ACL prevention in Norway and Australia

By John Orchard

The 2nd World Sports Injury Prevention Congress in Tromsø was a great opportunity to become aware of the latest advances in the field in both Norway and around the world. Although many sports injury areas were discussed, prevention of knee anterior cruciate ligament (ACL) injuries is still rightly considered the Holy Grail. ACL injuries are common in many of the world's most popular sports and they regular lead to knee osteoarthritis later in life. ACLs also disproportionately affect women. The rates of ACL injuries appear to have been increasing, partly because women worldwide have been increasingly participating in change-ofdirection sports in recent years.

Traditionally, ACL risk factors are divided into potentially-preventable and non-preventable risk factors and then into intrinsic (athlete-related) and extrinsic (non-athlete-related) risk factors. As an example, female gender is an intrinsic but non-preventable risk factor. My personal interest has been in researching some of the potentiallypreventable extrinsic risk factors for ACL injuries, particularly related to surface type in outdoor sports. In this sense I am unfashionable, in that the vast majority of researchers in the field of ACL prevention are firmly focused on studying the potentiallypreventable intrinsic risk factors. The Tromsø conference confirmed my pessimism that extrinsic risks were being ignored relative to intrinsic factors, but raised my hopes that the researchers who specialise in intrinsic risks were actually on to something. There seem to be an increasing number of trials showing, almost exclusively in females, that balance exercises can decrease the risk of ACL injuries 1. Importantly, some of the mechanisms by which these exercises work were becoming understood. These included increasing reaction time 2 and getting the semitendinosus tendon to fire earlier in a side-stepping movement ³. Both of these factors may contribute to decreasing the valgus force on the knee. It is promising that to date the ACL rate in Norwegian women,s handball may have lowered 4 but no major change has yet been seen in women,s college sports in the USA such as basketball and soccer 5.

There is some recent published extrinsic risk factor data, not surprisingly also coming out of Norway. Although I have argued recently that New Zealand is home to the world's best nationwide injury surveillance system 6, the greatest volume of quality of specific studies are certainly arising out of the Oslo Sports Trauma Research Centre (OSTRC). The Norwegians have found that wooden floors are less risky than artificial floors for ACL injury in handball 7. They have also compared ACL injury rates in Norway for football (soccer) games on natural grass and artificial turf and found no significant difference between the two surface types 8. I attended a session in which one of the most recent papers was presented and asked the author/ presenters which types of natural grass were most commonly used on football fields in Norway. This question seemed to come from left field, so to speak, and neither of the presenters knew anything about natural grass other than it wasn't artificial. To me this may be a critical omission of observation that may, sadly, lead to bad decision making up to the highest echelons in FIFA. There is no doubt that, in certain circumstances. artificial turf may be a 'safe' surface in soccer. However, I am concerned that these 'circumstances' might be cold weather and it may be a mistake to give artificial surfaces the green light in warm climates 9. Whether I am right depends on whether you believe the observational data from Europe in soccer is a better predictor of outcomes in other climates than the observational data from



Figure 1 - with Willem van Mechelen (Netherlands) under the midnight sun in Tromsø



Figure 2 – the old gravel surface used in arctic climates for soccer games

other forms of football. Even though there is one study (with a small number of ACLs) from soccer in the USA ¹⁰, the majority of warm-climate data on surface condition comes from other football codes ¹¹. I personally think it should apply for injuries categories such as ACLs in soccer.

In other football codes, rye grass has been found to have lower shoe-surface traction than other grasses and lead to a lower risk of ACL injuries ¹². With respect to all natural grasses, traffic appears to be a protective factor (the grass roots and lateral growth are cut up, which tends to lower shoe-surface traction) ¹³. Hence an 'early-season' bias for most outdoor football competitions is observed. Fans of the AFL will have noted that seven ACL injuries occurred in the pre-season period this year but the rate of ACL injury has been much lower in the regular season, a pattern which

Figure 3 – artificial turf, a big improvement on gravel for soccer in the deep north

is seen in most years. ACL risk on natural grass will always drop as the season progresses unless there is growth of new grass (which happens in the spring and summer and when new portable slabs are brought in ¹⁴). Some grasses have intrinsically more lateral growth and lead to more shoe-surface traction than others. Basically of the three most commonly used natural grass types around the world. ryegrass has less lateral

growth than the other two ¹³ and is more likely to result in lower shoe-surface traction and lower risk of ACL injuries (Table 1).

The situation with artificial turf is different. Traffic doesn't change the shoe-surface traction at all - if the artificial surface was cut up by traction then it wouldn't last! However, Astroturf seems to lead to a much higher risk of ACL injury in hot temperatures than in cold temperatures 915. It is notable that the actual temperature of an artificial surface (like sand at the beach) is much closer to the ambient temperature in hot weather than it is for natural grass (which can stay remarkably cool). In a football competition with games played on artificial turf over autumn to winter. this will also produce an early-season bias (for slightly different reasons than the same pattern seem on natural grass).

> With Astroturf it is simply a matter of the temperature getting colder; with natural grass it is the grass thickness changing. In temperate-warm climates on natural grass, there may actually be a change of grass type (from the higher-risk Bermuda to the lower-risk rye) over the course of the season as well. Unfortunately I haven't seen any data about temperature and

seasonal changes for the third-generation artificial surfaces but I don't have any reason to think the pattern would be different to Astroturf.

Hearing that new generation artificials in Norway (Figure 3) were of equivalent ACL risk to natural grass, I spent much of trip trying to observe and guess which types of natural grass they were using on sporting fields around Scandinavia. I was suspicious that the Norway studies might be comparing artificial surfaces at their best (in cold weather for most of the year) to natural grass not at its best (i.e. not ryegrass). My sample size was pretty small, was conducted in mid-summer only and my identification skills are not expert. However, I concluded that many sports fields in Norway probably use a hybrid natural grass surface (Figure 4; maybe fescues, Kentucky bluegrass, poa annua and a bit of ryegrass thrown in). This might make identifying grass types in Norway akin to identifying grape types in France – difficult because they mix it up so much. Maybe the researchers in Norway couldn't answer my question about grass type is because it is such a tough one! The result though was often a far bit of thatch (Figure 5) and potential for developing high traction. It may be quite likely that the climate in Norway is too cold to support rvegrass and there may be issues with increased shoe-surface traction once a natural surface freezes. It might be that. in Norway, artificial turf in the most sensible surface for a soccer ground.

What about a warmer climate, where the risk of playing on a third-generation artificial surface might be a lot higher? I haven't seen any soccer data from Africa, but I did hear at the conference. off the record, than the latest NFL data might be trending that way. I studied NFL ACL injuries in the Astroturf era and, whilst this surface was minimally riskier than natural grass, it was a fairly close comparison. If the NFL data is showing the disparity between third-generation artificials and natural grass is widening, it is a bit of a bombshell. I only hope that if these rumours are accurate that someone will come to a major sports medicine conference and present the findings (which didn't happen in Norway). As mentioned before, I hope that FIFA doesn't make the presumption that because artificial surfaces in Norway are safe that they also will be in Africa.

Table 1 - relative risk for ACL injuries on the major natural grass species

Grass species	Relative risk for ACL injuries	Preferred climate	Common distribution
Kentucky bluegrass (poa pratensis) – also known in UK as smooth-stalked meadow grass	Medium-High	Cool and dry	Northern USA, Canada, Continental Europe
Rye grass (Lolium perenne)	Low	Temperate and humid	United Kingdom, New Zealand, Japan, southern Australia & USA (winter)
Bermuda grass (cynodon dactylon) – also known in Australia as couch.	Medium-High	Warm	Northern Australia, Africa, Southern Asia, Southern Australia & USA (summer)

One of the non-reversible risk factors for ACL injury is previous ACL injury. In particular, ACL injury to the contralateral side is a known non-reversible risk. Reinjury to the ACL graft is also known but potentially modifiable and so is worth studying in more detail. For the nonserious athlete, the best way to reduce the risk of re-injury (both to graft, if used, and contralateral side) and subsequent arthritis is to retire from high risk sports. Therefore, many non-serious athletes may not need ACL grafts at all and, if they do require or elect to have ACL reconstruction, they are probably best advised to seek a surgical technique with minimum morbidity. The serious athlete who needs to keep playing is faced with the unfortunate reality that many ACL grafts don't hold up in difficult conditions. In the mid-1990s, particularly after David Schwarz suffered three ACL injuries on the same knee (one primary and two graft ruptures) within 12 months the belief in AFL medical circles was that perhaps the problem was that we were letting our players back too soon. However, sadly the rate of graft rupture in the AFL hasn't improved from the mid-1990s to today despite the average player missing closer to 10 months than the 6 months which was the standard of the 1990s. Even more sadly, ACL injuries are a common cause of career stagnation or regression for the 6% of players who have had one. At the end of the 2007 AFL season, the only two players ranked in Mike Sheahan's Top 50 who had previously had an ACL reconstruction were Cameron Mooney (no. 22) and Nick Malceski (no. 47). Perhaps at the end of 2008, Richardson, Didak, Hayes and Bradshaw (who

have all had knee reconstructions in the past) will break into Sheahan's Top 50. However, no player has ever won the Brownlow medal after having had an ACL reconstruction. In the NRL it looks slightly better if you create a 'Top 50' group of players from those who have played Origin or Test football in 2008. Of these, Justin Hodges, Brent Tate, Joel Monaghan and Ashley Harrison have come back from knee reconstructions. But in all codes there are many players who - even if they aren't in the 10% or so who re-rupture their graft - don't return to the same standard of play after an ACL reconstruction.

These fairly nasty statistics about outcomes would have been prominent in the discussions that the Swans had about the fairly radical option of a LARS (Ligament Augmentation and Reconstruction System) which they used for Nick Malceski. Even though he had ultimately had a relatively good outcome



Figure 4 - fairly thatchy hybrid grass from Tromsø in Northern **Norway**

from his patellar tendon traditional ACL reconstruction a few years earlier, he would have known first hand about the missed initial season and the slow return to good form in the season following. To date (at the time of writing) his LARS treatment has been a success, in that he is already playing games that he would have missed. We know that the older artificial ligaments from the 1980s generally led to bad results in ACL reconstructions, but a good article from The Age reminded use that Doug Hawkins was actually an isolated major success with an artificial ligament from this era 16. We certainly need more than the small number of 2008 cases to know whether the new generation artificial is going to be a major advance.

One lesson we can immediately take on board is that surgeons should look acutely for preservation of the native ACL if it has only been torn or avulsed (as opposed to completely ruptured)



Figure 5 – grass on a house in Western Norway with too much thatch?!

at the initial injury. It sounds like in Malceski's case of 2008 that the LARS ligament was used as a reinforcement of the intact part of his ACL. Another similar case that didn't get as much publicity was Daniel Giansiracusa of the Western Bulldogs, who injured his ACL in the 2008 preseason but apparently had no reconstruction at all as the injured ligament looked as though it may heal in a good position with conservative treatment. At the Roosters we had a case in 2006 where the player concerned tore all of ACL. PCL and MCL and was rightly considered a career-threatening injury at the time. However, the surgeon treating him (Merv Cross) made the call that ACL and PCL had been peeled off together at femoral end and the main body of both ligaments was still intact. He did a direct repair with no graft for all three ligaments and the player came back successfully at 7 months. As a measure of the success, he is now one of the 2008 State of Origin players listed above. Like the Swans and the Western Bulldogs, we took an option which had some degree of risk but also had the upside of not requiring the player to have any of his own tendons sacrificed. Alisa Camplin did something along the same lines using a donor (allograft) tendon for her reconstruction before the 2006 Winter Olympics. I am aware that some of the Sydney knee surgeons are getting parents to donate graft tendons to their athlete children for knee reconstructions. It would not be surprising to see professional footballers use either of these techniques in the near

With respect to the traditional autografts, in Australia at the community level the vast majority of patients (or their surgeons) are choosing hamstring grafts for the morbidity advantages mentioned earlier. Matthew Liptak has presented data from AFL reconstructions done in the 1990s suggesting that the hamstring grafts in general do better than the patellar tendon grafts ¹⁷, which supports this trend. However, for my elite players at the Roosters I still tend to recommend patella tendon grafts fixed with interference screws. I don't know whether it is the sport itself or other aspects of surgical technique, but our results have been hard to argue against from an observational viewpoint in terms of getting back to super-elite level. Justin Hodges had a patellar tendon graft when at the Roosters and would now be in the current top 10 players in the NRL competition. I don't know how much knee pain he still gets, but I do know that the most important thing a professional rugby league centre would want from a knee reco is to return to the highest possible level. Another one of our patella tendon reconstructions, Ryan Cross, now plays at the very top level of rugby union for the Wallabies, in a similar position.

When in Norway, I chatted to Oslo's top knee surgeon Lars Engebretsen about ACL reconstructions. They have a national database of ACL reconstructions, which leads the world and is something we should try to emulate in Australia ¹⁸. Ironically Lars doesn't use the LARS technique but prefers patellar grafts for high level athletes, hamstrings for low level and occasionally he'll do an allograft. He mentioned though that 80% of Norway surgeons use hamstring tendon allografts but for his elite athletes he still isn't convinced that they can get the players back to the same performance level as the patellar tendon grafts. Hans Mueller-Wolfhart at Bayern Munich, of Actovegin fame lately in Australia, sends his German elite players over to Colorado to get Richard Steadman to do patellar tendon autologous grafts for them. Dozens of elite English and European soccer players have been to Steadman to get their ACLs done, including Michael Owen and Ruud van Nistelrooy. When you travel, it seems that the patellar tendon autograft is still the world gold standard for super-elite athletes, even though hamstring autograft procedures are now undoubtedly the no. 1 procedure in the world for Joe Average.

Another very interesting thing I learnt in Norway was that Lars Engebretsen credits an ACL injury, to Susann Goksør Bjerkrheim, for the entire formation of the Oslo Sports Trauma Research Centre. Previous lobbying for the government and private sectors to support such a centre had failed, but Susann's ACL injury was the catalyst for the powers in Norway to change their minds. Susann was the top handball player in Norway and was thought to be the key to the national team winning a Gold medal at the Sydney Olympics. There was national mourning when she tore her

ACL in early 2000, as the entire country considered that the injury had cost them a certain Gold medal. Like the genesis of the AIS out of the grief of the terrible Australian Olympic performance at Montreal, the politicians decided that something needed to be done about ACL prevention and they funded the OSTRC.

My final anecdote from the many I have about the trip comes from the Leprosy museum we visited in Bergen. Leprosy has been virtually eradicated in the Western world and the Norwegians are the most responsible for this. In the 1850s, at the time that John Snow was working out the cause for cholera in London. Norway founded the first ever national medical register for the disease leprosy. Armauer Hansen gets most of the credit for conquering leprosy by discovering the bacteria responsible (actually predating Koch's discovery of the tubercle bacillis). Ove Høegh should get as much credit for creating the register, as it was from this that Hansen was in fact sure that leprosy was an infectious disease and that therefore he needed to find the agent. Expanding from this history it is easy to understand why the Scandinavians still lead the world in both medical registers and public contribution towards disease prevention.



Figure 6 – The Broad St pump in Soho, London, where John Snow determined the cause of cholera in the 1850s. About the same time the Norwegians were determining the cause of leprosy, bringing in the era of modern epidemiology and public health

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