For many individuals, low back pain (LBP) is predominantly a self-limiting symptom rather than a diagnosis. In athletes, life-time prevalence of LBP varies between 18%–65% with some sports more affected than others (Trompeter et al 2017). There are so-called “high-risk” sports in which participation is associated with higher rates of LBP compared to the age-matched general population. Athletes participating in activities such as rowing, cross-country skiing, dancing, fencing, gymnastics, and track and field events appear to be more commonly affected (Trompeter et al 2017).

The full list of sports with high prevalence of LBP (Trompeter et al 2017) is:

- rowing
- dancing
- fencing
- gymnastics
- underwater rugby
- water polo
- shooting
- basketball
- hockey
- ice hockey
- athletics
- figure skating.

Epidemiology

Unlike in general populations, where the majority of LBP has no identifiable pathoanatomical structural cause, aetiology of LBP in athletes is more likely to be associated with structural changes in the spine (Schroeder et al 2016). This is thought to be a result of athletes exposing their spine to high and repetitive loads over long periods of time. Subsequently, differential diagnoses, including serious pathology and specific injury, must be considered when assessing an athlete with LBP (Jakes et al 2015).

Unremitting LBP lasting longer than three to four weeks, particularly in a younger athlete (< 20 years), is considered a “red flag” and should be considered serious until proven otherwise. Therefore, although many athletes and support staff may consider LBP as “a sign training hard”, management of young athletes with LBP should include a thorough investigation to establish a diagnosis, with a “simple or non-specific” LBP to be considered as a diagnosis of exclusion only.

SERIOUS PATHOLOGY

LBP caused by a serious pathology is relatively rare (1%) but has to be considered in younger athletes in particular. Onset, duration and nature of LBP will help a clinician to differentiate non-specific LBP, i.e. unrelated to pathology, from a serious pathology including malignancy and tumours (e.g. ...
osteo-, osteoblastoma, bone cysts, osteogenic sarcoma), infection (osteomyelitis, discitis), inflammatory spondyloarthopathies (juvenile arthritis, ankylosing spondylitis, psoriatic arthritis), enthesitis or visceral pathology such as pyelonephritis (Jakes et al. 2015). Focused questioning to explore the existence of "red flags" is important and by definition, presence of any serious spinal pathology warrants a referral for further investigation (Jakes et al. 2015).

Features that indicate serious pathology, and should be seen as red flags:
- age <20 years, especially pre-pubertal
- sudden onset of severe back pain
- duration of >4 weeks
- thoracic spine pain
- night pain, or pain that wakes patient from sleep
- unrelenting pain, even when supine
- fever, chills and / or night sweats
- unexplained weight loss
- immunocompromise, e.g. HIV
- previous malignancy
- corticosteroid use
- recent trauma
- progressive neurological deficit
- bladder or bowel dysfunction
- saddle anaesthesia
- disturbed gait or limp, tripping and / or unexplained falls
- vertebral deformity.

**STRUCTURAL INJURIES OF THE SPINE**

Compared to the general population, athletes with LBP were found to have higher prevalence of structural pathology regardless of the sport they participate in (Schroeder et al. 2016). However, the patterns of injuries vary between adults and adolescent athletes. In young adults with LBP, 46% had radiological evidence of bony injuries such as pars interarticularis defects compared to 6% in older adults (Purcell & Micheli 2009). On the other hand, older adult athletes with LBP tend to have disc related injuries and these are seen in 48% of cases compared to 11% in adolescents (Purcell & Micheli 2009).

Consideration needs to be given to the relationship between spinal injury and LBP. Spinal injuries such as spondylolysis were found to be the most significant risk factor for LBP in NFL players (Iwamoto et al. 2004) and MRI evidence of lumbar degenerative disc disease increased risk of LBP in gymnasts (Koyama et al. 2013). There is other research, however, that demonstrates significant pathology in fully active individuals who are free of pain, for example, multilevel disc degeneration and pars stress lesions were found in asymptomatic and fully functioning cricket bowlers (Ranson et al. 2005). While this conflicting research triggered a debate on the role of screening for structural abnormalities in asymptomatic athletes, it is important to maintain that establishing accurate diagnosis, particularly in young athletes participating in "high-risk" sports, is critical. The additional clinical indicators of possibility of a structural injury to the spine are a sudden onset of focal LBP, cessation of training / competition and disturbed sleep (Kalpacioglu et al 2009).

**NON-SPECIFIC LOW BACK PAIN**

While athletes may be more susceptible to developing structural injuries of the spine, many will have symptoms that are benign and self-limiting. Importantly, just like in the general population, LBP in athletes can develop into a persistent pain disorder with associated loss of function driven by cognitive, lifestyle and behavioural factors rather than, often co-existing, structural changes in the spine. In such instances, the success of the management is dependent on a broader approach in the identification of dominant factors contributing to the disorder and a subsequent, individualised treatment pathway aimed at addressing the dominant pain drivers.

**Risk factors for LBP in athletes**

A multitude of risk factors are thought to be associated with LBP in athletic populations (table 1). The type of sport, for example, appears important when it comes to LBP in younger athletes. Biomechanical factors and muscle dysfunction have been associated with risk of LBP across the age groups (Nourbaksh & Arab 2002). From recent evidence, training load and years of exposure appear to be among the most significant risks for LBP in athletes (Wilson et al. 2020), as do non-modifiable factors such as age, skeletal maturity and a previous injury (Trompeter et al. 2017).

**TRAINING LOAD**

There is some evidence that excessive training volumes, periods of load increase and years of exposure are risk factors that contribute to the development of LBP (Wilson et al. 2020). Further, while many athletes will exhibit an acute response to training load, some athletes may develop persistent symptoms that last for months or years (Wilson et al. 2020). The role of training load in the development of LBP in athletes is complex and multifactorial, and requires a careful consideration of the athlete’s individual training history, current training load, and predisposing factors such as age, gender, and previous injury experience (Wilson et al. 2020).

**TABLE 1: Described risk factors for LBP in athletes**

<table>
<thead>
<tr>
<th><strong>FACTORS</strong></th>
<th><strong>MODIFIABLE</strong></th>
<th><strong>NON-MODIFIABLE</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INTRINSIC FACTORS</strong></td>
<td>Biomechanics of the spine, hip, shoulder</td>
<td>Gender (more common in females)</td>
</tr>
<tr>
<td></td>
<td>Muscle function (strength, neuromuscular control, endurance/capacity)</td>
<td>Age (young/mature athletes)</td>
</tr>
<tr>
<td></td>
<td>Psychological factors (mood, behaviours, cognitions)</td>
<td>Previous injury (+ return to play)</td>
</tr>
<tr>
<td><strong>EXTRINSIC FACTORS</strong></td>
<td>High training load</td>
<td>Skeletal maturity status (growth spurt)</td>
</tr>
<tr>
<td></td>
<td>Sport rules/regulations</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Coaching and training cultures</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Playing time</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Playing surface</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Equipment</td>
<td></td>
</tr>
</tbody>
</table>

Type of sport (high risk sports) Level of play (elite more affected) Playing position (bowlers, pitchers)
Training load monitoring has been proposed as a useful method to training load and concurrent fitness gain (Gabbett 2016). The “weekly” time window is an estimate which may differ depending on training schedule that can vary across different sports (Wang et al 2020).

TABLE 2: Strategies to monitor training load and considerations for its use

<table>
<thead>
<tr>
<th>STRATEGY</th>
<th>DESCRIPTION</th>
<th>CLINICAL CONSIDERATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avoid rapid changes in weekly training load (TL) (&gt;10% guideline)</td>
<td>Weekly increases in TL should not exceed 10% (Gabbett 2016).</td>
<td>The 10% guideline is a rough estimate that can be higher/ lower depending on the athlete’s level, i.e. novice/elite. For example, athletes with very high or very low chronic workload (CW) may not be able to tolerate even 10% weekly increase, whilst a seasoned athlete with moderate or high CW may be able to tolerate weekly increases greater than 10% (Gabbett 2016). The “weekly” time window is an estimate which may differ depending on training schedule that can vary across different sports (Wang et al 2020). Consideration also needs to be given to what unit of load is used and its measuring accuracy, e.g. rate of perceived exertion, or minutes, training or distance covered (Wang et al 2020).</td>
</tr>
<tr>
<td>Maintaining acute:chronic workload ratio low (ACWR)</td>
<td>ACWR should be kept between 0.8 – 1.3 (Gabbett 2016)</td>
<td>Some athletes may sustain injury when ACWR is lower than 0.8, others may tolerate ratios higher than 1.3. Given that the CW is defined as training average over four weeks with each week weighted equally, athletes with very different training patterns over that time may, in fact, have the same ACWR, i.e. the same perceived injury risk, even though their injury risk would likely differ depending on how they spread their load over the four weeks. Calculating ACWR using exponentially weighted moving averages may therefore be more sensitive measure (Wang et al 2020; Maupin et al 2020).</td>
</tr>
</tbody>
</table>

TABLE 3: Growth characteristics during the adolescent growth spurt for girls and boys (adapted from Birrer & Cataletto 2002)

<table>
<thead>
<tr>
<th>GROWTH CHARACTERISTICS</th>
<th>GIRLS</th>
<th>BOYS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at start</td>
<td>9-10 y</td>
<td>11-12 y</td>
</tr>
<tr>
<td>Age at maximum growth</td>
<td>12 y</td>
<td>14 y</td>
</tr>
<tr>
<td>Age at which growth slows</td>
<td>&gt;12 y</td>
<td>&gt;14 y</td>
</tr>
<tr>
<td>Age until growth continues</td>
<td>16-18 y</td>
<td>18-20 y</td>
</tr>
<tr>
<td>Age at maximum height growth (PHV)</td>
<td>11-13 y</td>
<td>13-15 y</td>
</tr>
<tr>
<td>Approx rate of growth during PHV</td>
<td>7-9 cm / year</td>
<td>8-10 m / year</td>
</tr>
</tbody>
</table>
Chronological age monitoring

Chronological age (CA) provides an estimate of growth during adolescence (table 3). An important indicator of skeletal maturity is the period of peak height velocity (PHV), which is a period of maximum growth during adolescence. Girls reach PHV at around age 11-13 years and boys between 13-15 years (Birrer & Cateletto 2002). While CA offers a quick and easy estimate of growth periods, only two-thirds of adolescents fall within “normal” age ranges for skeletal status, with as much as a five-year discrepancy (Birrer & Cateletto 2002).

Skeletal age monitoring

Annual screening techniques such as x-rays, ultrasound and magnetic resonance imaging (MRI) are accepted gold standard methods to establish skeletal maturity status (Bergeron et al 2015). Wrist and hand x-rays are most commonly used. Limitations include radiation exposures and the resources to cover the cost of annual screening. Nevertheless, this could be a highly effective and efficient risk management strategy for young athletes participating in sports associated with higher prevalence of spinal injury, e.g. gymnastics, rowing and cricket, to inform training load management on a case by case basis (Bergeron et al 2015).

Anthropometric screening

Anthropometric screening involves measuring, e.g. height, weight and leg length on a regular basis throughout the adolescent age (table 4). Athletics skills model (ASM) offers a digital growth calculating algorithm available at www.athleticsskillsmode.nl/en/growth-calculation. It is a quick and easy method to identify the onset of adolescent growth spurt from a set of basic variables, such as gender, date of birth, standing / sitting height, and weight. Providing the standardised measurement is followed, ASM is shown to be a valid and reliable estimate of growth (Mirwald et al 2002). Its one limitation is that its accuracy is dependent on access to the athlete’s measurements for a period of more than four years.

<table>
<thead>
<tr>
<th>METHOD</th>
<th>MONITORING FREQUENCY</th>
<th>ADVANTAGES</th>
<th>LIMITATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronological age</td>
<td>Annually</td>
<td>Easy method of monitoring requiring limited resources</td>
<td>Reliability is limited with 5 year+ discrepancy between individuals</td>
</tr>
<tr>
<td>Skeletal age (e.g. wrist, hand x-rays)</td>
<td>Monthly, 6-monthly or annually</td>
<td>Highly accurate not affected by puberty Gold standard of skeletal maturity</td>
<td>Invasive, potential exposure to radiation and associated costs</td>
</tr>
<tr>
<td>Anthropometric screening (e.g. ASM growth monitor)</td>
<td>Monthly</td>
<td>Considered useful, non-invasive method to help identify the onset of adolescent growth spurt</td>
<td>Series of data needs to be taken every 3-6 months for period of 4+ years, a period that may be difficult to achieve</td>
</tr>
</tbody>
</table>

TABLE 4: Summary of skeletal maturity status monitoring

BIOMECHANICS

How people with LBP move was observed to differ to those without LBP in a number of ways. These include smaller range and lower speed of lumbar motion, reduced proprioception and stiffer movement strategies (Laird et al 2014, 2019). It was unclear, however, whether these movement alterations precede the development of, or contribute to, the perpetuation of LBP. A systematic review of prospective studies showed that restricted lateral flexion and limited lumbar lordosis predicted the development of LBP in general populations (Sadler et al 2017). The picture is less clear in athletes. In cricket, for example, coupling of lateral flexion and axial rotation, also called the “crunch factor”, was implicated in the development of contralateral spine injuries in cricket fast bowlers (Glazier 2010). More recent, prospective and retrospective evaluation found no differences in biomechanical measures of those senior and junior cricketers with and without history of LBP, or in those who did and did not go on to develop LBP (Senington et al 2020).

There is some evidence to suggest that load-sharing between neighbouring anatomical regions may be important. Senington et al (2020) observed that cricket fast bowlers with no history of LBP had four times greater thoracic rotation during the back foot impact, serving as a “wind-up” mechanism to generate pace on the ball, when compared to those with history of LBP. Golf, squash and tennis are other examples of sports where players with LBP demonstrated significantly restricted range of motion (ROM) at the hip, and L-R hip ROM asymmetries compared to their pain free counterparts (Van Dillen et al 2008). Interestingly, a recent study of in-line hockey players suggests a cut-off point with hip external and total rotation ROM of less than 56.5 and 93 degrees, respectively, to predispose players to developing LBP (Cejudo et al 2020).

<table>
<thead>
<tr>
<th>BODY AREA</th>
<th>BIOMECHANICAL RISK</th>
<th>ASSESSMENT METHOD</th>
<th>TESTED POPULATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spine</td>
<td>Restricted lumbar lateral flexion (Laird et al 2014; Sadler et al 2017)</td>
<td>Tape measure assessment of the difference between middle finger position on ipsilateral thigh to most distal position of middle finger achieved in max lateral flexion</td>
<td>General public</td>
</tr>
<tr>
<td>Hip</td>
<td>Restricted internal rotation (IR) (Sadeghisani et al 2015)</td>
<td>Passive hip rotation in prone using inclinometer or goniometer</td>
<td>In-line hockey Rowing Hockey Golf Squash and tennis</td>
</tr>
</tbody>
</table>

TABLE 5: Clinical assessment tests screening for potential risk of LBP in selected sports
Mitigation of biomechanical risks

Biomechanical screening to assess LBP risk is frequently established on laboratory based movement analysis systems (Elliott 2000; Vad et al 2004), rendering them of limited clinical use. A summary of clinical assessment tests indicating a biomechanical risk for LBP in athletes in selected sports is summarised in table 5.

When it comes to biomechanical modifications, there appears to be insufficient evidence for these to successfully manage LBP in athletes (Thornton et al 2020). There is some low-level sport-specific evidence such as addressing hip asymmetries leading to a reduction of LBP in golfers (Reinhardt 2013), and moving rowers from end-range flexion in catch phase that coincided with a reduction in their LBP (Ng et al 2015). Coaching interventions in cricket also showed some promise, demonstrating that bowlers with mixed bowling action can be successfully moved towards a safer bowling technique, resulting in reduced incidence or progression of their lumbar disc degeneration compared to those who continued to use the mixed bowling action (Elliott & Khangure 2002). While these studies indicate that coaching modifications towards safer technique are possible, the Elliot & Khangure (2002) research took three years of intensive coaching input to produce their results. Furthermore, the impact of such interventions on the clinical outcomes, such as level of function or training time lost to LBP, has not yet been studied.

Muscle function

Impaired muscle function of the lumbo-pelvic-hip complex seems to be a hallmark of LBP (Nourbakhsh & Arab 2002). This appears important both in athletes and non-athletes with LBP demonstrating similar levels of trunk extensor deconditioning compared to pain free controls (Moreno Catalá et al 2018). The role of muscle function as a predisposing risk factor for LBP is, however, less clear. From sport-specific literature, tennis players with LBP had lower abdominal endurance and less co-contraction compared to matched pain free controls (Correia et al 2016). Also in tennis players, those with erector spinae neuromuscular imbalance were more likely to develop LBP, while completing back extensor programmes proportionately reduced their symptoms (Renkawitz et al 2006). Elite golfers with a side bridge endurance score of less than 12.5 seconds had increased risk of LBP (Evans et al 2005), and cricketers with LBP undergoing neuromuscular training were shown to reverse their impairments in neuromuscular control of transversus abdominis and multifidus, and this coincided with a reduction in their LBP (Morton et al 2014).

Importantly, the role of muscle function in predicting LBP appears to vary depending on the age of the athlete. While reduction in trunk muscle strength was predictive of LBP in adult athletes (Noll et al 2016), this was not the case in adolescent athletes whose trunk flexion and extension peak torque didn’t discriminate between those with and without LBP (Mueller et al 2017). This is likely to be a reflection of the multifactorial nature of LBP in athletes where factors such as training load may pose a greater risk for LBP than does their muscle strength.

Mitigation of risks related to muscle function

Optimal muscle strength and neuromuscular control is considered critical in compensating for external forces placed on the spine in athletic populations. Trunk muscle function screening is therefore a frequently clinically utilised risk management strategy. The Sorensen test (figure 1) is suggested as a useful proxy of trunk muscle endurance with good reliability, reproducibility and discriminative validity between athletes with and without LBP (Evans et al 2007). An important consideration is that this test was developed to measure muscle endurance in a single movement plane and thus may not be sensitive to detect unilateral or multiplanar deficiencies.

Functional movement screen (FMS) also demonstrated some utility in identifying athletes at risk of LBP. A study on collegiate female rowers found that those scoring ≤16 on FMS with a shorter plank test hold time (mean time 109.5 seconds) had a 1.4 times greater risk of developing LBP (Gonzalez et al 2018). While this study is promising in FMS utility to screen for LBP risk, the size of the risk was relatively small and

FIGURE 1: The Sorensen test: The Roman chair variant typically used in sport (image reproduced with permission)
was shown only in females, so further research is required in broader athletic populous.

**Psychosocial factors**
Psychosocial factors including low mood, anxiety, distress and depression have long been considered strong predictors of LBP in the general population. Low mood, psychological complaints and catastrophising were also found to be among the factors associated with significant injury in elite dancers (Cahalan et al. 2014). Noll et al. (2016) studied athletes from Brazil and found that “feeling lonely” and loss of sleep were among the highest contributors to LBP in the range of demographic, socioeconomic, hereditary, exercise-level, anthropometric, strength, behavioural and postural factors.

Mitigation of psychosocial risk
The diagnostic uncertainty and often long-term impact of LBP can become career limiting for athletes, causing distress and anxiety that may impact on their recovery. Early screening of psychosocial risk factors was therefore recommended as a means of preventing chronicity in athletes (Wippert et al. 2017a).

The Örebro Musculoskeletal Pain Screenings Questionnaire (OMPSQ) (Boersma & Linton 2005) and the STarT Back Tool (Hill et al. 2016) are two examples of how the risk of LBP chronicity is assessed in the general population. However, these tools have not been validated in athletes as it is argued that they operate in a different “pain” context (Wippert et al. 2017a).

Alternative tools have therefore been developed for athletes (Wippert et al. 2017b, 2020). These include the Risk Stratification Index (RSI), which gives an estimate of the risk of LBP chronicity in the athlete, and the Risk Prevention Index (RPI) that offers personalised recommendation for management (table 6). However, while the RPI demonstrates clinical benefit in the general population (Wippert et al. 2020), its effect in athletic populations is yet to be determined. Nevertheless, both tools outperform the OMPSQ in demonstrating excellent transferability, sensitivity, specificity and discriminative validity (Wippert et al. 2017a), and these are the first validated tools offering a promise to assess LBP chronicity risk in athletes.

**Summary and conclusions**
The impact and associated burden of LBP in athletes is comparable, if not excessive, to that seen in the general population. The nature of LBP in athletes, however, is different. In young athletes, LBP is atypical and, as such, needs to be considered serious until proven otherwise. Athletes also have higher rates of structural injuries of the spine, although the relationship with LBP is unclear. The potential impact of spinal injury and LBP on longer term health outcomes and on a sporting career is yet to be determined. The aim of this article is to summarise the evidence of potential risk factors for, as well as management of, LBP and spine injury in athletes to guide clinicians in helping to maximise the spinal health, and ensure long and thriving sporting careers of their athletes.

For many athlete patients, not being able to participate in their chosen sport is highly likely to result in a very poor functional pain score (FPS) on our Physio First Data for Impact (DfI) tool. Dealing with these cases correctly and enabling the safe return to sport will, therefore, promote a dramatic improvement in the FPS score for this patient population.

**About the author**
Dr Liba Sheeran is Reader in Physiotherapy and Population Health Research Theme Lead at Cardiff University. She is also a Consultant Physiotherapist for Welsh Athletics working with elite track and field athletes. Her interest is in enhancing health through physical activity and exercise with focus on musculoskeletal disorders (MSDs). Liba’s research track record is in the development and implementation of active physical interventions using health technologies. Her research involves exploring utility of wearable sensors, video-tracking and artificial intelligence (AI) models for assessment and personalised exercise management of MSDs. She speaks nationally and internationally and runs expert workshops in management of complex low back pain.

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**TABLE 6: Overview of chronic LBP screening tools suitable for athletic population**

<table>
<thead>
<tr>
<th>TOOL</th>
<th>ITEMS</th>
<th>DOMAIN</th>
<th>PREDICTION</th>
<th>TIME</th>
<th>TARGET GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk Stratification Index (RSI)</td>
<td>8-17</td>
<td>Biopsychosocial (e.g. fear avoidance, catastrophising, depression, lifestyle, work situation, financial incentives, exercise status)</td>
<td>Predictor of LBP chronicity</td>
<td>6-12 months</td>
<td>General population, athletes</td>
</tr>
<tr>
<td>Risk Prevention Index (RPI)</td>
<td>3-16</td>
<td>Biopsychosocial (e.g. fear avoidance, catastrophising, depression, lifestyle, work situation, financial incentives)</td>
<td>Identification of risk profile groups: (pain experience; social environment; stress; medical environment)</td>
<td>6-12 months</td>
<td>General population, athletes</td>
</tr>
</tbody>
</table>
References
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Halseon SL. Monitoring training load to understand fatigue in athletes. Sports Medicine 2014;44(2):139-147


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