-281.7) | (94.8-270.2) | (100.1-296.7) | (81.9-212.0) | (37.9-68.4) | (145.6-366.8) | (159.3-424.4) | (124.5-287.2) | (57.2-96.5) |

CI: Confidence Interval. MM: Missed Match. Significant difference ($p<0.05$) than: (a) adjustables; (b) hit-up forwards; (1) 2008; (2) 2009

214) injuries recorded with a total injury rate of 419.3 (95% CI: 379.1 to 463.9) per 1000 match hr. There was a difference observed that more injuries were recorded in 2009 (442.0 [95% CI: 386.6 to 505.4] per 1000 match hr.) than 2008 (392.8 [95% CI: 336.9 to 458.0] per 1000 playing hours; RR: 1.3 [95% CI: 1.1 to 1.6], p=0.009). There were fewer missed match injuries with a total missed match injury rate of 62.3 (95% CI: 47.9 to 80.9) per 1000 match hr. There was a three-fold difference observed that more missed match injuries were recorded in 2009 (90.9 [95% CI: 50.9 to 67.6] per 1000 match hr) than 2008 (28.9 [95% CI: 16.4 to 50.9] per 1,000 match hr; RR: 3.7 [95% CI: 1.9 to 6.9]). There was a four-fold increased risk of injuries for adjustables (RR: 4.3 [95% CI: 1.2 to 15.2], p=0.012) and a three-fold increased risk for outside backs (RR: 3.5 [95% CI: 1.2 to 10.6], p=0.018) and hit-up forwards (RR: 3.4 [95% CI: 1.3 to 9.2], p=0.011) for missed match injuries.

Hit up forwards (520.5 [95% CI: 442.1 to 612.9] per 1000 match hr) recorded more total injuries than adjustables (506.1 [95% CI: 363.4 to 704.8] per 1000 match hr; RR: 1.3 [95% CI: 1.1 to 1.7] p=0.023) and outside backs (312.3 [95% CI: 258.6-377.1] per 1000 playing hours; RR: 1.2 [95% CI: 0.9 to 1.4] p=0.247) (see Table 2). More injuries were recorded to the upper (155.7 [95% CI: 131.9 to 183.8] per 1000 match hr) than the lower (115.7 [95% CI: 95.4 to 140.2] per 1000 match hr; p=0.021) limb, the head and neck (90.1 [95% CI: 72.5 to 112.0] per 1000 match hr; p<0.001) and the chest and back (57.8 [95% CI: 44.1 to 75.9] per 1000 match hr; p<0.001) throughout the duration of the study. There was a notable difference that adjustables recorded fewer lower limb injuries (97.6 [95% CI: 66.9 to 142.3] per 1000 match hr) than hit-up forwards (159.1 [95% CI: 118.4 to 213.7] per 1,000 match hr; RR: 1.6 [95% CI: 1.0 to 2.6] p=0.044). Adjustables had a two-fold (RR: 2.5 [95% CI: 0.5 to 12.9] p=0.257) increased risk of concussion when compared with hit-up forwards, and a three-fold (RR: 3.5 [95% CI: 0.7 to 16.8] p=0.096) increased risk of concussion when compared with outside backs. As highlighted in Table 2 the tackle was identified as the most common cause of injury for all player positions. There was a two-fold increased risk of injury for the ball carrier, when compared with the tackler, for outside backs (RR: 1.7 [95% CI: 1.2 to 2.6] p=0.009) and hit-up forwards (RR: 1.7 [95% CI: 1.2 to 2.3] p=0.005) however, not for adjustables (RR: 1.2 [95% CI: 0.8 to 1.7] p=0.301). There was a notable difference identified that hit-up forwards recorded more transient injuries than outside backs (RR: 1.4 [95% CI: 1.1 to 1.8] p=0.021). There was a two-fold increased risk of injury by hit-up forwards in the second (RR: 2.1 [95% CI: 1.5 to 3.0]; p<0.001) than the first half of matches whereas outside backs were slightly less (RR: 1.7 [95% CI: 1.2 to 1.5] p=0.007).

## DISCUSSION

To date international studies on amateur rugby league injuries are limited. Although studies undertaken in New Zealand are increasing in number[15,16,17,18] they are typically limited to either a single team or competition over a short duration and only report on the total injuries sustained. The present study however, was designed to investigate the incidence of injury in amateur rugby league participants by positional groups in a team competing at the premier level of a division 1 zonal competition in New Zealand. As there are no published studies reporting on match injuries by positional groups for amateur rugby league between study comparisons is limited. It is envisioned that this study will facilitate further player positional studies to be undertaken at the amateur level of participation.

The overall injury rate of 420 per 1000 match hr reported in this study is lower than that previously reported for division 2 amateur rugby league in New Zealand (700 per 1000 match hr),[16] yet higher than that of amateur (160.6 per 1000 player-position match hr)[2] and semi-professional rugby league in Australia (68 per 1,000 match hr)[5] and New Zealand (115 per 1,000 match hr).[17] This increased incidence may be related to fatigue and or accumulative microtrauma within amateur New Zealand rugby league players.[2] Together with previous studies[5,13,16,24] on rugby league injuries, it is further demonstrated that rugby league match-play is associated with high injury rates when compared with other team sports. Further, the results of this study highlight the importance of injury prevention strategies designed to reduce the incidence of injury in rugby league.

The finding that hit-up forwards recorded more total injuries than outside backs and adjustables is likely reflective of the high offensive and defensive work-rates of this positional group.[5] Interestingly when examined by match quarters, adjustables recorded more injuries in the first and second quarters than hit up forwards before the incidence of injury decreased. This change in the incidence of injuries by positions could be attributed to two possible factors; 1) adjustables may be involved in more ball contact and handling activities than hit-up forwards in the first compared with the second half of matches, and 2) hit-up forwards have been reported to have a lower level of aerobic fitness[23] when compared with both adjustables and outside backs. This lower level of aerobic fitness may become more pronounced as the match duration progresses leading to the onset of fatigue. Additionally, a lower level of aerobic fitness is reported to be a significant predictor of a contact injury in rugby league players.[9] Hit-up forwards have a high work-rate and, when combined with a lower level of aerobic fitness, the physiological strain on this positional group may be higher.[4] The recovery required for this positional group may be reduced between the bouts of high-intensity activities that hit-up forwards undertake and as such the onset of fatigue may be hastened as a result.

Similar to other studies reporting on injuries in amateur rugby league,[2,16,20] musculoskeletal injuries were the most commonly reported injuries within this study. The type of musculoskeletal injuries varied by positional group with hit-up forwards recording more contusions and fractures than both adjustables and outside backs. This may be reflective of hit-up forwards greater involvement in physical collisions (ie, tackles and hit-ups)[1] in combination with their higher body mass[6] than outside backs and adjustables. Furthermore, the majority of injuries were recorded as having occurred in the tackle situation which is similar to other studies reporting on rugby league injuries.[22] Hit-up forwards recorded more injuries as the ball-carrier than the tackler whilst conversely outside backs and adjustables recorded more injuries as the tackler than the ball carrier. Poor tackle technique, tackling when fatigued, lower aerobic fitness and limited training prior to competing in matches have all been reported as contributing to the incidence of tackle related injuries.[8,9] Future research,

**Table 2.** Player number, injury site, injury type, injury cause, injury severity and match period of injury of New Zealand amateur rugby league participants for positional groups by rates per 1,000 match hr with 95% confidence interval and percentage of injuries.

| | Outside Backs | | | | Adjustables | | | | Hit Up Forwards | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Player** | **No of Injuries** | **Rate (95% CI)** | **%** | **Player** | **No of Injuries** | **Rate (95% CI)** | **%** | **Player** | **No of Injuries** | **Rate (95% CI)** | **%** |
| 1 | 26 | 375.9 (256.0 to 552.1) | 24.1 | | | | | | | | |
| 2 | 18 | 260.3 (164.0 to 413.1) | 16.7 | 6 | 35 | 506.1 (363.4 to 704.8) | 28 | 8 | 44 | 636.2 (473.4 to 854.9) | 30.6 |
| 3 | 18 | 260.3 (164.0 to 413.1) | 16.7 | 7 | 21 | 303.6 (198.0 to 465.7) | 16.8 | 10 | 38 | 549.5 (399.8 to 755.1) | 26.4 |
| 4 | 25 | 361.5 (244.3 to 535.0) | 23.1 | 9 | 39 | 563.9 (412.0 to 771.8) | 31.2 | 11 | 39 | 563.9 (412.0 to 771.8) | 27.1 |
| 5 | 21 | 303.6 (198.0 to 465.7) | 19.4 | 13 | 30 | 433.8 (303.3 to 620.4) | 24 | 12 | 23 | 332.6 (221.0 to 500.5) | 16 |
| Total | 108[b] | 312.3 (258.6 to 377.1) | 100 | Total | 125 | 506.1 (363.4 to 704.8) | 100 | Total | 144[a] | 520.5 (442.1 to 612.9) | 100 |
| **INJURY SITE** | | | | | | | | | | | |
| Head/Neck | 23 | 66.5 (44.2 to 100.1) | 21.3 | | 30 | 108.4 (75.8 to 155.1) | 24 | | 28 | 101.2 (69.9 to 146.6) | 19.4 |
| Upper Limb | 37 | 107.0 (77.5 to 147.7) | 34.3 | | 52 | 188.0 (143.2 to 246.7) | 41.6 | | 51 | 184.4 (140.1 to 242.6) | 35.4 |
| Lower Limb | 33 | 95.4 (67.8 to 134.2) | 30.6 | | 27[b] | 97.6 (66.9 to 142.3) | 21.6 | | 44[a] | 159.1 (118.4 to 213.7) | 30.6 |
| Chest/Back | 15 | 43.4 (26.2 to 72.0) | 13.9 | | 16 | 57.8 (35.4 to 94.4) | 12.8 | | 21 | 75.9 (49.5 to 116.4) | 14.6 |
| **INJURY TYPE** | | | | | | | | | | | |
| Contusion | 41[b] | 118.6 (87.3 to 161.0) | 37.6 | | 52 | 188.0 (143.2 to 246.7) | 40.9 | | 62[c] | 224.1 (174.7 to 287.5) | 41.6 |
| Sprain | 15 | 43.4 (26.2 to 72.0) | 13.8 | | 11 | 39.8 (22.0 to 71.8) | 8.7 | | 8 | 28.9 (14.5 to 57.8) | 5.4 |
| Strain | 27 | 78.1 (53.5 to 113.9) | 24.8 | | 32 | 115.7 (81.8 to 163.6) | 25.2 | | 32 | 115.7 (81.8 to 163.6) | 21.5 |
| Fracture | 9 | 26.0 (13.5 to 50.0) | 8.3 | | 8 | 28.9 (14.5 to 57.8) | 6.3 | | 14 | 50.6 (30.0 to 85.4) | 9.4 |
| Concussion | 7 | 20.2 (9.7 to 42.5) | 6.4 | | 2 | 7.2 (1.8 to 28.9) | 1.6 | | 5 | 18.1 (7.5 to 43.4) | 3.4 |
| Dislocation | 2 | 5.8 (1.4 to 23.1) | 1.8 | | 2 | 7.2 (1.8 to 28.9) | 1.6 | | 7 | 25.3 (12.1 to 53.1) | 4.7 |
| Other | 8[ab] | 23.1 (11.6 to 46.3) | 7.3 | | 20[c] | 72.3 (46.6 to 112.1) | 15.7 | | 21[c] | 75.9 (49.5 to 116.4) | 14.1 |
| **INJURY CAUSE** | | | | | | | | | | | |
| Tackler | 36[e] | 104.1 (75.1 to 144.3) | 33.3 | | 51 | 184.4 (140.1 to 242.6) | 40.8 | | 49[e] | 177.1 (133.9 to 234.4) | 34 |
| Ball carrier | 62[d] | 179.3 (139.8 to 230.0) | 57.4 | | 62 | 224.1 (174.7 to 287.5) | 49.6 | | 81[d] | 292.8 (235.5 to 364.0) | 56.3 |
| Collision Other | 1 | 2.9 (0.4 to 20.5) | 0.9 | | 0 | 0.0 - | 0 | | 1 | 3.6 (0.5 to 25.7) | 0.7 |
| Other | 9 | 26.0 (13.5 to 50.0) | 8.3 | | 12 | 43.4 (24.6 to 76.4) | 9.6 | | 13 | 47.0 (27.3 to 80.9) | 9 |
| **INJURY SEVERITY** | | | | | | | | | | | |
| Transient | 80[b] | 231.3 (185.8 to 288.0) | 74.1 | | 98 | 354.3 (290.6 to 431.8) | 78.4 | | 112[c] | 404.9 (336.4 to 487.2) | 77.8 |
| Mild | 13 | 37.6 (21.8 to 64.7) | 12 | | 16 | 57.8 (35.4 to 94.4) | 12.8 | | 15 | 54.2 (32.7 to 89.9) | 10.4 |
| Moderate | 6 | 17.4 (7.8 to 38.6) | 5.6 | | 6 | 21.7 (9.7 to 48.3) | 4.8 | | 5 | 18.1 (7.5 to 43.4) | 3.5 |
| Major | 9 | 26.0 (13.5 to 50.0) | 8.3 | | 5 | 18.1 (7.5 to 43.4) | 4 | | 12 | 43.4 (24.6 to 76.4) | 8.3 |
| **MATCH PERIOD** | | | | | | | | | | | |
| 1st quarter | 6[a] | 17.4 (7.8 to 38.6) | 5.6 | | 19[c] | 68.7 (43.8 to 107.7) | 15.2 | | 12 | 43.4 (24.6 to 76.4) | 8.3 |
| 2nd quarter | 34 | 98.3 (70.3 to 137.6) | 31.5 | | 40 | 144.6 (106.1 to 197.1) | 32 | | 34 | 122.9 (87.8 to 172.0) | 23.6 |
| 3rd quarter | 35 | 101.2 (72.7 to 141.0) | 32.4 | | 33 | 119.3 (84.8 to 167.8) | 26.4 | | 38 | 137.4 (100.0 to 188.8) | 26.4 |
| 4th quarter | 33[b] | 95.4 (67.8 to 134.2) | 30.6 | | 33[b] | 119.3 (84.8 to 167.8) | 26.4 | | 60[ac] | 216.9 (168.4 to 279.3) | 41.7 |

CI: Confidence Interval. Significant difference ($p<0.05$) than: (a) adjustables; (b) Hit-up forwards; (c) outside backs; (d) tackler; (e) ball carrier

investigating the nature of the rugby league tackle, injuries that result from a tackle situation and tackle related involvement prior to an injury occurring is warranted in order to assist in the development of injury prevention strategies. In addition coaching on tackle techniques, game specific tackling, and defensive drills undertaken both prior to and post fatigue may also assist in injury prevention.[5]

The upper limb was recorded as the most common injury region. This is in conflict with some,[2,16,20] but not all,[7,17] studies reporting on rugby league injuries. The head and neck[2] and lower limbs[16] have previously been reported as the most common injury region at the amateur level of rugby league participation. Such trends are also similar for semi-professional[3] and professional[11] levels of participation however, within junior participation the shoulder,[7] ankle and head/face[25] have been reported as the most common injury sites. Despite the overall total incidence of injuries, the injury region varied by player positional group with hit-up forwards recording more lower limb injuries than outside backs and adjustables, whilst both outside backs and adjustables recorded more upper limb injuries than hit-up forwards. The difference in the injury region may be reflective of the positional demands of match-play, whereby outside backs and most of the adjustable positional group defend outside of the hit-up forwards undertaking more running and agility type activities throughout a match. Typically they have a lower body mass than hit-up forwards and this may be reflected in the injury region whereby most of the injuries recorded were to the upper limb region as the tackler as opposed to being the ball carrier. Also typical of the adjustables is their ability or inability to defend against

much larger forwards (hit-up or wide-running) specifically targeting them during match-play. Further research is warranted to explore the positional group differences in the role of tackler and ball carrier.

Within the current study transient injuries were the most common injury severity type across all positional groups, accounting for 74.1%, 78.4% and 77.8% of injuries to outside backs, adjustables and hit-up forwards, respectively. This finding is in line with previous epidemiological research suggesting that non-time loss injuries account for 72-95% of the total injury occurrence.[22] The increased incidence of transient injuries to hit-up forwards (n=112) in comparison to outside backs (n=80) is likely attributable to hit-up forwards increased exposure to both tackle and collision events.[1,2] Whilst these injuries do not result in either lost training or game time, it is important to acknowledge their impact on the financial resources of teams and player participation.[22]

## CONCLUSION

This study has identified that there are positional differences in the rate, site and type of injuries that occur from rugby league match participation. Hit-up forwards recorded more injuries as the ball-carrier than the tackler, whilst outside backs and adjustables recorded more injuries as the tackler than the ball carrier. The study further identified that rugby league match-play is associated with high injury rates and that further injury prevention strategies designed to reduce the incidence of injury in rugby league are required.

## CONFLICT OF INTEREST

No sources of funding were used to conduct this study. The authors have no conflict of interests that are directly relevant to the content of this study.

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## REFERENCES

1 Cummins C & Orr R. Analysis of physical collisions in elite national rugby league match play. *Int J Sports Physiol Perform* 2015; **10**(6):732-739.

2 Gabbett T. Incidence, site, and nature of injuries in amateur rugby league over three consecutive seasons. *Br J Sports Med* 2000; **34**(2):98-103.

3 Gabbett, T. Incidence of injury in semi professional rugby league players. *Br J Sports Med* 2003; **37**(1):36-44.

4 Gabbett T. Influence of training and match intensity on injuries in rugby league. *J Sports Sci* 2004; **22**(5):409-417.

5 Gabbett T. Influence of playing position on the site, nature and cause of rugby league injuries. *J Strength Cond Res* 2005; **19**(4):749-755.

6 Gabbett T. Science of rugby league football: A review. *J Sports Sci* 2005; **23**(9):961-976.

7 Gabbett T. Incidence of injury in junior rugby league players over four competitive seasons. *J Sci Med Sport*. 2008; **11**(3):323-328.

8 Gabbett T. Influence of fatigue on tackling technique in rugby league players. *J Strength Cond Res* 2008; **22**(2):625-632.

9 Gabbett T, & Domrow N. Risk factors for injury in subelite rugby league players. *Am J Sports Med* 2005; **33**(3):428-434.

10 Gabbett T, King T, & Jenkins D. Applied physiology of rugby league. *Sports Medicine* 2008; **38**(2):119-138.

11. Gissane, C., Jennings, D., Cumine, A., Stephenson, S., & White, J. Differences in the incidence of injury between rugby league forwards and backs. Aust J Sci Med Sport. 1997; 29(4):91-94.

12 Gissane C, White J, Kerr K, & Jennings D. Physical collisions in professional Super League rugby, the demands on different player positions. *Cleve Med J* 2001; **4**:137-146.

13 Hawkins R, & Fuller C. A prospective epidemiological study of injuries in four English professional football clubs. *Br J Sports Med* 1999; **33**(3):196-203.

14 Hodgson Phillips L, Standen P, & Batt M. Effects of seasonal change in rugby league on the incidence of injury. *Br J Sports Med* 1998; **32**(2):144-148.

15 King D, & Gabbett T. Injuries in a national women's rugby league tournament: An initial investigation. *NZ J Sports Med* 2007; **34**(2):18-22.

16 King D, & Gabbett T. Amateur rugby league match injuries in New Zealand. *NZ J Sports Med* 2009; **36**(1):16-21.

17 King D, & Gabbett T. Injuries in the New Zealand semi-professional rugby league competition. *NZ J Sports Med* 2009; **36**(1):6-15.

18 King D, Gabbett T, Dreyer C, & Gerrard D. Incidence of injuries in the New Zealand national rugby league sevens tournament. *J Sci Med Sport* 2006; **9**(1-2):110-118.

19 King D, Gabbett T, Gissane C, & Hodgson L. Epidemiological studies of injuries in rugby league: Suggestions for definitions, data collection and reporting methods. *J Sci Med Sport* 2009; **12**(1):12-19.

20 King D, & Gissane C. Injuries in amateur rugby league matches in New Zealand: A comparison between a division one and a division two premier grade teams. *Clin J Sports Med* 2009; **19**(4):277-281.

21 King D, Gissane C, Clark T, & Marshall S W. The incidence of match and training injuries in rugby league: a pooled data analysis of published studies. *Int J Sports Sci Coach* 2014; **9**(2):417-432.

22 King D, Hume P, Milburn P, & Guttenbeil D. Match and training injuries in rugby league: A review of published studies. *Sports Med* 2010; **40**(2):163-178.

23 Meir R, Newton R, Curtis E, Fardell M, & Butler B. Physical fitness qualities of professional rugby league football players: determination of positional differences. *J Strength Cond Res* 2001; **15**(4):450-458.

24 Orchard J, & Seward H. Epidemiology of injuries in the Australian football league, seasons 1997 - 2000. *Br J Sports Med* 2002; **36**(1):39-45.

25 Orr R, & Cheng H L. Incidence and characteristics of injuries in elite Australian junior rugby league players. *J Sci Med Sport*. 2016; **19**(3):212-217.

# Foot to shank ratio: Does it influence ankle dorsiflexion range of motion in the knee-to-wall assessment technique?

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## ABSTRACT

### Aim

To establish the correlation between scores of ankle dorsiflexion range of motion (DROM) when assessed using a knee-to-wall assessment score and standard extendable joint goniometer.

### Study Design

Foot to shank ratio influence was assessed on two separate occasions by the same rater using two measures of ankle DROM ie, i) the knee-to-wall assessment linear score, and ii) the joint angle recorded with a standard extendable joint goniometer.

### Participants

Nine females and six males (combined mean age = 26.5 ± 6.2 yr; range = 19-37 years, height = 1.73 ± 0.85 m, mass = 76.5 ± 18.6 kg) volunteered to participate in this study.

### Methods

Foot and shank length was determined prior to assessment of ankle DROM. Following a standardised warm-up, ankle DROM was assessed using two methods: i) knee-to-wall method; ii) and standard extendable joint goniometer. Measures were taken twice with each technique and on two separate occasions. Measurement of ankle DROM alternated between each ankle (ie, one measurement taken on the right ankle followed by one measurement on the left ankle and then repeated), with the first measure on both test occasions being the knee-to-wall assessment.

### Results

A significant positive correlation was found between foot length and shank length for both right ($p < 0.001$, $r = .877$) and left ($p < 0.001$, $r = .880$) side. No other significant correlations were found between all other variables ($p > 0.05$).

### Conclusion

Findings indicate that neither the length of the foot nor the length of the shank influences ankle joint mobility. Further, their relative lengths, as determined by a foot-to-shank length ratio, had no impact on ankle DROM when measured using the knee-to-wall assessment method. As a result, either assessment method can be used with confidence by clinicians and sports trainers to determine ankle DROM.



### Keywords

ankle DROM; knee-to-wall assessment; anthropometric measures of lower limb



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## INTRODUCTION

Passive and active methodologies for assessing basic movement quality within major joints of the body can provide useful information related to performance and injury predisposition in athletic populations.[7,9,20] One such joint in the lower body that is of particular interest to clinicians (eg, physiotherapists, sports medicine practitioners, and strength and conditioning coaches) is the ankle. Its range of motion is usually assessed by measurements that rely on the relative positions of the foot and lower leg (shank).[1,22] The ankle is often considered to be a basic hinge joint with its primary movement occurring in the sagittal plane as dorsiflexion and plantar flexion.[16]

With this plane providing the largest range of movement, it is essential for function in both highly dynamic movements, but also in activities of daily living.[13,21,22] However, it must be noted that ankle rotations are triplanar, across multiple joints and include movements such as inversion and adduction (supination) and eversion and abduction (pronation). Therefore, sagittal dorsiflexion range of motion for the foot relative to the shank, actually includes a complex multi-joint combination of rotations.[16] As a result, although the joint is often assumed to behave as a pure hinge in the sagittal plane, it can also make rolling and gliding movements. These are possible because of multiple oblique rotational axes that allow for the foot to adapt and change its position.[3]

Restricted ankle dorsiflexion range of motion (DROM) can also be a contributing factor in a range of ankle and knee injuries, eg, Achilles tendinitis and tendinopathy, calf muscle tightness leading to muscle strains, anterior cruciate ligament injury, and plantar fasciitis.[7] Further, having a restricted functional ROM at the ankle is also considered one of the main factors contributing to ankle injuries during highly dynamic activities.[11,17] Therefore, having adequate ankle mobility is considered important for functional movements such as squatting,[15] balance tasks and movements of daily living,[12] as well as for dynamic sporting movements such as high velocity landings.[2,22]

Being able to assess functional weight-bearing ankle DROM does not require lengthy clinical training. While a standard extendable joint goniometer is typically used to assess the DROM in the ankle, other methods (eg, digital inclinometer, knee-to-wall load

bearing lunge) are also commonly employed. These have been found to have good levels of reliability and low measurement error between raters of varying experience (novice to experienced).[1] Of these methods the knee-to-wall weight-bearing lunge has been commonly used with athletic populations as a method for assessing injury predisposition, eg,[5,6,10] However, what is not clear is whether this method of assessing ankle DROM may be confounded by the relative lengths of an individual's foot and shank.

One might expect that length of the foot relative to the shank could have either a negative or positive influence on the score produced during assessment using the knee-to-wall method. This might be the case where the foot is longer relative to a shorter shank length or vice versa, possibly producing an under or overestimation of the ankle's DROM. For example, an overestimation of the ankle's DROM may be produced where the foot is shorter relative to a longer shank length. Such anatomical impacts may result in the clinician or sports medicine practitioner incorrectly diagnosing the ankle as having a functionally unacceptable or acceptable ROM.
Given the above, it was the primary aim of this study to establish whether foot to shank length ratio has an influence on the knee-to-wall assessment score. This study also aimed to establish the extent of any correlation between the knee-to-wall assessment score and DROM measurement using a standard extendable joint goniometer.

## METHODS

### Experimental Approach to the Problem

Ankle DROM was assessed using two different techniques, these were: i) an extendable joint goniometer, and ii) the knee-to-wall weight-bearing lunge. Participants attended two testing sessions and had measurements recorded at the same time of day (across participants this varied from 10:00 am to 3:00 pm based on availability) for each session, with a minimum of 48 and a maximum of 96 hours between sessions. The minimum and maximum time between testing sessions were dictated by participant availability. Testing was conducted on two separate occasions to determine the reproducibility (reliability/agreement) of observed scores.

Upon arrival at the laboratory participants had their foot length (tip of the akropodion, ie, tip of longest toes, to the pternion, ie, most posterior point on the calcaneus) and shank length (tibiale mediale-sphyrion tibiale score)[19] determined prior to ankle DROM being assessed.

Scores were recorded for ankle DROM following a standardised warm up procedure. The same rater (ISAK level 3 accredited anthropometrist) performed all measurement techniques. The benchmark was deemed to be the angular range of motion measured by a standard extendable Lafayette 01135 joint goniometer (Lafayette Instrument Co. Inc., Nashville, IN), against which the foot-to-wall distance produced by the knee-to-wall test were correlated (see below for details of each assessment method). Each participant initially completed one repetition of the weight-bearing lunge on one ankle using a trial-and-error approach to determine the point of maximum ankle dorsiflexion. Once established the participant then repeated the procedure with the opposite ankle. This process was then repeated one more time on each ankle, ie, the knee-to-wall method was repeated a total of two times on each ankle. Once this method of assessment was completed the same approach was adopted with the extendable joint goniometer.

### Participants

Nine female and six male participants (mean age = 26.5 ± 6.2 years, height = 1.73 ± 0.85 m, mass = 76.5 ± 18.6 kg) volunteered for this study. All participants were actively engaged in regular weekly physical exercise at the time of their participation in the study. Participants were informed of the benefits and risks of the investigation prior to signing an institutionally approved informed consent document to participate (approval number: ECN-15-053). Participants were advised that they could withdraw their consent at any time

### Procedures

All testing sessions were conducted at the same time of day (between 1.00 and 4.00 pm) and in the same location. Participants were recruited from within a sample of convenience (population of enrolled sport and exercise science students at the university). Email invitations were distributed to all enrolled (220+) students with those expressing interest in participating being provided with further

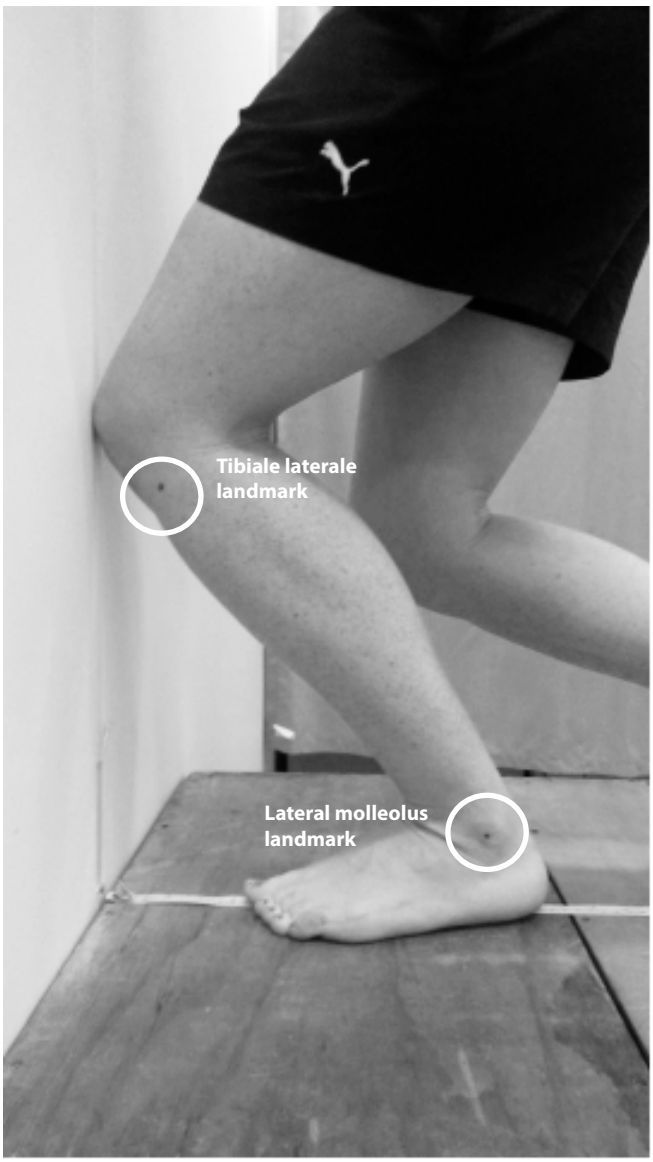


**Figure 1.** Position on platform when performing weight-bearing lunch for knee-to-wall assessment - goniometer landmarks identified.

information, ie, an information sheet detailing the purpose of the study and the nature of their involvement and any potential risks. Upon agreeing to participate participants were instructed not to complete any lower body training in the 24 hours immediately before their scheduled testing sessions. At the first testing session, a brief demonstration of all procedures was provided and participants were asked to complete informed consent and a standard pre-participation health screening questionnaire. Participants were only assessed if free from lower body injury and illness.

### 1 Knee-to-wall Method

Following participant preparation, each testing session commenced with the knee-to-wall assessment to determine the foot-to-wall distance.[1,8] Participants wore socks or bare feet as preferred and placed the middle of the big toe and heel of the measured leg on a string line, which was placed on top of a non-extensible measuring tape attached to the surface of an elevated platform placed securely against a wall. Participants were instructed to place their hands on the wall for support and to use the non-measured leg for balance as they performed a weight-bearing lunge movement. This required them to push the knee toward a vertical string line affixed to the wall, which was aligned with the same line on the elevated platform surface (see Figure 1).

Participants were encouraged to dorsiflex their ankle maximally while also flexing the knee in weight-bearing. The heel had to remain firmly placed on the surface of the platform at all times – determined visually by the rater and research assistant. Participants pushed the middle of the patella toward the vertical string line and if the participant either found it easy to make contact with the surface of the patella on the wall, or it was too far away for contact to be made, small incremental adjustments to the foot position were made until the patella made light contact with the wall; indicating that maximum end range ankle DROM had been reached. Once this end range was achieved the rater used the measuring tape to measure the distance from the wall to the tip of the big toe. The measure to the nearest millimetre was recorded. This process was then repeated on the opposite leg and then again for both ankles.

**Table 1.** Mean (±SD) scores for all basic measures (N=15).

| Item | Mean Right Side | Mean Left Side |
|---|---|---|
| Shank length* (cm) | 38.20 ± 2.80 | 38.35 ± 2.91 |
| Foot length** (cm) | 25.61 ± 1.99 | 25.61 ± 2.16 |
| Foot to shank ratio | 0.67 ± 0.02 | 0.67 ± 0.03 |
| Knee-to-wall measure (cm) | 14.9 ± 3.9 | 15.0 ± 3.7 |
| Goniometer (o) | 42.7 ± 9.2 | 44.9 ± 7.8 |

*Tibiale mediale-sphyrion tibiale length
**Tip of the akropdion to the pternion

### 2 Goniometer

In order for ankle DROM assessment to be conducted participants had to have landmarks identified on the lower leg and ankle. These were identified (see Figure 1) by the rater with a black felt tipped pen at the site of the tibiale laterale landmark (the most superior point on the lateral border of the head of the tibia)[19] and the lateral malleolus.[22] These landmarks were used to locate the extendable goniometer in the appropriate position ready for assessment of the ankle DROM angle. When ready, participants were asked to stand approximately 1200 mm away from the wall and to place their hands on the wall, while also using the non-measured leg for balance, as they performed the weight-bearing lunge movement. In setting up for each assessment procedure participants were required to ensure that the centre of the big toe and the middle of the heel on the assessed limb were once again aligned with the measuring tape and string line. They were then instructed to push the middle of the patella toward the vertical line attached to the wall (without making contact), while keeping the heel firmly on the surface of the elevated platform.

Once the participant reached their self-perceived end range of DROM they were asked to hold that position while the rater placed the goniometer on the landmarks to measure the angle of the tibia relative to the platform. After each goniometer reading the mobile arm was moved to ensure that the new reading had to be reacquired by re-establishing the location of the tibiale laterale landmark.

### Statistical Analyses

The best score produced over the two sessions (4 trials per ankle per assessment method in total) was used in the statistical analysis. The mean group score for the items/variables measured is presented in Table 1. All variables of interest were analysed for normality through Shipro-Wilks tests and interpretation of box plots and normal Q-Q plots, before Pearson Correlations (with 95% confidence intervals) were conducted to determine the relationship between variables. Statistical significance was set at $p < 0.05$ and all statistical analyses were conducted using SPSS version 22.

## RESULTS

All variables were found to be normally distributed ($p > 0.05$). A significantly strong positive correlation was found between foot length and shank length for both right ($p < 0.001$, $r = .877$ [.966, .765]) and left ($p < 0.001$, $r = .880$ [.980, .699]). Significant strong correlations were also found between the knee-to-wall and goniometer measures for both right ($p < 0.001$, $r = -.901$ [-.958, -.764]) and left ($p < 0.001$, $r = -.924$ [-.967, -.852]) legs. No other significant correlations were found between ankle mobility measures (knee-to-wall and goniometer) and anatomical measures (shank length, foot length and shank-to-foot ratio) ($p > 0.05$) (see Table 2).

## DISCUSSION

Findings suggest that neither the length of the foot nor length of the shank have an influence on the assessment of ankle DROM in the sample analysed. More importantly, shank to foot length ratio did not impact on ankle DROM when measured using the knee-to-

**Table 2.** Pearson's Correlation [95% confidence intervals] between ankle mobility measures and anatomical measures for each leg (N = 15).

| | Right Shank Length (cm) | Right Foot Length (cm) | Right Shank-to-Foot Ratio |
|---|---|---|---|
| Right Knee-to-Wall (cm) | - .110 [-.424, .174] | - .155 [-.515, .248] | .112 [-.422, .561] |
| Right Goniometer (o) | .230 [-.301, .649] | .153 [-.401, .580] | .133 [-.358, .654] |
| | Left Shank Length (cm) | Left Foot Length (cm) | Left Shank-to-Foot Ratio |
| Left Knee-to-wall (cm) | -.094 [-.391, .502] | .131 [-.287, .630] | -.107 [-.540, .283] |
| Left Goniometer (o) | .131 [-.353, .661] | .063 [-.442, .513] | .119 [-.293, .563] |

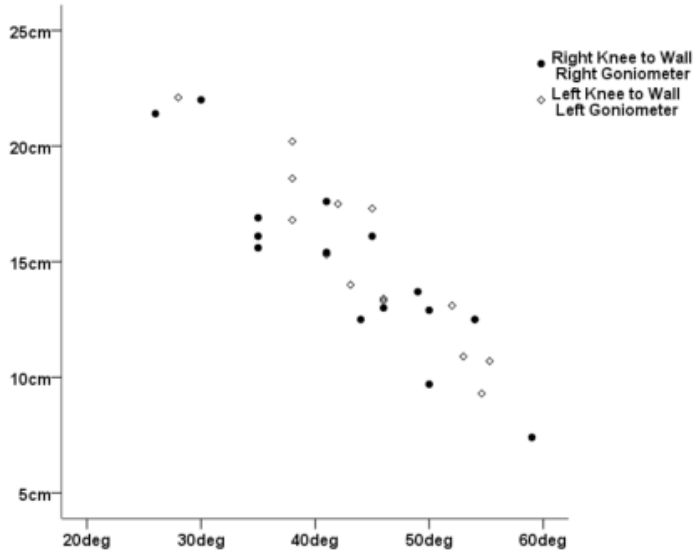


**Figure 2.** Scatterplot showing the relationship between the knee-to-wall and goniometer measures for both the right and left legs.

wall assessment method. This might be considered surprising, as intuitively, it would be reasonable to speculate that a shorter foot length relative to a longer shank length, for instance, might substantially affect a linear score of ankle DROM that implicitly relies on these parameters. However, this was not the case.

A number of methods can be used to assess ankle plantarflexion and dorsiflexion either passively or actively.[13] The range of ankle DROM that can be achieved is influenced by factors such as whether the knee is in extension or flexion, and whether the movement is produced while load bearing or non-load bearing. For the sports medicine practitioner, however, having a simple method of assessing ankle DROM is essential for expediently and accurately determining optimal functional range of motion for athletic populations.

Scientists have long known of the key role of ankle ROM in absorbing force when performing a landing from typical jumping heights[4] and specifically during exercise techniques such as plyometrics, and other load bearing movements, eg, squatting.[15] Having a simple measure of ankle DROM may assist in the identification of those athletes that may be predisposed to injuries resulting from a less than optimal functional range of movement at this joint. This may become more important with older athletes, since joint motion is generally more restricted in older age groups.[18] However, ankle DROM values reported in the literature vary widely,[21] with the score produced also varying with the method of assessment.[14]

The evidence reported here indicates that the knee-to-wall ankle DROM assessment technique can be used by sports medicine practitioners with confidence. This is an easy to use method that does not require high levels of clinical training to use in athletic populations. Further, this method requires very little in the way of equipment making it easy to utilise in most sports training environments. Importantly, as shown in this research, this method does not appear to be influenced by anthropometric parameters, such as the ratio of shank length to foot length. The strong correlation found between foot and shank length in this current study suggests that this measure is reasonably consistent across the population studied, thus has no influence on the knee-to-wall assessment method. Notwithstanding this finding, it must be acknowledged that participants in this research study represent a seemingly homogenous, although randomly selected, sample of convenience. There was no evidence of any outliers within this group of participants and it may still be reasonable to expect that substantial anatomical variations in relative anatomical segmental lengths may potentially influence assessments.

What this study adds:

- This study has shown that a weight bearing knee-to-wall lunge (linear output measure) is a reliable test of ankle DROM, and can be used as a reliable alternative to the use of an extendable joint goniometer (angular output measure) in an athletic population.
- Importantly, the knee-to-wall ankle DROM assessment technique does not appear to be influenced by anatomical factors, such as the ratio of shank length to foot length.
- The knee-to-wall method is an easy to use assessment technique that can provide important information about ankle joint DROM, which may be a factor in a range of lower body functional movements.


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## REFERENCES


1 Bennell K, Talbot R, Wajswelner H, Techovanich W, Kelly D. Intra-rater and inter-rater reliability of a weight-bearing lunge measure of ankle dorsiflexion. *Aust J Physio* 1998; **44**(3):175-180.

2 Bohannon RW, Tiberio D, Waters G. Motion measured from forefoot and hindfoot landmarks during passive ankle dorsiflexion range of motion. *J Orthop Sports Phys Ther* 1991; **13**(1):20-22.

3 Bosch F, Klomp R. Running: Biomechanics and exercise physiology applied in practice. Philadelphia, PA; Elsevier, 2005, pp. 8-11.

4 Devita P, Skelly WA. Effect of landing stiffness on joint kinetics and energetics in the lower extremity. *Med Sci Sports Exerc* 1992; **24**(1):108-15.

5 Gabbe BJ, Finch CF, Bennell KL, Wajswelner, H. Risk factors for hamstring injuries in community level Australian Football. Brit J Sports Med 2005; 39(2):106-110.

6 Gabbe BJ, Finch CF, Wajswelner H, Bennell, KL Predictors of lower extremity injuries at the

community level of *Australian Football. Clin J Sports Med* 2004; **14**(2):56-63.

7 Jeon IC, Kwon OY, Yi CH, Cynn HS, Hwang UJ. Ankle-Dorsiflexion range of motion after ankle self-stretching using a strap. *J Athl Train* 2015; **50**(12):1226-1232.

8 Konor MM, Morton S, Eckerson JM, Grindstaff TL. Reliability of three measures of ankle dorsiflexion range of motion. *Int J Sports Phys Ther* 2012; **7**(3):279-287.

9 Kritz M, Cronin J, Hume, P. The bodyweight squat: A movement screen for the squat pattern. *Strength Cond J* 2009; **31**(1):76-85.

10 Lundgren LE, Tran TT, Nimphius S, Raymond E, Secomb JL, Farley RO, Newton RU, Steele JR, Sheppard JM. Development and evaluation of a simple multifactorial model based on landing performance to indicate injury risk in surfing athletes. *Int J Sports Physio Perf* 2015; **10**(8):1029-1035.

11 Lundgren L, Tran T, Farley O, Secomb J, Nimphius S, Newton R, Sheppard J. Ankle range of motion among surfing athletes. *Aust Strength Cond J* 2013; **21**(S2):121-124.

12 Menz HB, Morris ME, Lord SR. Foot and ankle characteristics associated with impaired balance and functional ability in older people. *J Gerontol A Biol Sci Med Sci* 2005; **60**(12):1546-1552.

13 Moseley AM, Crosbie J, Adams R. High and low ankle flexibility and motor task performance. *Gait Posture* 2003; **18**(2):73-80.

14 Moseley AM, Crosbie J, Adams R. Normative data for passive ankle plantarflxion-dorsiflexion flexibility. *Clinical Biomechanics* 2001; **16**(6):514-521.

15 Myer GD, Kushner AM, Brent JL, Schoenfeld BJ, Hugentobler J, Lloyd RS, Vermeil A, Chu DA, Harbin J, McGill SM. The Back Squat: A proposed assessment of functional deficits and technical factors that limit performance. *Strength Cond J* 2014; **36**(6):4-27.

16 Oatis CA, Kinesiology: The mechanics and pathomechanics of human movement, 2nd edn. Philadelphia, PA; Lippincott, Williams & Wilkins, 2009, pp. 807-837.

17 Pope R, Herbert R, Kirwan J. Effects of ankle dorsiflexion range and pre-exercise calf muscle stretching on injury risk in army recruits. *Aust J Physio* 1998; **44**(3): 165-177.

18 Roaas A, Anderson GBJ. Normal range of motion of the hip, knee and ankle joints in male subjects, 30-40 years of age. *Acta Orthop Scand* 1982; **53**(2):205-208

19 Stewart A, Marfell-Jones M, Olds T, De Ridder HJ. International Standards for Anthropometric Assessment. Underdale, SA: International Society for the Advancement of Kinanthropometry, 2011, pp. 91-102.

20. Thomas V, Reilly T. Fitness assessment of English League soccer players through the competitive season. *Brit J Sports Med* 1979; **13**(3):103-109.

21. Weir J, Chokalingam N. Ankle joint dorsiflexion: assessment of true values. *Int J Ther Rehabil* 2007; **14**(2):76–82.

22. Whitting JW, Steele JR, McGhee DE, Munro BJ. Dorsiflexion capacity affects achilles tendon loading during drop landings. *Med Sci Sports Exerc* 2011; **43**(4): 706-713.

# Recreational technical diving and its effects on respiration and working capacity

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## ABSTRACT

### Aim

Oxidative stress is increased in recreational technical diving. In an explorative retrograde study we investigated the hypothesis that, (1) repetitive, multi-day dives with oxygen-enriched breathing gases will have an impact on physiological measures, (2) negative effects can be assessed within one week post-dive.

### Study Design

Exploratory retrospective crossover study.

### Setting

Open-water study.

### Participants

Ten healthy experienced technical divers.

### Interventions

Repetitive dives with oxygen-enriched breathing gases during a 5-days diving trip to the Adriatic Sea (Croatia).

### Outcome Measures

Basic measures of medical examination, electrocardiography, echocardiography, body plethysmography, and spiroergometry.

### Results

The divers (32±8 years; mean±SD) performed 11±4 dives. Static and dynamic pulmonary measures were slightly decreased after the trip. On the bicycle ergometer, arterial oxygen partial pressure ($pO_2$) remained almost unchanged from rest to 300 W before the trip (87.5±7.9 vs 85.4±9.7 mmHg) it decreased after the trip (90.4 ±7.2 vs 81.8±8.2 mmHg; $p<0.05$). Peak oxygen uptake (at 300 W) was decreased after the trip (45.1±9.9 vs 41.7±9.8 ml/min; $p<0.05$). In parallel, the aerobic threshold was decreased (234±43 vs 186±39 W; $p<0.05$).

### Conclusions

Pulmonary injury is not detected at rest but at substantial physical load on the bicycle. Even after maximum one week, $pO_2$ at 300 W was decreased together with the pulmonary $O_2$ uptake, thus decreasing the aerobic threshold.

In conclusion: (1) Multi-day, repetitive tech dives seem to induce – at least transient – pulmonary injury. (2) Pauses during a diving trip or more conservative diving are recommended. (3) A major physiological study on open-water divers can be performed by one single medical practice with an adequately equipped laboratory.



### Key words

oxidative stress; technical diving; trimix; pulmonary injury



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## BACKGROUND

"Any dive on which the parameters preclude the possibility of a safe and direct ascent to the surface should be considered technical diving of some sort, and must require specialised training and associated advanced certification". This text is one of quite a few definitions of technical diving. Tech divers practice deep and/or long dives that require decompression stops, ie, direct safe ascent is not possible. To shorten these stops, either oxygen-enriched air or pure oxygen is used as the breathing gas for easier unloading of nitrogen during surfacing. Oxidative stress increases with increase in oxygen partial pressure ($pO_2$) during the dive and during surfacing,[11,27,30] as does the formation of reactive oxygen species (ROS),[5,42] which are extremely toxic and ultimately can cause cell injury and death.[44]

Lorrain Smith's early descriptions of oxygen-induced injury to the lungs[36] were later convincingly confirmed by others.[32,40] As knowledge improved further, NO-based mechanisms of pulmonary oxygen toxicity were suggested. In addition, lung injury would be associated with oxidative or nitrosative stress.[37] Moreover, hyperoxic injury in the rat lung was found to be characterised by pulmonary inflammation,[9] haemorrhage and eventually death of pulmonary capillary endothelial cells and alveolar epithelial cells.[31] The latter injury would impair the gas exchange and induce pulmonary edema.[5]

Although possible adverse effects of an increased proportion of $O_2$ in breathing gases are still not firmly settled, use of $O_2$-enriched air (=nitrox) has become popular for recreational divers, technical divers and dive guides.[11]

Increased $O_2$ necessarily decreases the

proportion of nitrogen ($N_2$) in the breathing gas, which helps both to reduce the formation of unwanted $N_2$ bubbles[3,41] and to preserve cognitive competence.[4,15]

Up to now, the overwhelming majority of pertinent studies on this crucial issue have been performed by investigators from universities, military facilities or dive-related associations. It was one aim of this exploratory physiological study on recreational tech divers to shed some light on the effects of breathing gases with an enriched oxygen proportion (nitrox / trimix) on pulmonary measures after repetitive dives in open sea water. Thus, on the basis of two voluminous data sets we tested the hypothesis that $O_2$-enriched breathing gases cause pulmonary dysfunction which can be documented even up to one week after the end of a 5-day dive excursion.

## PARTICIPANTS AND METHODS

Among other divers who regularly consulted the diving physician (KD) in order to obtain the fit-to-dive certificate were 10 technical divers. This group is very interested not only in technical characteristics of their diving but also in possible negative health outcomes of their demanding dives. Because these 10 divers had planned a 5-day diving trip, they wanted to be examined very carefully.

Physiological measures, in particular spirometric and spiroergometric measures were assessed within one week prior to their recreational diving in the Adriatic Sea (Croatia). After they returned from their trip, the divers wanted to learn whether the increased oxidative stress during repetitive diving had any adverse effects on their health status. Thus, the same measurements were repeated within one week of return. Because there was only one drop-out, ten pairs of full data were available that were made anonymous before analysis. Note that the diving physician did not participate in the diving trip.

As many valuable data existed, KD and the senior author (JDS) decided that the data was well suited for publication as an explorative retrograde study.

### Anthropometric data, smoking habits, medication and diving history

The following data were assessed: age, height, body mass, smoking habits, and medication. In addition, diving history was evaluated, and the divers were asked, whether they had previously suffered from nitrogen narcosis or oxygen toxicity or symptoms of them.

### Medical Practice

Within the week before and the week after the diving trip a comprehensive medical examination was completed: an electrocardiogram (Cardiovit CS-200; Schiller; Baar; CH) was recorded and M-mode, B-mode, Colour-Doppler, PW-Doppler / CW- Doppler echocardiography (phased array sector probe; 1.5–3.3 MHz; Technos MP; Esaote; Genoa; IT) was used to assess cardiac properties.

On both occasions the divers were examined using body plethysmography and spirometry. The following measures were assessed: total lung capacity, vital capacity, forced expiration in 1 s and CO transfer capacity (nSpire Health / ZAN 600; Ferraris Meßgeräte GmbH; Oberthulba; DE). Vigorous verbal encouragement was provided during the measurements.

Spiroergometry was performed using a recumbent bicycle ergometer (ER900; ergoline; Bitz; DE). The load on the bicycle was increased stepwise from rest to maximum subjective power. A reminder. As maximum power during stress ECG 3 W/kg are recommended for males of that age.[16, 26] The average age of 32 years and the average body mass of 90 kg gave a maximum power of about 270 W. Because of their good physical condition, a power of ≥300 W could be expected. Variables were analysed at rest and at 300 W. At 300 W, variables were considered to represent peak values. The participants were instructed to maintain a cadence of 60–70 $min^{-1}$. During this stress test breath-to-breath analysis of $O_2$ inspiration and $CO_2$ expiration was documented to determine the aerobic threshold. The aerobic threshold was reached after the respiratory coefficient equalled '1'.

Blood samples were taken from the ear lobe before, during and after the stress test and were analysed for hemotocrit, haemoglobin, lactate, $pO_2$, $pCO_2$ and pH (Bayer Rapid Lab 248; Bayer; Leverkusen; DE). Blood was immediately analysed after sampling in the laboratory of the first Author (KD).

### Diving

The open-water dives were performed starting from shore sites of the Isle of Vis (Adriatic Sea; Croatia) depending on the plans of the individual group. Air, nitrox (Nx32 or Nx 36), trimix (Tx19±2/37±19) and oxygen were employed as breathing gases depending on the intended dives. It is mentioned that decompression with 75% $O_2$ was performed at 9 m and 6 m for on average 5±3 and 22±12 min, respectively.

Oxygen toxicity units (OTU) for the intended dives were determined by the divers according to validated tables.[46]

### Statistics

IBM SPSS Statistics (version 21 for Windows; New York; US) was employed for statistical analysis. Because data were not normally distributed, the nonparametric Wilcoxon signed rank test was used for pair wise comparing values before and after the trip. Mean values with their standard deviation are presented for illustration.

A $p \leq 0.05$ was regarded to represent significant differences.

## RESULTS

The 10 participants (1 female) were 32±8 ys (mean±SD; range: 21–41 years), 1.82±0.10 m tall and weighed 92.0±23.4 kg, with a body mass index (BMI) of 27±5 kg/$m^2$. Seven of the divers were mild smokers (<5 cigarettes/d), 3 took Ca2+-antagonists, and 2 β-blockers, but their medication did not prevent their diving.

### Diving

The water temperature was 27–28 °C, and the ambient temperature approximately 30 °C. Visibility was always 10–20 m. All dive groups consisted of three participants that knew each other well from home. The divers had performed 410±423 dives prior to this study, and did additional 11±4 dives during the trip using different gas mixes. The bottom time lasted on average 24±12 min at an average depth of 51±17 m. At that depths the water temperature was 17-19 °C.

Total oxygen toxicity units (OTUs) averaged 725±311. The maximum value found in one diver was 1,150 OTU.

### Blood

Erythrocyte (5.1±0.2 vs 5.1±0.3•106/µl) and leucocyte numbers (7.3±2.1 vs 7.4±1.5•$10^3$/µl) were almost identical before and after the trip, as were haematocrit (45±2 vs 44±3%) and haemoglobin (15.5±0.8 vs 15.4±1.1 g/100 ml).

**Table 1** Body plethysmography; Variables of 10 experienced divers before and after the dive trip. Data are means±SD.

| | pre dive | post dive | p |
|---|---|---|---|
| total lung capacity (TLC) [l] | 7.1±1.3 | 7.0±1.3 | 0.508 |
| vital capacity [l] | 5.6±0.7 | 5.4±0.8 | 0.169 |
| residual volume (RV) [l] | 1.5±0.7 | 1.6±0.5 | 0.824 |
| RV / TLC [%] | 21.1±5.7 | 22.9±9.5 | 0.575 |
| FEV1 [%] | 83.0±4.7 | 81.7±6.1 | 0.136 |
| peak expiratory flow [l/s] | 9.1±2.3 | 8.8±1.5 | 0.403 |
| MEF25 – 75 [l/s] | 4.8±0.7 | 4.7±0.9 | 0.398 |
| Rawtot [kPa/l•s] | 0.09±0.05 | 0.09±0.07 | 0.753 |
| TLCO [mmol/kPa/min] | 10.7±1.4 | 10.5±1.3 | 0.575 |

MEF25-75=MMEF: maximum midexpiratory flow, Rawtot: total airway resistance

TLCO: carbon monoxide transfer factor

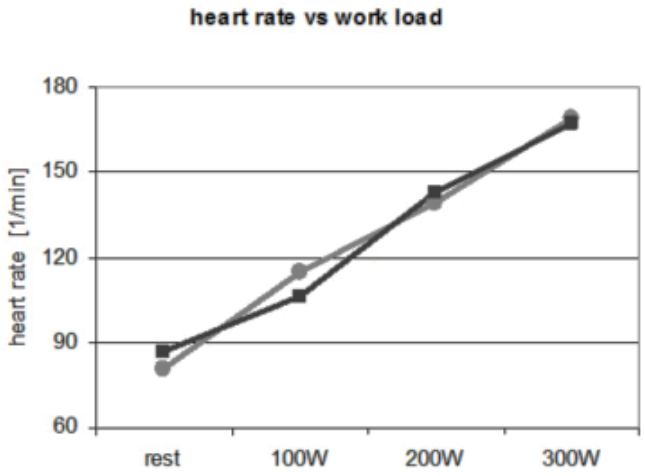


**Figure 1** Heart rate during rest and with increasing workload. The almost linear increasing heart rates after dives with air (gray line) and after dives oxygen-enriched gases (black) were almost identical.

**Table 2** Heart rate and blood pressure for 10 experienced divers at rest and at 300 W on a bicycle each before and after the dive trip.

| | Rest | | | 300 W | | |
|---|---|---|---|---|---|---|
| | pre dive | post dive | p | pre dive | post dive | p |
| heart rate [/min] | 81±14 | 87±15 | 0.443 | 176±15 | 178±11 | 0.507 |
| syst. pressure [mmHg] | 125±12 | 126±13 | 0.646 | 211±18 | 214±30 | 0.395 |
| diast. pressure [mmHg] | 84±9 | 84±9 | 1.000 | 82±11 | 82±13 | 0.721 |

**Table 3** Blood gases and pH at rest and at 300 W each before and after the dive trip.

| | Rest | | | 300 W | | |
|---|---|---|---|---|---|---|
| | pre dive | post dive | p | pre dive | post dive | p |
| pO2 [mmHg] | 87.5±7.9 | 90.4±7.2 | 0.203 | 85.4±9.7 | 81.8±8.2* | 0.169 |
| pCO2 [mmHg] | 40.7±4.8 | 37.0±3.2 | <0.05 | 35.9±6.2° | 35.4±5.4 | 0.575 |
| pH | 7.45±0.04 | 7.45±0.03 | 0.959 | 7.32±0.04° | 7.35±0.04 | <0.05 |

*p<0.05 vs post dive rest; °p<0.05 vs pre dive rest

**Table 4** Spiroergometry for 10 experienced divers at rest and at 300 W on a bicycle each before and after the dive trip.

| | Rest | | | 300 W | | |
|---|---|---|---|---|---|---|
| | pre dive | post dive | p | pre dive | post dive | p |
| respiration rate [/min] | 13±4 | 14±4 | 0.573 | 38±7 | 40±11 | 0.553 |
| ventilation (vE) [l/min] | 10±4 | 12±3 | <0.05 | 134±41 | 129±34 | 0.508 |
| tidal volume [l] | 0.77±0.24 | 0.86±0.28 | <0.05 | 3.53±1.27 | 3.23±1.07 | 0.472 |
| $EQ_{O2}$ | 22.1±4.9 | 27.3±3.0 | <0.05 | 30.8±5.7* | 31.3±5.4 | 0.337 |
| RER | 0.8±0.1 | 1.2±0.1 | <0.05 | 0.9±0.1 | 1.2±0.1 | 0.208 |

EQO2: ventilatory equivalent for oxygen; RER: Resp. Exchange Ratio CO2/O2

*p<0.05 vs pre dive rest

### Body plethysmography

All static and dynamic pulmonary measures were not significantly affected by the diving trip (Table 1), nor were airway resistance (Raw) or the carbon monoxide transfer factor (TLCO).

### Spiroergometry

Heart rate and blood pressure. After the trip, heart rate (HR) at rest was slightly higher (7%) than before the trip (Figure 1). With increasing loads, HR increased almost linearly and did not exhibit significant differences between pre- and post trip values (Table 2). At a load of 300 W, HR had more than doubled compared with resting conditions before and after the dive trip (Table 2).

Systolic and diastolic blood pressures at rest were within the normal range. No differences were found between before and after the dive trip. At a load of 300 W the systolic pressures had increased by about 70%, while diastolic pressures remained almost unchanged (Table 2).

Blood gases and pH. At rest, arterial $pO_2$ was slightly increased after the trip, $pCO_2$ was significantly decreased, whereas pH was unchanged (Table 3). At 300 W, $pO_2$ was slightly decreased, $pCO_2$ was almost unchanged, while pH was significantly increased after the dive trip (Table 3) compared with pre dive values.

Before the trip, $pO_2$ during ergometry, ie, from rest to 300 W, remained almost unchanged but significantly decreased after the trip. In turn, $pCO_2$ significantly decreased before but remained unchanged after the trip. During ergometry, pH decreased significantly before and insignificantly after the trip. In addition, post dive pH at 300 W was higher compared with the pre dive value at 300 W (Table 3).

Respiration; Post dive respiration rate at rest and at 300 W was slightly higher compared with pre dive values. At rest, post dive minute volume and hence tidal volume were significantly increased. At 300 W, pre dive tidal volume had increased by 460%, but only by 380% post dive trip. The ventilatory oxygen equivalent at rest was increased after the trip and was also higher at 300 W than before the dive (Table 4). The respiratory exchange ratio (RER) after the trip was significantly increased at rest, but not at 300 W (Table 4).

Oxygen uptake and aerobic threshold. At rest, oxygen consumption per body mass ($VO_2$) was almost identical before and after the trip (4.8±1.1 vs 4.8±1.0 ml/min/kg). However, peak $VO_2$, ie, at 300 W, was decreased by 8.2% after the trip (45.1±9.9 vs 41.7±9.8 ml/min/kg; $p<0.05$) (Figure 2 top). In parallel, the aerobic threshold (AT) was decreased (234±43 vs 186±39 W; $p<0.05$; Figure 2 bottom).

### Echocardiography

Left ventricular end-diastolic and end-systolic dimensions at rest were almost identical before and after the trip (Table 5). Thus, fractional shortening and ejection fraction remained almost unchanged. As heart rate and stroke volume had not changed significantly, resting cardiac output remained unchanged, as well.

With no major changes in blood pressure and maintained cardiac output, total peripheral resistance was maintained after the dive trip.

The diameters of left atrium, right ventricle and aortic root were all moderately increased (Table 5). After the dive trip, the maximum aortic blood velocity (AOV) was slightly increased, while the ratio (E/A) of the early (E) to late (A) left ventricular filling velocities was significantly increased (Table 5).

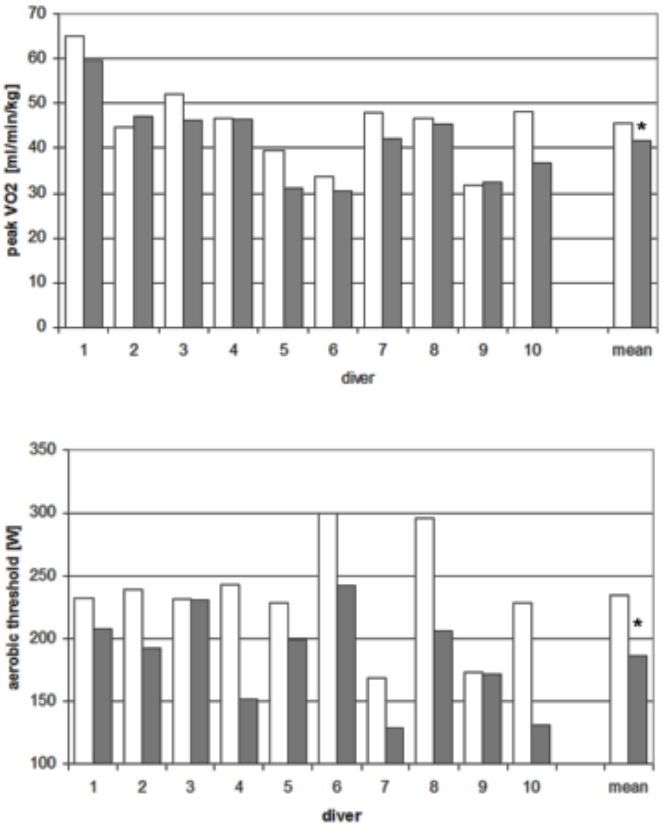


**Figure 2.** ***top***: Peak VO2 uptake in ten divers before (white) and after (grey) the dive trip. Mean peak VO2 was significantly decreased after the trip. *p<0.05 indicates significant difference. ***bottom:*** Aerobic threshold in ten divers before (white) and after (grey) the dive trip. Mean aerobic threshold was also significantly decreased after the trip
*p<0.05 vs pre dive values.

**Table 5** Cardiovascular measures of ten experienced divers at rest.

| | pre dive | post dive | p |
|---|---|---|---|
| LVedd [mm] | 55.0±3.6 | 55.6±4.6 | 0.444 |
| LVesd [mm] | 33.8±2.9 | 33.7±4.3 | 0.928 |
| SV [ml] | 68.6±9.9 | 72.8±16.6 | 0.251 |
| EF [%] | 68±5 | 72±7 | 0.508 |
| CO [l/min] | 5.7±1.2 | 5.7±0.5 | 0.869 |
| TPR [mmHg/ml] | 7.3±2.5 | 8.2±1.9 | 0.269 |
| left atrium [mm] | 36.7±4.6 | 37.3±4.3 | 0.239 |
| right ventricle [mm] | 34.5±3.3 | 35.0±4.8 | 0.697 |
| AOV [m/s] | 1.02±0.20 | 1.04±0.16 | 0.247 |
| E/A | 1.74±0.47 | 1.93±0.33 | 0.050 |

EF: ejection fraction; CO: cardiac output; SV: stroke volume; TPR: total peripheral resistance

AOV: maximum aortic blood velocity

## DISCUSSION

The five most important findings of this retrospective study are:

- Intense diving within 5 days using oxygen-enriched breathing gases impaires pulmonary function,

- the presumed injured alveolar-capillary barrier led to an reduced gas exchange at high work load,
- the pulmonary injury can be at least partially assessed within one week post-dive.
- A major physiological study on open-water divers is feasible by using data generated in a single medical office with an adequately equipped laboratory.

### Limitations

Because of the limited sample size this study has an exploratory character. Fortunately, data sets were complete for all ten individuals, so that paired comparisons could be performed.

If the immediate diving-induced effects of hyperoxia were in the focus, then a field study is mandatory with adequate on-site personnel and equipment. The presented setting seems only meaningful, if lasting effects were in the focus. Such effects have already been described for up to 2–3 days after a single dive (27) or up to 2 days after repeated exposure to elevated $pO_2$.[35] Here, we report for the first time on pulmonary effects lasting for up to one week after 5 days of intensively diving.

Any toxic effects, on the other hand, might have disappeared if measurements were much delayed, because protective mechanisms exist in hyperoxia.[1] For example, oxidative stress during diving also influences recruitment or survival of endothelial progenitor cells,[6] causes persistent change in pathways controlling apoptosis,[10] or induces an antioxidant response in plasma and erythrocytes without the appearance of cellular damage,[38] thus protecting the diver against damage.[24]

### Participants

Our ten participants seem representative for sport divers. Unlike recreational divers, they are organised in a diving club and regularly practice to maintain a certain level of fitness. Thus, at their age (=32±8 yrs), they easily acquire the fitness-to-dive certificate. The average BMI of our subjects was relatively high, because six of them were in the 'overweight' or 'obese' BMI category. In the German population of that age, about 30% belong to these categories.[12] It is remembered that the BMI does not take into account the muscle / fat relation. As some of the participants were competitive athletes, the relatively high BMI values indicate an increased muscle mass. To support this view: The maximum power in one participant was equal to 404 W.

For some years the proportion of female divers in German dive clubs has been increasing but even now amounts to only about one third. Among the tech divers, this proportion is even smaller.

### Dives

On average 11 dives were performed during 5 days of the dive trip in relatively warm water (27–28 °C) with good visibility and without noteworthy current. All dives were performed after stringent planning. In particular, the maximum limit of 1.3 bar for the oxygen partial pressure ($pO_2$) was respected. As a result, oxidative stress was moderate. In one single diver, oxygen toxicity units (OTU) had accumulated to maximum 1,150 units after 5 days of diving. Because a total of 2,300 OTU is allowed after 5 consecutive days of exposure,[14] oxygen-induced injuries should, if anything, have been small.

### Blood

The effects of hyperoxia on blood cells much depends on the type of dive. A reduction in haemoglobin concentration is consistently reported after deep saturation dives.[17] In contrast, no statistically significant increase in lipid per-oxidation was reported either in blood plasma or in erythrocytes after a 40-min air dive to 6 m.[24] After 25-min air diving to 40 m circulating erythrocytes and haemoglobin concentrations remained unaltered, but neutrophils were increased, indicating an inflammatory process.[38]

After a considerable delay in measuring any effects, it is not surprising that neither decreases in haematocrit and erythrocyte counts nor an increase in leukocyte counts were noticeable in this study.

### Pulmonary System

Hyperoxia increases production of reactive oxygen species, which can be toxic to the pulmonary system[19,33] in particular to pulmonary endothelial and alveolar epithelial cells.[31] The resulting damage is characterised by an inflammatory response[10] and a dysfunctional alveolar-capillary barrier, which can lead to impaired gas exchange and pulmonary edema.[31]

The damage seems to depend not only on the dives but also on the divers. In older experienced recreational divers, respiratory function was decreased compared with the standard population, suggesting slight small airways disease.[19] On the other hand, substantial exposure to elevated $pO_2$ while diving was not associated with an accelerated long-term decline in respiratory function in professional divers.[39]

In the present study neither restrictive and obstructive measures nor total airway resistance before and after the dive trip were significantly different. On the other hand, the transfer capacity for carbon monoxide is acutely reduced after breathing gases with an elevated pO[29,34] Here, this measure was not significantly reduced some days after the dive trip.

Other measures of resting respiratory function had changed after the trip. Minute volume increased at maintained respiration rate, and hence tidal volume was increased. As a result, the ventilatory oxygen equivalent (EQO2) was increased decreasing breathing economy.

### Cardiovascular System

Multiple factors acutely affect hemodynamics during a dive. Hyperoxia is the principal factor which increases parasympathetic tone[8,43] thereby decreasing heart rate.[2,21] Another effect is arterial vasoconstriction,[18] which increases afterload[13,25] and decreases left ventricular (LV) performance[25] and right ventricular function due to overload.[7] Hyperoxia additionally impairs LV relaxation and increases filling pressures.[22]

Moreover, hyperoxia increases arterial stiffness.[23] Beside its effects on vascular tone, hyperoxia impairs endothelial function thereby reducing flow mediated dilatation.[23,27,45]

In this study, all cardiac measures were unchanged after the diving trip. In addition, with preserved heart rate, no dive-induced changes in ejection fraction and cardiac output were observed. Accordingly, no persistent post-dive vasoconstrictive effects of hyperoxia were detected, as total peripheral resistance was maintained. On the other hand, the ratio between early and atrial LV filling (E/A ratio) was significantly increased pointing to an improved LV filling pattern.

Although function may be impaired for up to

3 days after a single day of diving,[28] measures of cardiac function in this study were either maintained or even improved roughly five days after repetitive dives.

### Working Capacity

Oxygen uptake ($VO_2$) increased almost linearly with exercise intensity before and after the diving trip. But exercise intensity can continue to increase via anaerobic energy production. The point at which $VO_2$ plateaus is $VO_{2max}$. To strain the participants to full capacity, $VO_2$ at 300 W was assessed. This measure was decreased after the dive trip in parallel with the aerobic threshold.

The oxygen transport capacity of the blood is one important determinant of $VO_2$. In this study, cardiac output, haemoglobin concentration and arterial $pO_2$ were unchanged at rest. However, the decreased post-dive $pO_2$ at 300 W suggests a reduced gas exchange that could possibly have contributed to the decreased peak $VO_2$.

The other important determinant affecting maximum working capacity is oxygen supply. Yet, after the dive trip, neither restrictive nor obstructive measures of pulmonary function were noticeably impaired during resting conditions. In addition, pulmonary transfer capacity, symptomatic of either alveolar membrane injuries or pulmonary capillary alterations,[20] was not affected at rest after the diving trip. Hence, likely reduced transfer capacity only became evident during high work load, i.e. using spiroergometry.

Whether this injury must be blamed on dysfunctional pulmonary capillary endothelium, alveolar epithelium, or compromised small airways or all three options remains speculative.

## CONCLUSION

Pulmonary injury after successive dives with oxygen-enriched breathing gases, ie, with elevated levels of oxidative stress, can be observed within one week after the end of the dive series via an increased ventilatory equivalent for oxygen at rest and both a decreased $pO_2$, a decreased peak $VO_2$ and a decreased aerobic threshold at peak workload. It is shown that an ambitious experimental open-water study is sensibly feasible even if the medical laboratory and with the dive site being located at great distances.

The reported results imply a need to pay more attention to practicing a conservative dive style and on the necessity of sufficient pauses between deep dives.

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## REFERENCES

1 Allen BW, Demchenko IT, Piantadosi CA. Two faces of nitric oxide: implications for cellular mechanisms of oxygen toxicity. *J Appl Physiol Bethesda Md 1985* 2009; **106**: 662–667

2 Boussuges A, Lafay V. [Changes in cardiac function during recreational diving]. *Arch Mal Coeur Vaiss* 1997; **90**: 263–268

3 Boussuges A, Molenat F, Carturan D, Gerbeaux P, Sainty JM. Venous gas embolism: detection with pulsed Doppler guided by two-dimensional echocardiography. *Acta Anaesthesiol Scand* 1999; **43**: 328–332

4 Brebeck A-K, Deussen A, Schmitz-Peiffer H, Range U, Balestra C, Cleveland S, Schipke JD. Effects of oxygen-enriched air on cognitive performance during SCUBA-diving - an open-water study. *Res. Sports Med*. Print ( April 11, 2017). doi: 10.1080/15438627.2017.1314289.

5 Chabot F, Mitchell JA, Gutteridge JM, Evans TW. Reactive oxygen species in acute lung injury. *Eur Respir J* 1998; **11**: 745–757

6 Culic VC, Van Craenenbroeck E, Muzinic NR, Ljubkovic M, Marinovic J, Conraads V, Dujic Z. Effects of scuba diving on vascular repair mechanisms. *Undersea Hyperb Med J Undersea Hyperb Med Soc Inc* 2014; **41**: 97–104

7 Dujic Z, Obad A, Palada I, Valic Z, Brubakk AO. A single open sea air dive increases pulmonary artery pressure and reduces right ventricular function in professional divers. *Eur J Appl Physiol* 2006; **97**: 478–485

8. Eckenhoff RG, Knight DR. Cardiac arrhythmias and heart rate changes in prolonged hyperbaric air exposures. *Undersea Biomed Res* 1984; **11**: 355–367

9 Eftedal I, Jørgensen A, Røsbjørgen R. Early genetic responses in rat vascular tissue after simulated diving. *Physiol Genomics* 2012; **18**: 1201–7

10 Eftedal I, Ljubkovic M, Flatberg A, Jorgensen A, Brubakk AO, Dujic Z. Acute and potentially persistent effects of scuba diving on the blood transcriptome of experienced divers. *Physiol Genomics* 2013; **45**: 965–972

11 Fock A, Harris R, Slade M. Oxygen exposure and toxicity in recreational technical divers. *Diving Hyperb Med* 2013; **43**: 67–71

12 GBE. Informationssystem Gesundheitsberichterstattung Bundes: 2015. http://www.gbe-bund.de/gbe10/pkg_isgbe5.prc_isgbe?p_uid=gast&p_aid=0&p_sprache=D.

13 Gole Y, Louge P, Boussuges A. Specific diving training-induced arterial circulation changes. *Br J Sports Med* 2009; **43**: 526–30

14 Hamilton RW. Tolerating oxygen exposure. *SPUMS J* 1997; **27**: 43–47

15 Hemelryck W, Rozloznik M, Germonpre P, Balestra C, Lafere P. Functional comparison between critical flicker fusion frequency and simple cognitive tests in subjects breathing air or oxygen in normobaria. *Diving Hyperb Med* 2013; **43**: 138–142

16 Herdy AH, Uhlendorf D. Reference values for cardiopulmonary exercise testing for sedentary and active men and women. *Arq Bras Cardiol* 2011; **96**: 54–59

17 Hofsø D, Ulvik RJ, Segadal K. Changes in erythropoietin and haemoglobin concentrations in response to saturation diving. *Eur J Appl Physiol* 2005; **95**: 191–6

18 Lafay V. [The heart and underwater diving]. *Arch Mal Coeur Vaiss* 2006; **99**: 1115–1119

19 Lemaitre F, Tourny-Chollet C, Lemouton M. Ventilatory function in experienced recreational scuba divers: Evidence of small airways disease? *Int J Sports Med* 2006; **27**: 875–9

20 Lipscomb D, Patel K, Hughes J. Interpretation of increases in the transfer coefficient for carbon monoxide (TLCO/VA or KCO). Thorax 33: 728–33, 1978.

21 Lund V, Laine J, Laitio T, Kentala E, Jalonen J, Scheinin H. Instantaneous beat-to-beat variability reflects vagal tone during hyperbaric hyperoxia. *Undersea Hyperb Med J Undersea Hyperb Med Soc Inc* 2003; **30**: 29–36

22 Mak S, Azevedo ER, Liu PP. Effect of hyperoxia on left ventricular function and filling pressures in patients with and without congestive heart failure. *Chest* 2001; **120**: 467–73

23 Marinovic J, Ljubkovic M, Breskovic T, editors. Effects of successive air and nitrox dives on human vascular function. *Eur J Appl Physiol* 2012; **112**: 2131–7

24 Mila-Kierzenkowska C, Woźniak A, Szpinda M. Oxidative stress in blood of healthy people after diving. *J Sports Med Phys Fit* 2015; **55**: 352–60

25 Molénat F, Boussuges A, Grandfond A. Haemodynamic effects of hyperbaric hyperoxia in healthy volunteers: an echocardiographic and Doppler study. *Clin Sci* 2004; **106**: 389–95, 2004.

26 Neder JA, Nery LE, Peres C, Whipp BJ. Reference values for dynamic responses to incremental cycle ergometry in males and females aged 20 to 80. *Am J Respir Crit Care Med* 2001; **164**: 1481–1486

27 Obad A, Marinovic J, Ljubkovic M. Successive deep dives impair endothelial function and enhance oxidative stress in man. *Clin Physiol Funct Imaging* 2010; **30**: 432–8

28 Obad A, Palada I, Valic Z, Ivancev V, Bakovic D, Wisloff U, Brubakk AO, Dujic Z. The effects of acute oral antioxidants on diving-induced alterations in human cardiovascular function. *J Physiol* 2007; 578: 859–870, 2007.

29 van Ooij PJAM, van Hulst RA, Houtkooper A, Sterk PJ. Differences in spirometry and diffusing capacity after a 3-h wet or dry oxygen dive with a PO(2) of 150 kPa. *Clin Physiol Funct Imaging* 2011; **31**: 405–410

30 Pendergast DR, Senf CJ, Fletcher MC, Lundgren CEG. Effects of ambient temperature on nitrogen uptake and elimination in humans. *Undersea Hyperb Med J Undersea Hyperb Med Soc Inc* 2015; **42**: 85–94, 2015.

31 Porzionato A, Sfriso M. Effects of hyperoxic exposure on signal transduction pathways in the lung. *Respir Physiol Neurobiol* 2015; **209**: 106–14

32 Shykoff BE. Pulmonary effects of submerged oxygen breathing: 4-, 6-, and 8-hour dives at 140 kPa. *Undersea Hyperb Med* 2005; 351–61

33 Shykoff BE. Pulmonary effects of submerged oxygen breathing in resting divers: repeated exposures to 140 kPa. *Undersea Hyperb Med* 2008; **35**: 131–143

34 Shykoff BE. Cumulative effects of repeated exposure to pO2 = 200 kPa (2 atm). *Undersea Hyperb Med J Undersea Hyperb Med Soc Inc* 2014; **41**: 291–300

35 Shykoff BE, Florian JP. Small Airway Function after Repeated Dives with Elevated PO2: 2057 Board #209 June 2, 3: 30 PM - 5: 00 PM. *Med Sci Sports Exerc* 2016; 48: 576

36 Smith JL. The pathological effects due to increase of oxygen tension in the air breathed. *J Physiol* 1899; **24**: 19–35

37 Sureda A, Batle JM, Capo X, Martorell M, Cordova A, Tur JA, Pons A. Scuba diving induces nitric oxide synthesis and the expression of inflammatory and regulatory genes of the immune response in neutrophils. *Physiol Genomics* 2014; **46**: 647–654

38 Sureda A, Ferrer MD, Batle JM, Tauler P, Tur JA, Pons A. Scuba diving increases erythrocyte and plasma antioxidant defenses and spares NO without oxidative damage. *Med Sci Sports Exerc* 2009; **41**: 1271–1276

39 Tetzlaff K, Friege L, Theysohn J, Neubauer B, Muth CM. Lung function in military oxygen divers: a longitudinal study. *Aviat Space Environ Med* 2005; **76**: 974–977

40 Thorsen E, Kambestad BK. Persistent small-airways dysfunction after exposure to hyperoxia. *J Appl Physiol Bethesda Md 1985* 1995; **78**: 1421–1424

41 Vann RD, Butler FK, Mitchell SJ. Decompression illness. Review. *Lancet* 2011; **8**: 153–64

42 Wang Q, Guerrero F, Mazur A. Reactive Oxygen Species, Mitochondria, and Endothelial Cell Death during In Vitro Simulated Dives. *Med Sci Sports Exerc* 2015; **47**: 1362–71

43 Winklewski PJ, Kot J, Frydrychowski AF, Nuckowska MK, Tkachenko Y. Effects of diving and oxygen on autonomic nervous system and cerebral blood flow. *Diving Hyperb Med* 2013; **43**: 148–156

44 Xu D, Guthrie M JR, Mabry S. Mitochondrial aldehyde dehydrogenase attenuates hyperoxia-induced cell death through activation of ERK/MAPK and PI3K-Akt pathways in lung epithelial cells. *Am J Physiol Lung Cell Mol Physiol* 2006; **291**: 966–75

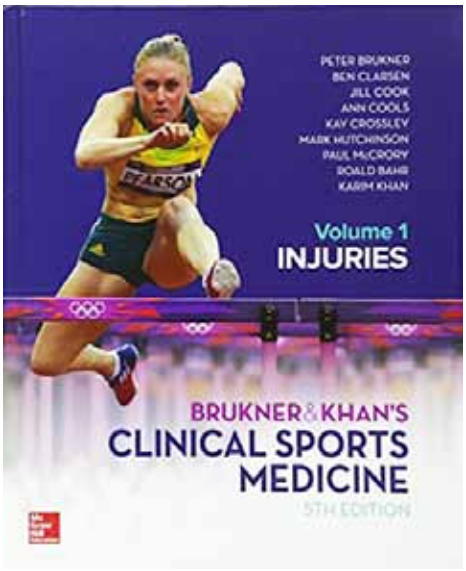
45 Zenske A, Kähler W, Koch A, Stirban A, Krüll M, Schipke JD. Is nitrox dangerous for the recreational diver? In: 42. Annual Scientific Meeting of the EUBS. Geneva: 2016, p. 32.

46 jumpsaildive [Online]. 2017. https://jumpsaildive.com/diving [18 Apr. 2017].

# Brukner and Khan's CLINICAL SPORTS MEDICINE 5th Edition Volume 1 - Injuries

STUART ARMSTRONG

Undoubtedly the prime reference text for sports medicine, the sports medicine equivalent of Harrison's Principles of Internal Medicine, has a new version out and it is so extensive it has now been split into 2 volumes. I have to thank my close colleague Dr Chris Milne for lending me his copy of volume 1 – Volume 2 is due to be released shortly and will encompass the medical aspects of sports medicine practice.




Volume 1 is split into 3 sections encompassing fundamental principles, regional problems and practical sports medicine. The bulk of the material is in the section on regional problems and similar to previous editions gives a clear and concise in-depth look at all aspects of sports medicine injuries. It is organised in a logical anatomical manner and as always includes some of the best photography seen in modern text books. There is also good use of graphics to complement both the pictures and text.

Additional chapters include pain, core stability, patient reported outcome measures, training programming/ prescription and return to play decisions. I think these are good sections to include and while they were part of previous editions they are now big enough subjects to warrant their own dedicated section.

But this new edition is not just about additional chapters it also includes some important updates to existing chapters. Highlights include updating the concussion section to include SCAT3 and SCAT3 child, although as is the nature with text book publications this has already been superseded by the publication of SCAT 5.[1] The section on shoulder examination, back pain and tendon injuries includes the specificity and sensitivity of examination findings. The importance of biopsychosocial aspects in low back pain and more detail on the management of hamstring avulsion injuries.

There are very few negatives around this publication. The main issue I have is the change of references to no longer being included at the end of each chapter, but being accessed online. I am sure this is done for practical reasons to save publication space, but does make the process of reading in further depth rather laborious.

Overall a very good update to an already essential sports medicine text book. I will look forward to purchasing and reading volume 2 when it comes out and from what I have seen of volume 1, you will too.

## References

1 The Sport Concussion Assessment Tool 5th Edition (SCAT5): Background and rationale. R J Echemendia, W Meeuwisse, P McCrory etal. British Journal of Sports Medicine 2017; 51 848-850

# Brain Storm - Concussion Conference

## Havelock North, 17-18 November 2017

STEPHEN KARA

For those that were not able to sneak off to the sunny Hawkes Bay, this inaugural conference presented both an update as to where the evidence is at from some leading practitioners but also the opportunity to sample the 'fruits' that the regions produces. The conference was well attended with over 150 delegates from many health professional backgrounds, all involved in dealing with the concussed patient at varying ends of the time spectrum and causation.

The legacy of pioneering New Zealand practitioners Mr Phillip Wrightson (Neurosurgeon) and Dr Dorothy Gronwall (Neuropsychologist) in the area of concussion was an appropriate opening as the history of Managing Concussion in the NZ Context took us on a journey as to how we got to where we are.

Keynote speaker and Concussion in Sport Group member, Prof Gavin Davis (Neurosurgeon), presented an overview on the Berlin Concussion Consensus Statement 2016. The history, the process, the outcome and the publications from this group informed the group of the rigorous methodological process in the production of the consensus statement. Prof Davis referred to each of the individual 11 R's of the statement and for those involved with sporting teams this is a must with some valuable practical clinical tools. The statement is available on Open Access, BJSM April 2017. He emphasised the essential first 2 R's – Recognise and Remove - for non-medical people within the community and he reiterates my own opinions that these 2 R's are all that is required initially and if we can achieve this consistently then we will give the patient the best chance of a good outcome. The usefulness of the SCAT-5 as both a diagnostic tool and a serial symptom monitoring tool (note the 10 word lists in the SCAT-5 to negate a potential ceiling effect for those that clinicians think can easily cope with the 5 word list) formulated the Re-evaluate, but personally as a diagnostic tool valid only within the first 3-4 days post injury. Rest recommendation of 24-48 hours is now standard with evidence supporting a slower recovery with more prolonged rest,[1] leading practitioners to prescribe light activities post this period using symptom provocation as a measure for overdoing it. Rehabilitation was addressed by the next Keynote Dr John Leddy (see below) with a more active approach being undertaken all around. Whom to Refer? Those adults with persistent symptoms at Day 14 post-injury, children <13yrs age with persistent symptoms at 4 weeks post-injury (Prof Davis stated that in the population he deals with this is approximately 30%), those with modifiers that could lead to a slower Recovery and those with a previous history of a protracted concussion or multiple concussions. Return to sport must follow a graded protocol once the patient is asymptomatic. Prof Davis spoke of the need for this to be a tailored approach specific to the individual. Whilst he is correct in theory, the reality is different. National sporting organisations mandate a minimum stand-down period as a conservative 'protect the greater good' philosophy and to achieve an individualised approach for all we would need to up-skill the primary care workforce to cope with such a protocol. Reconsider was Prof Davis' panel question and he used this to expand more upon the process around systematic review that led to the statement production. The key point here was that children must learn (be back in school) prior to any return to sport, a message that parents must understand. One slide only summed up the controversial Residual Effects as he avoided much comment on the association of chronic traumatic encephalopathy (CTE) whilst Risk Reduction for helmet use has only been shown in skiing and snowboarding, with equivocal evidence for the use of mouthguards. It does seem a 'no-brainer' to mandate the wearing of mouthguards in contact sport with no real downside. However there was no mention of the use of cervical strengthening especially the deep cervical flexors as an intervention with minimal negative effects to reduce risk.[2]

Dr John Leddy, from the internationally regarded Buffalo group, beamed in on a live video, to present his lecture titled 'Sport & the Buffalo Concussion Treadmill Test'. These opening lectures had a real focus on the end of the concussion spectrum that our members have a real affinity to. Dr Leddy commenced with some nice neuro-imaging data showing physiological changes on diffusion tensor imaging in the brainstem, at a level where vagal nerve and vestibular nuclei are in close proximity. Studies using cold water immersion of a single limb have shown a blunting of the autonomic response to heart rate (HR), blood pressure (BP), stroke volume (SV) and cardiac output (CO) reinforcing the imbalance of sympathetic and parasympathetic systems. Concussion and the deconditioning effects are also associated with cerebral blood flow reduction, neuroplasticity impairment, psychological and mood dysfunctions. The Buffalo group is internationally recognised for their work in the use of sub-symptom threshold exercise prescription and use of a modified Balke treadmill protocol in the assessment of persistent post-concussion symptoms.[3-5] Exercise effects on the brain and on central and systematic physiology improve autonomic system balance and CO2 sensitivity, improvement in cerebral blood flow, up-regulate brain derived neurotropic factor (BDNF) whilst having benefits for sleep and mood. Using exercise tolerance testing as a tool for other pathologies was discussed and this group has published on the high rates of those presenting with additional pathologies. Cervicogenic disorders (also termed cervico-cephalic syndrome) was an important factor with similar symptoms, needing to be screened for in those still symptomatic. Dr Leddy presented additional work currently in press trialing earlier use exercise at day 5 post-injury with no additional increase in symptoms or symptom severity and no delay in time to recovery. Validation trials are in press for publication for the use of the Buffalo Concussion Bike Test (BCBT) in lieu of treadmill testing with specific details of a

weight gauged increase in resistance to match similar treadmill progressions.

Sports and Exercise Medicine Physician, Dr Hamish Osborne, Otago, added to the sports concussion theme with a presentation on the treatment and management in this country including the graduated return to sport.
Dr Nathan Zasler, Medical Director of the Concussion Care Centre of Virginia, USA, again beamed in on live video, providing some very simple messages on improving outcomes in concussive brain injury:

1 Early prompt assessment and treatment.
2 Be cognisant of the language you use to describe the condition.
3 Educate early on typical recovery
4 Dispel misunderstandings
5 Listen and acknowledge.

Other presentations from national based practitioners in the field are summarised below:

- Niall McCormack (Optometrist) – refer those immediately with diplopia but those with other visual abnormalities can be referred at a time depending on the level of dysfunction as they can resolve given time (although older age refer early).
- Dr Robin Sekerak (Rehabilitation Medicine Specialist, Concussion Services) – the bio-psychosocial model of concussion reinforcing the multidisciplinary team (MDT) approach for those with persistent symptoms. Our challenge in the sports concussion patient is to recognise these patients early and refer for MDT approach. Markers of high symptom severity scores (representing marked somatization), vestibular signs, adolescent females and modifiers may be some of these indicators
- Dr Doug King (Clinical Nurse Specialist, Hutt Valley Hospital ED) – presenting on his previously published data on the use of King-Devick testing as a sideline indicator of concussion in community rugby and rugby league. Caution here in that whilst Doug is passionate about this modality as being a simple and effective test, there is no evidence to validate this tool as a stand-alone test and could be considered only as part of your sideline screening kit.[6] Further studies are awaiting publication from the use of this tool in conjunction with the HIA from UK Premiership Rugby last season
- Dr Debbie Snell (Rehabilitation Medicine Specialist, Concussion Services) – on the topic of neuropsychological consequences of injury, she presented data from her previous research screening people 6 weeks post-injury using psychological measures, with 3 clusters identified. Those with more psychological symptoms at this early stage, representing fear and anxiety re their condition or background traits, had the slowest recovery and were still not asymptomatic at 6 months post-injury. Perhaps this begs us to screen early in those patients whom are presenting with persistent high symptoms as further information for early referral to a MDT. She reassured us that mild cognitive symptoms on group data will have resolved by 3 months post-injury but acknowledged as this is group data, individual differences will occur. Those that avoid cognitive activity (termed cognophobia) demonstrate a fear avoidance behaviour and this means they also avoid physical activity (see Dr John Leddy aforementioned).
- Dr Richard Seemann (Rehabilitation Medicine Specialist, Concussion Services) spoke on headache in concussion with nothing too much new here. Consider the role of the cervical spine in these patients and need to screen for it. On the pharmacological front useful 'non-drug' type medications for this condition in his opinion were Riboflavin and Melatonin at 3mg nocte
- Hannah Carter (Vestibular Therapist) used a case presentation with video support to demonstrate the vestibular dysfunction that patients present with and the successful role of rehabilitation. If you are not screening for this in your persistently symptomatic patients then you may well be missing. Use of the vestibulo-ocular motor screening (VOMS) tools is recommended. Should we be commencing rehab earlier rather than waiting a recommended time period of 14 days if patients are positive for this dysfunction at initial presentation?
- Dr Danielle Salmon (NZ Rugby) presented NZR strategy on concussion with the use of RugbySmart and the intended roll out into secondary schools of baseline testing via CSx with a database for use by practitioners.

So watch out for the conference, purported to now be an annual event, for those managing sports concussion patients.

## REFERENCES


1 Silverberg ND, Iverson GL. Is rest after concussion the best medicine? Recommendations for activity resumption following concussion in athletes, civilians, and military service members. *J Head Trauma Rehabil* 2013; **28**:250-9.

2 Collins C, Fletcher E, Fields S, et al. Neck strength: a protective factor reducing risk in concussion in high school sports *J Prim Prev* 2014.

3 Leddy J, Kozlowski K, Donnelly J, Pendergast D, Epstein L, Willer B. A preliminary study of subsymptom threshold exercise training for refractory post-concussion syndrome. *Clin J Sport Med* 2010; **20**:21-7.

4 Leddy J, Hinds A, Sirica D, Willer B. The role of controlled exercise in concussion management. *Journal of Physical Medicine and Rehabilitation* 2016: S91-S100.

5 Leddy J, Baker J, Kozlowski K, Bisson L, Willer B. Reliability of a Graded Exercise Test for Assessing Recovery from *Concussion. Clin J Sport Med* 2011; **21**:89-94.

6 Molloy J, Murphy I, Gissane C. The King-Devick(K-D) test and concussion diagnosis in semi-professional rugby union players. *J Sci Med Sport* 2017; **20**:708-11.