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MSc Sports and Exercise Medicine

Lumbar spine intervertebral disc pathology, pathomechanics and adaptability, considerations for the athlete: A narrative review of in vivo studies

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Word count: 10,704

July 2023

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Abbreviations

ADC - Apparent diffusion coefficients values

LIVD – Lumbar intervertebral disc

LIVDs – Lumbar intervertebral discs

MRI – Magnetic Resonance Imaging

ADC - Apparent diffusion coefficients values

Abstract

[Motivation] Mechanical loading is integral to the LIVDs homeostasis; load can create catabolic effects and macroscopic injury and conversely, anabolic effects and positive macroscopic adaptions to the LIVD. In athletic populations, there is evidence that physical activity can both positively and negatively influence the properties of LIVDs in young and elite adult athletes. Understanding how exercise effects the LIVD in vivo will help to formulate load management strategies for athletes and the general population for injury prevention and rehabilitation. **[Research Focus]** The focus of this dissertation is to review studies that focus on the effects of exercise and sport on the LIVD in vivo with specific considerations to athletic populations. **[Research Methods]** An extensive literature search across multiple search engines was performed, relevant literature to the research focus was retrieved and reviewed. **[Findings]** The findings from this review suggest that sport type is associated with differing morphological LIVD outcomes. LIVD degeneration is associated with same and adjacent level changes in lumbar spine biomechanics. Acutely, the LIVD is affected by both exercise stress and unloading postures. **[Conclusion]** Chronic long distance running and cycling is associated with better LIVD health, in contrast sports that place high load with larger and faster ranges of motion are associated with a higher prevalence of LIVD degeneration. **[Recommendation]** This review recommends an interventional study investigating the effect of long term slow long distance running or cycling coupled with a post exercise LIVD unloading protocol to investigate the effect on LIVD health.

Key words: Lumbar, intervertebral disc, exercise, in vivo

1. Background

Lumbar intervertebral discs (LIVDs) are composed of two parts: the nucleous pulposus (NP), which is enclosed by the annulus fibrosus (AF), superior and inferior to the LIVD lie the cartilaginous end plates (CEP) (Desmoulin et al., 2019). In a healthy state, the NP is rich in proteoglycans that maintain hydrostatic pressure (Desmoulin et al., 2019). This hydrostatic pressure creates intradiscal pressure, which tensions the AF creating uniform pressure on the CEP (Vergroesen et al., 2015). The AF is a fibrous structure comprising 15-25 obliquely alternating concentric lamella composed of collagen fibres embedded with proteoglycans (Desmoulin et al., 2019).

Mechanical loading is integral to the LIVDs homeostatic mechanisms (Neidlinger-Wilke et al., 2014). The cells of the AF and NP are known to respond to tension, compression, hydrostatic pressure and osmotic stimuli (Fearing et al., 2018), a process termed mechanobiology (Vergroesen et al., 2015). LIVD extracellular matrix health is maintained through mechanobiology; in response to loading, especially hydrostatic pressure, nuclear chondrocytes are stimulated to produce proteoglycans and type II collagen (Belavý et al., 2016).

LIVDs are dynamic tissues; spontaneous reabsorption of LIVD herniation has been reported widely in the literature (Yu et al., 2022). They are responsive to acute loading, Yanagisawa et al. (2021b) via MRI demonstrated that deadlifts performed at 6 repetitions (at 70% 1 repetition max) 70%, for 5 sets, significantly reduced apparent diffusion coefficients values (ADC) (an index of water movement) of the NP, demonstrating a loss of water from the NP. Further,

prolonged overloading can cause LIVD cellular degeneration and damage to the extracellular matrix, reducing hydrostatic pressure (Belavý et al., 2016). Several studies have investigated the effect of exercise with pre and post intervention LIVD MRIs. Studies have reported no observable changes to the LIVDs (Owen et al., 2020; Khanzadeh, Mahdavinejad, and Borhani, 2020), whilst other researchers found evidence of positive changes on MRI (Jeong et al., 2017; Lee and Cho 2016).

A review summarised the types of LIVD failure in Table 1 (Desmoulin et al., 2019. P.458) and the biomechanical aetiology of LIVD tissue failure in Table 2 (Desmoulin et al., 2019, p. 460).

Table 1. summary of LIVD failure types, reproduced from Desmoulin et al., (2019. P.458).

TABLE 1. Summary of Failure Types	
Failure Type	**Description**
Annulus tears	A rupture or tear of the annulus fibrosus.
Disc prolapse	Involves a tear in the annulus fibrosus which allows the nucleus pulposus to bulge out beyond the damaged outer rings.
Endplate damage	Damage to the vertebral interface with the disc.
Schmorl nodes	An upward or downward protrusion of the disc soft tissue into an adjacent vertebral body.
Internal disc disruption	Biochemical loss of integrity of the annulus fibrosus generally focused on the inner rings.
Disc narrowing	A thinning of the disc, reducing the distance between vertebral bodies.
Radial disc bulging	Involves the disc moving off center from the vertebral body, effectively being pushed out to the side.
Vertebral osteophytes	Small bony growths that develop near the edges of the endplates

The findings in table 2 are based on in vivo animal studies (Solomonow et al., 1999), animal cadaveric spines (Callaghan and McGill, 2001; Marshall and McGill, 2010; Paul et al., 2017; Veres, Robertson and Broom, 2010; Wade et al., 2016) and human cadaver lumbar spines (Adams and Hutton, 1981, 1982 and 1985; Brinckmann, 1986; Farfan et al., 1970). Daly et al., (2016) highlighted the difference across animal species of the LIVD size, geometry and how human

LIVDs are significantly exposed to vertical forces and bipedal gait versus animals who are exposed to a quadrupedal gait. The viscoelastic properties of LIVDs demonstrate a time-dependent response dictated by fluid loss during loading, gain during unloading, and total content, affecting NP pressure, mechanics and failure rates (Schmidt et al., 2016). In vitro LIVD models, when compared to in vivo, exhibit incomplete fluid recovery during unloading cycles, demonstrating discrepancies in fluid flow (Schmidt et al., 2016). Neuromuscular response to load has a significant effect on tissue stress, and this response is absent from in vitro studies (An and Masuda, 2006). As previously discussed, LIVDs response to load is characterised by catabolic and anabolic processes and subsequent positive or negative adaptions (Fearing et al., 2018), a process which does not occur in cadaveric tissues. Thus in vitro models may have limited external validity to the living human LIVDs response to loading.

Table 2 LIVD failure and associated mechanical load, reproduced from Desmoulin et al., (2019, p. 460).

TABLE 2. Structural Failures Associated and Their Link to Mechanical Loadings

Loading Type	Possible Resulting Failure
Compression	Annulus tear disc prolapse Endplate damage Disc bulging
Flexion	Disc narrowing
Axial rotation	Annulus tear
Complex loading	Annulus tear Disc prolapse

Injury to LIVDs can cause a loss of intradiscal pressure leading to reduced AF tension, disc height and biomechanical sequelae of an enlarged neutral zone in

bending, shear, torsion and increased stress concentrations (Vergroesen et al., 2015). Breen et al., (2020) investigated the effect of early to moderate LIVD degeneration on intervertebral motion in asymptomatic adults. Using weight bearing and recumbent MRI and quantitative fluoroscopy, the researchers found that disc height loss correlated negatively with lumbar flexion during weight bearing, but this did not reach significance. However, Lee, Daffner, and Wang, (2014) reported that LIVD degeneration in weight-bearing MRI was significantly associated with reduced lumbar spine range of motion compared to controls.

In athletic populations, there is evidence that sports can positively influence the properties of LIVDs (Owen et al., 2021). Owen et al., (2021) found that soccer and basketball players compared to age and height matched sedentary controls, demonstrated greater LIVD hydrated and hypertrophy on MRI. A prospective study by Frenken et al., (2022) conducted MRI studies using gagCEST saturation transfer, a surrogate measure of LIVD glycosaminoglycan content (GAG) of elite rowers during peak preseason preparation and six months later during the post season recovery. Compared to non-athletic controls, the MRI during peak pre-season demonstrated significantly higher CAG content compared to controls. However, the post season MRI did not reveal any significant differences in GAG content between the rowers and controls. Further, there was a significant difference in the rowers gagCEST between pre-season training and post season recovery. Frenken et al., (2022) concluded that the increased gagCEST values were transiently associated with elite-level rowing training and demonstrated the ability of LIVDs respond to training. Positive LIVD adaptations of increased

hydration and proteoglycan content have also been observed in running populations (Belavý, et al., 2017).

To the contrary, sport has also been associated with increased MRI signs of LIVD degeneration compared to non-athletes in both young (Witwit et al., 2018) and adult (Abdalkader et al., 2020) elite athletes. Owen et al., (2021) found a weak association between greater training loads and career duration with worse LIVD hydration. In adult weightlifters, Hung et al., (2014) found that the risk of LIVD degeneration increased with cumulative loading; there was a dose-response relationship associated with higher loading and elevated odds of LIVD degeneration on MRI. However, higher training volume in runners was associated with positive LIVD adaptations (Belavý, et al., 2017).

In summary load can create catabolic effects and macroscopic injury, and conversely, anabolic effects and positive macroscopic adaptions to the LIVD. Narrative reviews on the response of the LIVD to load have been published, however their conclusions and recommendations are primarily based on in vitro studies (Desmoulin et al., 2019; Neidlinger-Wilke et al., 2014). Due to the previously mentioned limitations of in vitro model external validity, a review of exclusively in vivo studies is being proposed. The proposed study may arrive at different conclusions to in vitro studies, thus affecting recommendations and future research related to LIVD loading in athletic populations. To the author's knowledge, there are no narrative reviews of exclusively in vivo studies on this topic.

1.1 Research Aim

This research will aim to improve understanding of how load can affect pathology and positive adaption to the LIVD in athletic populations.

1.2 Objectives

1. Critically appraise studies that investigate the effect of chronic load on the LIVD in vivo, in athletic populations.

2. Review the effects of LIVD degeneration and injury on lumbar spine biomechanics in vivo.

3. Objective 3 Investigate research on LIVD load management.

1.3 Research Methods

The literature search will be conducted on the following electronic Databases - Pubmed, University of South Wales Findit, and Google Scholar. Multiple databases were searched to increase the chance of finding all relevant literature (Bramer et al., 2018). The following keyword searches were used on each database: lumbar AND intervertebral discs AND in vivo; lumbar AND intervertebral discs AND MRI; lumbar and intervertebral disc and exercise; lumbar and intervertebral disc and athlete. The search was performed in the “title and abstract” function for Pubmed, in “any field” for University of South Wales Findit, and “allintitle” “insubject” for “Google Scholar”. The initial results were checked for known relevant literature, some known literature did not appear in the search, the search was subsequently adjusted, and search terms were expanded to included “exercise” and “athlete” (Bramer et al., 2018). Hand searches of the retrieved literature were conducted. To reduce publication bias,

grey literature was searched as it is more likely to consist of data with null or negative results (Paez, 2017). A searchable grey literature source is Google Scholar; therefore, it was included in the search strategy (Haddaway et al., 2015). Studies that investigate the effect of load on the LIVD in vivo and biomechanical sequelae were included. Due to the limited word count and time constraints of a student dissertation, only the following sports were included as they represent a diverse range of LIVD loading, running cycling, weightlifting and cricket.

1.3.1 Inclusion criteria

All study types

English Language studies

Human Lumbar Spine Intervertebral Discs

In vivo studies

Published between and inclusive of the years 1960 – 2023

1.3.2 Exclusion criteria

Non-English language studies

Non-human LIVDs

In vitro studies

Non freely available studies

Publishes before 1960

Published after March 2023

2. The effect of chronic load on the LIVD in vivo, in athletic populations

This section will critically appraise studies that investigate the effect of chronic sport and exercise on LIVD health. The following sports have been chosen due to relevant published research, their popularity, and the contrasting loads they place on the LIVD.

2.1 Running

During running the lumbar spine experiences cyclic axial loading (Mitchell et al., 2020), lateral flexion, rotation, flexion, extension, and compared to walking, L3/L4, L4/L5 and L5/S1 motions are larger than L1/L2 and L2/3 (MacWilliams et al., 2014).

To investigate the effect of running on LIVD health, Belavý et al., (2017) used MRI to measure LIVD composition in three groups with differing physical loading histories. A non-sporting group: no regular exercise or sport for the preceding 5 years, jogger group: 20-40km running per week for the preceding 5+ years, and long-distance runners: 50+km running per week for the preceding 5+ years, no other exercise, more than once per week except resistance exercise. Participants age was limited to 25-35 years to minimise the impact of normal aging on the findings. MRI methodology was standardised to reduce confounding variables; MRIs were conducted after midday; participants were required to sit for 20+ minutes prior and were instructed to not exercise on the day of the scan.

Compared to non-sporting controls, LIVD T2-times were significantly higher (+11.4%) in long-distance runners and joggers (+9.2%), this affect was present at

all LIVD levels (Belavý et al., 2017). Running was associated with greatest T2-times in the central NP region of long-distance runners +15%, and joggers +11% compared to non-sporting controls (P<0.01) as demonstrated by figure 1 (Belavý et al., 2017, p.4).

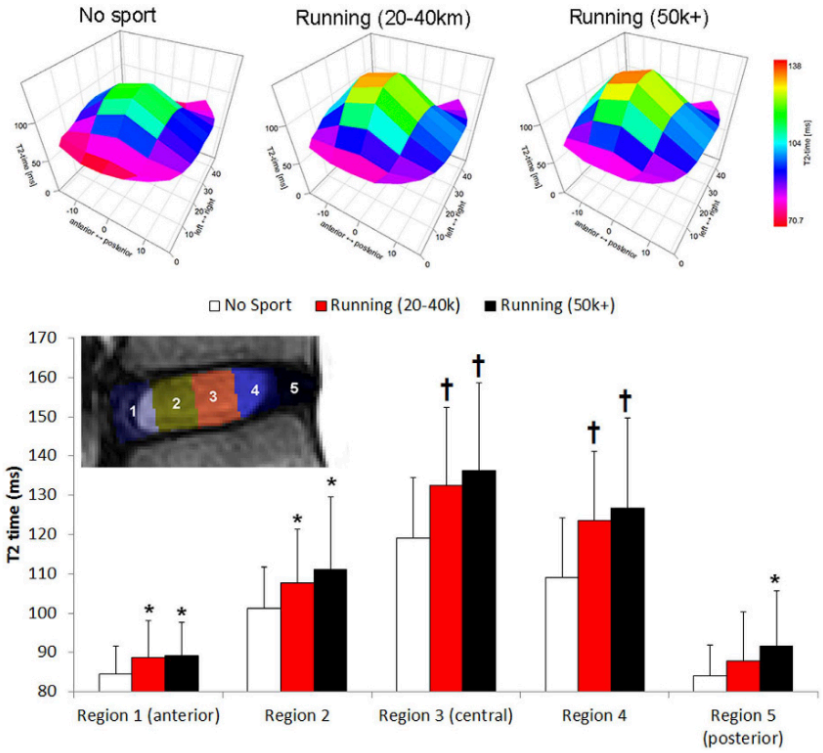

Figure 1. 3D box plot and graph representing T2-times of the non-sporting controls, joggers and long-distance runners. Reproduced from (Belavý et al., 2017, p.4). The 3D plot displays the mean T2-times of the LIVDs in the 3 different groups, the bottom of the figure demonstrates the significantly greater T2-times of LIVD regions 3 and 4 (P<0.01) and regions 1, 2 and 5 (P<0.05).

Higher LIVD T2-times are indicative of increased LIVD hydration and glycosaminoglycan content and therefore improved LIVD composition (Marinelli et al., 2009). The relative height of the LIVD compared to vertebral body height was also greatest in the long-distance runners. This suggests a hypertrophic response and positive adaption to chronic running.

Similar to Belavý et al., (2017), Mitchell et al., (2020) performed a study to assess LIVD health in male middle-aged long-term runners (n=9, age 48 years (mean)) compared to a control group (n=8, age 50 years (mean)). The runner's activity levels were greater than Belavý et al., (2017): running history of 10-39 years, 23 years (mean); 56-129km per week, 82.6km per week (mean), 4-6 days per week, 6 days per week (mean); 3.9-4.8 minutes per km, 4.3km minutes per km (mean). Mitchell et al., (2020) found that the LIVD of runners tended to be healthier than controls. The runners Pfirrmann score was averagely half a grade lower than the control group; grade 1.9 (mean) vs 2.5 (mean) respectively. Further L5/S1 Pfirrmann scores were significantly 1 grade lower than controls. Controls had greater vertebral body height than the runners, however runners LIVD height was 20% greater (P=0.002). Average total LIVD height to vertebral body height ratio was significantly greater in the running group compared to controls (seven percentage points) (P=0.001), this was also seen at individual LIVD levels: L2-L3 (36%), L3-L4 (25%) and L5/S1 (24%) compared to controls. ADC were also higher in runners; however this did not approach significance (p=0.566). Significant associations between lower Pfirrmann grades with greater total running years (P=0.006) and total weekly running distance (P=0.034) were found. Pfirrmann grading criterium is detailed in table 3 (Pfirrmann et al., 2001, p.1874). Greater weekly running distance was associated with longer LIVD anterior annulus T2-times (P=0.021) as demonstrated in table 4 (Mitchell et al., 2020, p.7). Further, runners had longer total LIVD T2-times than the controls, with the total mean time being 91.3ms versus 86.8ms respectively, however this

did not approach significance. Comparatively, Belavý et al., (2017) did find that runners LIVD T2 times were significantly greater than controls.

Table 3. Pfirrmann LIVD degeneration grading criterium, reproduced from (Pfirrmann et al., 2001, p.1875)

Grade	Structure	Distinction of Nucleus and Anulus	Signal Intensity	Height of Intervertebral Disc
I	Homogeneous, bright white	Clear	Hyperintense, isointense to cerebrospinal fluid	Normal
II	Inhomogeneous with or without horizontal bands	Clear	Hyperintense, isointense to cerebrospinal fluid	Normal
III	Inhomogeneous, gray	Unclear	Intermediate	Normal to slightly decreased
IV	Inhomogeneous, gray to black	Lost	Intermediate to hypointense	Normal to moderately decreased
V	Inhomogeneous, black	Lost	Hypointense	Collapsed disc space

Table 4. Summary of findings reproduced from Mitchell et al., (2020, p.7).

	Primary analysis			Secondary analysis		
	Runners (n = 9)	**Control (n = 8)**	**P-value**	**Runners (n = 9)**	**Control (n = 8)**	**P-value**
Vertebral body height, mm	26.8 (1.0)	28.0 (1.0)	**0.043**	26.8 (1.4)	28.0 (1.5)	**0.016**
IVD height, mm	8.3 (0.7)	6.8 (0.7)	**0.002**	8.3 (1.2)	6.8 (1.0)	**<0.001**
IVD-vertebral body height ratio	0.31 (0.04)	0.24 (0.02)	**0.001**	0.31 (0.05)	0.24 (0.03)	**<0.001**
IVD T2-time, ms						
Total	91.3 (11.2)	86.8 (23.9)	0.649	91.3 (14.9)	86.8 (24.5)	0.610
Anterior annulus	73.2 (6.1)	75.7 (19.2)	0.731	73.2 (7.3)	75.7 (18.2)	0.702
Anterior nucleus	89.1 (12.6)	84.3 (22.2)	0.625	89.1 (16.6)	84.3 (23.9)	0.584
Centre nucleus	101.7 (13.3)	92.5 (24.9)	0.396	101.7 (20.5)	92.5 (27.7)	0.336
Posterior nucleus	96.8 (14.6)	89.1 (25.1)	0.491	96.8 (19.5)	89.1 (27.1)	0.439
Posterior annulus	78.4 (5.8)	78.1 (19.3)	0.970	78.4 (7.2)	78.1 (18.7)	0.966
Apparent diffusion coefficient, mm^2/s	249.0 (175.2)	202.3 (149.5)	0.566	-	-	-
Pfirrmann, grade (1–5)	1.9 (0.2)	2.5 (0.7)	**0.036**	1.9 (0.6)	2.5 (0.9)	**0.015**

All data are mean (standard deviation). Primary analyses were independent t-tests using the average of lumbar levels (L2-S1). Secondary analyses were linear mixed models with random effects for within participant variance across all lumbar levels (L2-S1). Apparent diffusion coefficient was measured at L5-S1 only.

There are several factors which may explain the difference in results. Mitchell et al., (2020) running group ran 82.6 (27.9) (mean (SD) km per week versus 28.0(6.7) (mean (SD)) km per week in the jogging group and 66.6(19.5) (mean(SD) km per week in the long distance running groups for Belavý et al., (2017). Although the long-distance running group did show better LIVD properties when compared to the jogging group, Belavý et al., (2017) did not find a statistically significant difference. This may indicate that an upper celling effect of running volume was being approached (Belavý et al., 2017), which may have

also been exceeded by Mitchell et al., (2020) running group. There is also evidence that the running speed of the Mitchell et al., (2020) might have been a confounding factor. Belavý et al., (2017) included objective accelerometry of their cohort and found that total physical activity levels were not associated with LIVD characteristics, however, LIVD NP T2-times were strongly associated with accelerations in the 0.44 and 0.59 *g* mean amplitude deviations (MAD) (range) suggesting a likely “anabolic window”. Ambulation at 2 m/s falls inside this range, whereas walking at <1.5 m/s fell below and running >2.5 m/s and jumping were above this range (Belavy et al., 2017) Mitchell et al., (2020) 4.3(0.4) minutes per kilometre which equates to 13.95 km per hour or 3.88 meters per second, which by Belavý et al., (2017) data is >0.86 MAD and above the likely “anabolic window”. The Participants in Belavý et al., (2017) groups were 29.3, 30.2 and 30.1 years, compared to 48 and 50 years in (Mitchell et al., 2020). Wang et al., (2014) found that LIVD T2-times significantly reduce with age in the general population as demonstrated by figure 2 (Wang et al., 2014, p.261), further Wu et al., (2013) found a significant negative correlation of LIVD T2-times to age and a significant difference between <45 and >45 years of age.

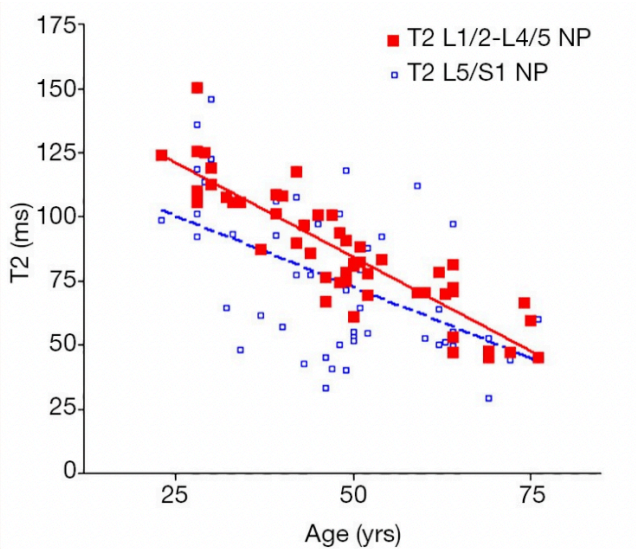

Figure 2. Relationship between age and LIVD NP T2-times, reproduced from Wang et al., (2014, p.261).

Running has also been associated with positive LIVD health in university athletes. Hangai et al., (2009) performed a cross sectional MRI study of university athletes (n=308, age=19.5 years (mean)) from 6 sports: Baseball (n=57), Swimming (n=47), Basketball (n=63), Kendo (n=51), Soccer (n=47), Runners (n=43), and a Non-athlete control group (n=71, age=19.5 years (mean)) to investigate LIVD degeneration. Hangai et al., (2009) used midsagittal T2-weighted images and graded LIVDS as being degenerated if they were scored 3≥ using Pfirrmann classification. The running group had a lower prevalence of LIVD degeneration than all other athletes and non-athletes, however this did not approach significance. Utilising the non-athlete control group as a reference, logistic regression showed lower rates of LIVD degeneration in runners (OR, 0.75; CI, 0.31-1.73 however this finding was not significant (p=0.5075). Further, LIVD health also appears to be better in retired elite runners when compared to retired athletes from other sports. Videman et al., (1995) conducted a retrospective cohort study of former Finnish elite athletes, a subgroup of which

were long distance runners (n=24, age = 59 years (mean)). Compared to soccer players (n=26, age=55.3 years (mean)), weightlifters (n=19, 57.5 years (mean)), and shooters (n=25, age=59.8 (mean)), runners and shooters had the lowest LIVD degeneration scores. Compared to weightlifters, runners LIVD signal intensity) was 16% (mean) better at L1-S1 (P=0.024) and weightlifters disc bulge score was 34% worse than runners (p=<0.001). All Finnish men complete compulsory military service therefore this is a consistent variable amongst the included participants and does not jeopardise internal validity, but due to the unique physical demands of military service, external validity should be considered when inferring these findings to wider populations.

In summary, running is associated with higher T2-times indicating higher glycosaminoglycan composition and hydration (Belavý et al., 2017; Mitchell et al., 2020), LIVD hypertrophy indicated by higher LIVD height to vertebral body height ratio (Belavý et al., 2017; Mitchell et al., 2020), lower Pfirrmann grade compared to controls (Mitchell et al., 2020), lower prevalence of LIVD degeneration compared to athletes from other sports and non-athlete controls (Hangai et al., 2009) and amongst former elite athletes (Videman et al.,1995).

2.2 Weightlifting

Weightlifting places high shear, axial compression, flexion, and extension forces on the lumbar spine (Eltoukhy et al., 2016).

To investigate the effect of Olympic weightlifting on the lumbar spine Shimozaki et al., (2018) conducted a three-year prospective cohort study of 12 (6 female) child and adolescent Olympic weightlifters. At baseline MRI the age of the

participants was 11.4 ± 2.0 years (mean). The athletes performed Olympic weightlifting 2 hours per day, 5 days per week for an average of 500 hours per year. The Pfirrmann classification was used to grade LIVDs, at baseline MRI assessment of the 12 weightlifters, 2 (17%) demonstrated LIVD degeneration, and no signs of herniation. The year 2 MRI revealed 8 (67%) weightlifters displayed LIVD degeneration and no cases of herniation. The year 3 MRI found 9 (75%) weightlifters displayed LIVD degeneration, and 2 (17%) weightlifters showed LIVD protrusion or extrusion.

Shimozaki et al., (2018) suggest that the prevalence of LIVD degeneration is high for the age group and references Tertti et al., (1991) who found a prevalence of LIVD degeneration of 26% in 15-year-olds without low back pain and 38% in those with low back pain. The findings may have been impacted by the small sample size, however competitive Olympic weightlifters in this age group do not comprise of large numbers to select from (Shimozaki et al., 2018).

In an older population, Vadalà et al., (2014) analysed LIVD health and the role of MRI T1ρ in assessing for early degenerative changes in asymptomatic male weightlifters (n=13, 25.3 years mean age, range 18-29) compared to age and sex matched healthy controls (n=13, 23.5 years mean age, range 21-25 years). The weightlifter's activity levels: weight training 3-4 times per week, for 17.7 ± 1.2 months, 1 hour of strength and cardiovascular exercise and 1 hour of skill training per day, all weightlifters were able to lift 1.5 times body weight, the authors do not elaborate on weightlifting technique used. Sedentary controls met inclusion criteria if they did not currently or historically compete in organised

sport. T1 and T2-weighted MRI found no significant morphological LIVD differences or Pfirrmann classification differences between groups; no significant abnormalities were found in either group. T1 maps however, demonstrated significantly lower ($p<0.05$) NP T1ρ values at L2-S1 suggesting lower NP proteoglycan content in the weightlifting group compared to controls, indicating early stages of LIVD degeneration. In both groups, a significant decreasing trend in T1 ρ values ($p<0001$) from L1/L2 to L5/S1 was seen.

Vadalà et al., (2014) highlight that the T1ρ values were widely heterogeneity distributed especially for Pfirrmann grade 1, non-degenerated LIVDs (65.3 to 172.5 milliseconds), the distribution was narrower for Pfirrmann grade 2 (65.3 to 172.5 milliseconds), and even narrower for grade 3 (59.08 to 83.62 milliseconds). This finding is supported by Zobel et al., (2012) who observed that non-degenerated LIVDs (Inclusive of both Pfirrmann grades 1 and 2) demonstrated a wide range of T1 ρ relaxation time. This demonstrates that Pfirrmann classification has low sensitivity to detect early LIVD changes (Vadalà et al., 2014) and therefore T1 ρ weighted MRI images might have higher sensitivity to detect early LIVD degenerative changes when compared to the T2 weighted Pfirrmann classification (Zobel et al., 2012).

In a retrospective cohort study of former Finnish elite athletes weightlifters (n=19, 57.5 years (mean), 45-65 years (range)) mean LIVD bulging scores were 34% higher than runners (n=24, age = 59 years (mean), 50-67 years (range)), 35% higher than shooters (n=25, age=59.8 (mean), 50-68 years (range)), and 4% higher than soccer players (n=26, age=55.3 years (mean), 45-64 years (range)

(P< 0.001 between groups). LIVD T2 weighted signal intensity of the NP of weightlifters was 34% worse than soccer players at L1-L4 IVDs, and 6% worse at L5/S1 IVD.

Videman et al., (1997) investigated LIVD degeneration in male monozygotic twins with contrasting exercise histories. Twins (n=12 pairs, 46.5years (mean) 35-62 years (range)) with discordant weightlifting histories were included. The exercise history for the weightlifting was 2,300 hours (mean) during 13.8 years (mean), compared to their twins 200 hours (mean) during 1.1 years (mean). No significant differences in LIVD signal intensity, height or bulging were seen between the twins with discordant weightlifting histories.

Videman et al., (1997) may not have seen a significant difference in LIVD health between groups due to the amount of exposure to weightlifting. The exercise history for the weightlifters was 2,300 hours (mean) over 13.8 years (mean), 2,300 hours divided by 13.8 years equates to 166.7 hours per year, divided by 52 weeks equals an approximate mean of 3.2 hours per week of weight training. In comparison, Shimozaki et al., (2018) cohort of weightlifters were training for 10 hours per week and an average of 500 hours per year. The athletes in Videman et al., (1997) may have been training in a more optimal range for LIVD health.

Weightlifting is associated with increased LIVD degeneration in adolescent weightlifters (Shimozaki et al., 2018), male weightlifters aged 18 to 32 years (Vadalà et al., 2014), and former elite athletes (Videman et al., 1997).

2.3 Cycling

The lumbar spine biomechanics of cycling is typified by a long duration flexed posture (Streisfeld et al., 2017), with cyclic axial rotation (Burnett et al., 2004).

Belavý et al., (2019) compared 18 high-volume cyclists (age = 29.3 years (mean), 10 females, >150km per week, for 5+ years) with 18 age and height matched nonsporting control group (age = 29.9 years (mean), 9 females, <150 minutes of moderate physical activity per week and walking commute <15 minutes). The high-volume cyclists cycled for 267±100km per week, 11.9±3.2 hours per week, and had cycled for 9.2±5.0 years. Belavý et al., (2019) methodology helped to reduce confounding variables known to influence LIVDs, prior to MRI which was performed after midday, participants were instructed to not exercise and asked to sit for >20 minutes prior to scan. Age was limited to 25-35 years (range) and participants were excluded if they had current pain and a history of spine trauma or smoking. LIVD Pfirrmann grade was calculated utilizing T2-weighted images. High-volume cyclists were found to have significantly greater LIVD height (P = <0.01) and relative LIVD height to vertebral body height (P=<0.05) than nonsporting controls, as demonstrated in figure 3 (Belavý et al., 2019, p.215). The high-volume cyclists showed a 10.5 ±18.3ms longer average LIVD T2-time compared to non-sporting controls (P=0.021), the effect was most prominent in the NP compared to the AF and in upper LIVDs, L1/ L2 P=0.011 and L2/L3 (P=).029), no significant differences were seen at L3-S1 IVDs. Pfirrmann grades were not significantly different between groups.

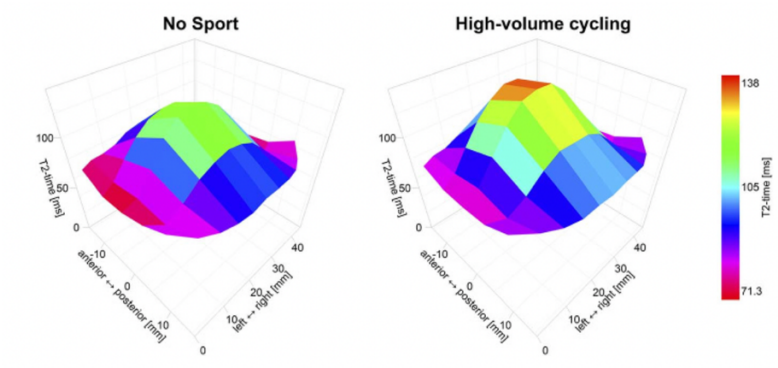

Figure 3. “Three-dimensional representation of the effect of cycling LIVD T2 time, reproduced from Belavý et al., (2019, p.215).

Greater LIVD height and Longer T2-times indicated better hydration and glycosaminoglycan content thus indicating better quality LIVD tissue composition in the high-volume cyclists compared to controls (Belavý et al., 2019).

2.4 Cricket Fast Bowling

The biomechanics of cricket fast bowling involves lumbar spine rotation, lateral flexion, extension, flexion, and axial loading (Senington et al., 2020).

Crewe et al., (2012) performed an MRI study to analyse lumber spine pathologies in 46 district and state level cricket fast bowlers. The cricketers mean age was 16.1, range = 13-18, and they were sub-grouped by age: under-15 (n=14), under-17 (n=18) and under-19 (n=14). Typically, the cricketers bowled 90-200 balls per week, over 3-4 days per week. LIVD degeneration by age group: under 15 age group – 29% 1≥ degenerated LIVD, under-17 age group - 33% 1≥ degenerated LIVD, and under-19 age group - 43% 1≥ degenerated LIVD. Of the 16 participants with LIVD degeneration, 12 displayed disc bulges and 2 displayed an annular fissure (Crewe et al., 2012). Hardcastle et al., (1992) performed an MRI analysis of young cricket fast bowlers (m=24, age = 16-18 years (range)), and found a higher LIVD degeneration prevalence of 63%, a

similar prevalence to Ranson et al., *2005). Ranson et al., (2005) performed a lumbar spine MRI study of male professional cricket fast bowlers (n=36, age=26 years (mean)) and age and sex matched active controls (n=17, age=25 years (mean)). The cricketers bowled on average >3 times per week and the active controls engaged in a range of different physical activities and sports for approximately three 2-hour sessions per week. Compared to controls, the professional fast bowlers had a high prevalence of multi-level LIVD degeneration. LIVD abnormalities in at least one level was seen in 22 (61%) cricket fast bowlers, and 9 (52%) active controls. Pfirrmann Grades IV and V (severe LIVD degeneration) were found in 12 (33%) cricket fast bowlers, compared to 2 (12%) of active controls.

Cricket fast bowling technique appears to be a modifiable risk factor in LIVD degeneration. In a group of 24 cricket fast bowlers aged 13.7 years (mean), Elliott et al., (1993) observed 21% (n=5) of bowlers displayed LIVD degeneration or bulging. Kinematic analysis of the cricketers bowling technique revealed that LIVD pathology was associated mixed action bowling technique and the use of trunk rotation by a mean of 30 degrees were more like to display LIVD degeneration or herniation on MRI (Elliot et al., 1993).

Burnett et al., (1996) performed a longitudinal study investigate thoracolumbar (T12-S1) IVD degeneration and bowling technique in 19 male cricket fast bowlers aged 13.6 years (mean) who represented school and club level. The participants received an MRI of at the beginning of the 1991-92 cricket season and a second MRI 2.7 years later at the end of the end 1993/4 season. After baseline

assessment the researchers provided an educational clinic to the fast bowlers which taught them how to reduce mixed bowling and promoted a front-on or side-on bowling technique. Between the two scans there was a significant increase in the occurrence of LIVD degeneration from 5 (21%) subjects at baseline, to 15 (58%) subjects with degenerated LIVDs at follow up (age 16.3 years (mean)). A biomechanical assessment found a small reduction in mixed bowling technique from 15 mixed bowlers at baseline versus 12 mixed bowlers at follow up. Fast bowlers who used mixed bowling technique at baseline and follow up were more likely to show progression of intervertebral disc degeneration compared to front-on and side-on technique (P=0.015). Compared to a side-on or a front-on bowling technique, the mixed technique demands more lumbar axial rotation, flexion, extension, and lateral flexion (Burnett et al., 1996).

Based on mixed bowling technique being a risk factor for LIVD pathology, Elliott and Khangure, (2002) performed a 3-year intervention study to investigate the effect of an educational intervention to reduce mixed bowling technique and LIVD degeneration prevalence in 2 groups of Western Australian Cricket Association fast bowling development squads. Group 1 – 1997, 14 males, age 13.4 years (mean) at baseline, attended 3 out of 4 yearly testing sessions. Group 2 – 1998, 12, age = 13.2 years at baseline, attended 2 or 3 out of 3 yearly testing sessions The Groups were pooled for analysis. The educational coaching intervention was conducted 6 times per year, the fast bowlers were encouraged to adopt either a side-on or front-on rather than a mixed action technique, emphasising a reduction in counter rotation of the spine. The level of transverse plane shoulder

counter rotation alignment significantly reduced from 35.4° mean to 21.3° mean (F=8.3505; P=0.004) between years 1 and 4. Of the bowlers who bowled with a mixed action, 27% displayed LIVD degeneration in year 1, compared to 12.5% of the bowlers who used front-on or side-on technique. Further, progression of LIVD degeneration was only seen in the bowlers who used mixed action technique.

At baseline Elliott and Khangure, (2002) cohorts age (13.2 years (mean) and LIVD degeneration prevalence (24.4%) was similar to Burnett et al., (1996) age (13.6 years (mean)) and LIVD degeneration prevalence (21%). Over a 2.7-year period in Burnett et al., (1996) cohort, transverse plane shoulder counter-rotation technique did not decrease and LIVD degeneration prevalence increased to 58%. In contrast, the fast bowlers in Elliott and Khangure, (2002) reduced mixed bowling technique from 80.5% to 33% and LIVD degeneration only reached 33% at age 16.5 years. Interestingly the prevalence of LIVD degeneration increased at similar rates from baseline to year 1 of the intervention in both studies, this may demonstrate that the intervention needs to be longer than 1 year (Elliott and Khangure, 2002). Comparison of results can be seen in Figure 4 (Elliott and Khangure, 2002, p.1717).

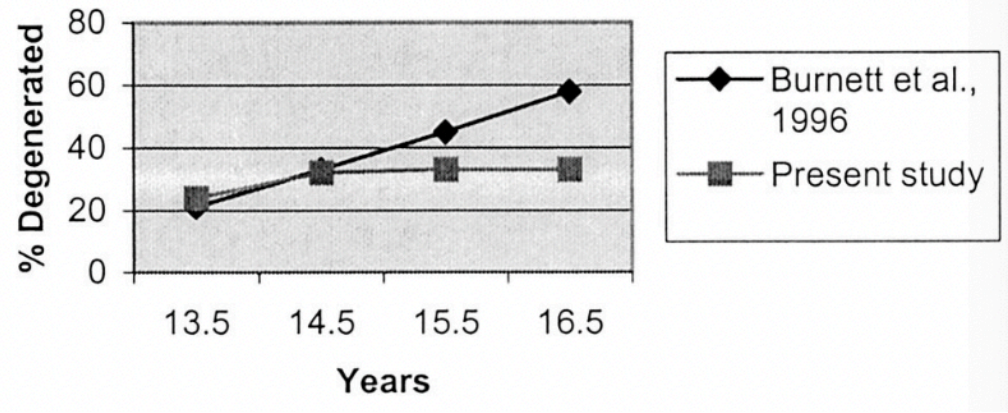

Figure 4 – Comparison of LIVD degeneration data between Burnett et al., (1996) and Elliott and Khangure (2002), reproduced from Elliott and Khangure, (2002, p.1717).

As can be observed in table 5, Crewe et al., (2012) reported lower LIVD degeneration in the Under 19s cohort compared to Hardcastle et al., (1992), this might be due to a difference in grading criteria. Hardcastle et al., (1992) provide minimal detail in their methodology, in comparison Crewe et al., (2012) used Pfirrmann score to grade LIVD degeneration.

The prevalence of LIVD degeneration in non-athletic asymptomatic adolescents is reported as 20%–33% (Kjaer et al., 2005; Tertti at al., 1991; Gibson et al., 1986), and 37% in asymptomatic adults aged 20-29 (Brinjikji et al., 2015), which increases with age as demonstrated by Table 6 (Brinjikji et al., 2015, p.813). Therefore, the cricket fast bowlers from Ranson et al., 2005; Crewe et al., (2012) Burnett et al., (1996) Hardcastle et al., (1992) demonstrate a high prevalence of LIVD degeneration compared to the general population.

Table 5. Collation of data of included studies on LIVD degeneration in cricket fast bowlers.

Table 5. Summary of LIVD Degeneration in Cricket fast bowlers		
Age	% of Participants with LIVD Degeneration	Study
13.3 years (mean)	24.4	Burnett et al., (1996)
13.6 years (mean)	21	Elliott and Khangure, (2002)
13.7 years (mean)	21	Elliott et al., (1993)
Under 15s	29	Crewe et al., (2012)
16.3 years (mean)	58	Burnett et al., (1996)
16.5 years (mean)	33*	Elliott and Khangure, (2002)
Under 17s	33	Crewe et al., (2012)
17.9 years (mean)	63	Hardcastle et al., (1992)
Under 19s	43	Crewe et al., (2012)
26 years (mean)	61	(Ranson et al., 2005)

*Post intervention

Table 6. Prevalence by age group of LIVD pathology in asymptomatic Brinjikji et al., (2015, p.813).

	Age (yr)						
Imaging Finding	**20**	**30**	**40**	**50**	**60**	**70**	**80**
Disk degeneration	37%	52%	68%	80%	88%	93%	96%
Disk signal loss	17%	33%	54%	73%	86%	94%	97%
Disk height loss	24%	34%	45%	56%	67%	76%	84%
Disk bulge	30%	40%	50%	60%	69%	77%	84%
Disk protrusion	29%	31%	33%	36%	38%	40%	43%
Annular fissure	19%	20%	22%	23%	25%	27%	29%
Facet degeneration	4%	9%	18%	32%	50%	69%	83%
Spondylolisthesis	3%	5%	8%	14%	23%	35%	50%

[a] Prevalence rates estimated with a generalized linear mixed-effects model for the age-specific prevalence estimate (binomial outcome) clustering on study and adjusting for the midpoint of each reported age interval of the study.

In conclusion, Cricket fast bowling is associated with increased prevalence of LIVD degeneration in both adolescent and adults at the professional level.

3. The effects of LIVD degeneration and injury on lumbar spine biomechanics in vivo

3.1 Effect of Posture on LIVD Herniation

Using positional MRI Nordberg et al., (2021) investigated the effects of 3 different positions on LIVD herniation in 37 patients (age = 36.7±11.9 years) with sciatica or radiculopathy. The 3 positions included were: supine conventional (relative lumbar flexion with pillow under knees), supine with lumbar extension (with lumbar pillow) and standing neutral. LIVD degeneration was graded using Pfirrmann scale and LIVD Herniation was categorised as central (central canal zone) or paracentral (subarticular zone) and extrusion or protrusion. The included patients mean Pfirrmann grade was IV (SD±0.9), with 43 LIVD herniations (44.2% central, 55.8% Paracentral). Paracentral LIVD herniations were observed to create higher grades of nerve root compression in standing (31.2% nerve roots) compared to supine conventional position (6.3%) ($p=0.005$). In standing compared to the conventional supine position, a significant difference in LIVD herniation cross sectional area mean difference (0.48 cm^2, 95% CI: 0.03 to 0.93) ($p=0.033$) and LIVD herniation diameter mean difference (1.10 mm, 95% CI: 0.38 to 1.82) ($p=0.001$) were observed. In contrast, significant differences were not observed when comparing other postures. Nordberg et al., (2021) suggests their findings indicate that increased size of LIVD herniation in standing is due to the increased core muscle activation and axial load from gravity rather than any increase lumbar extension associated with standing.

Changes in upright posture have also been associated with changes in LIVD biomechanics. Using kinematic MRI Zou et al., (2009) investigated the effect postural change on the bulging of degenerated LIVDs in back pain patients

(n=513, female = 298, age = 42.6 years (mean), range = 19-74 years). The subjects received MRI in axially loaded upright flexion, neutral and extension. Zou et al., (2009) observed that LIVD bulging increased with severity of LIVD degeneration as graded using Pfirrmann classification; grade II (mildly degenerated LIVDs) demonstrated less bulging than grade III and IV LIVDs ($P<0.05$). Grade I LIVDs at all levels, moved in a posterior direction with flexion and anterior direction with extension. However, this was not homogenous at other LIVD degeneration grades where the LIVD moved less predictably. Grade II-V (mild to severe) degenerated LIVDs were found to behave differently in response to changes in posture. At L1/L2, L2/L3, L3/L4 and L4/L5 Grade II LIVDs moved posteriorly with flexion and extension at L5/S1. Grade III LIVDs, during flexion moved posteriorly at L1/L2 and L3/L4, and during extension at L2/L3. Grade IV LIVDS during flexion moved posteriorly at L4/L5, during extension moved anteriorly at L3/L4, and posteriorly at L5/S1. Grade V LIVDs at L2/L3 moved posteriorly with flexion.

In agreement with Zou et al., (2009), Hu et al., (2011) found that increasing LIVD degeneration was associated with increased migration of LIVD bulging when compared to non-degenerated LIVDs ($p=0.001$). Hu et al., (2011) conducted a positional MRI study in 329 low back pain patients (age: 43.5years (mean) 18-80 years (range)) to investigate the effects of upright flexion, extension and neutral on the LIVD, unlike Zou et al, (2009) the subjects were not axially loaded.

LIVD degeneration has also been associated with changes in lumbar spine segmental biomechanics. Hu et al., (2011) in a positional MRI study investigated

the relationship between LIVD degeneration and motion segment angular motion (the difference between extended and flexed positions in degrees). Hu et al., (2011) observed that as LIVD degeneration increased, segmental angular motion decreased compared to non-degenerated LIVDs. Hu et al., (2011) developed their own LIVD degeneration grading system, as displayed in Table 7 (Hu et al., 2011, P.46). Grade 2 LIVDs tended to have less angular ROM than Grade 1 LIVDS, but this did not approach significance, grade 3 LIVDs however had significantly less ROM than grade 1 LIVDS ($p<0.001$). Segmental translation however was found to increase with increasing LIVD degeneration, with grade 3 LIVDs demonstrating significantly more translation than grade 1 LIVDs ($p<0.001$).

Table 7. LIVD degeneration grading system, reproduced from Hu et al., (2011, P.46).

Grades	Nucleus Signal Intensity	Disc Height	Structure of FSU
1	Hyperintense	Normal	Without disc herniation
2	Intermediate/hypointense	Normal/slight decrease	With/without disc herniation
3	Hypointense	Decreased/collapsed	With disc herniation/osteophyte

FSU, functional spinal unit.

Kong et al., (2009) performed a kinetic MRI study in upright flexion, neutral and extension of 316 symptomatic low back pain patients (100 male, age =42.1 years (mean), 16-85 years (range). The researchers observed that with increasing Pfirrmann grades I-IV segmental translation motion increased at L3/L4, L4/L5, L5/S1 and L1-L5 grouped ($P<.05$). Grade V LIVDs however did not follow this pattern, at L3/L4, L4/L5 and L1-L5 grouped, less segmental translation was observed, however this did not approach significance, and at L5/S1 there was similar translational range of motion in grade IV and grade V LIVDs. This is

different from Hu et al., (2011) who observed increasing segmental translation when the lumbar spine segments were grouped (total from L1-S1) with increasing LIVD degeneration, this may be due to the differing grading scales; grade 3 in Hu et al., (2011) is similar to grade IV and V in the Pfirrmann scales used in Kong et al., (2009). Angular motion was decreased in grade V LIVDs at L3/L4, L4/L5 and L1-L5 grouped compared to all other LIVD grades ($P<0.05$), this was also seen at L5/S1 but did not approach significance (Kong et al., 2009). This finding agreed with Hu et al., (2011).

Utilising flexion-extension radiographs and MRI, Galbusera et al., (2021) investigated the association between LIVD degeneration and lumbar spine range of motion in 602 patients. Galbusera et al., (2021) found that LIVD degeneration was consistently associated with reductions in flexion-extension segmental range of motion. Using Pfirrmann classification, significant difference in mean ROM were observed between grade I (8.2°) and grade II (6.9°) ($p=0.006$), no significant differences were seen between grades II (6.9°) and III (7.0), whereas grades IV (5.6°) and V (3.7°) did show significant mean reductions in ROM, as demonstrated by figure 5 (Galbusera et al., 2021, p.1111). Further, with progressive LIVD degeneration the centre of rotation of the intervertebral segment became increasingly dispersed. Galbusera et al., (2021) references non in vivo studies that indicated changes in the centre of rotation influence facet joint forces and subsequent progression of degeneration. A CT and histological study support this finding, Cui et al., (2019) performed a study on low back pain patients (n=29, woman = 14, age = 65 years (mean), 41-84 years (range)) who

had failed with conservative management and were undergoing posterior lumbar interbody fusion. Cui et al., (2019) found that LIVD degeneration was associated with facet joint tropism.

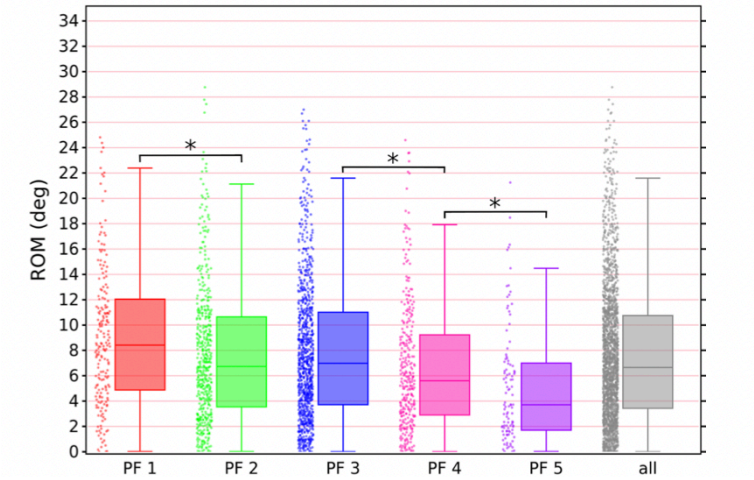

Figure 5. LIVD Degeneration score and associated segmental range of motion (PF= Pfirrmann Score), reproduced from Galbusera et al., (2021, p.1111).

In further support of Galbusera et al., (2021), a dual fluoroscopic imaging study of upright weightbearing functional tasks demonstrated that LIVD degeneration of L4/L5 and L5/S1 is associated with altered facet joint range of motion at the same and adjacent level of the degeneration (Li et al., 2011). At the same level, lateral flexion, flexion, and extension in participants with LIVD degeneration, facet joint range of motion was increased compared to controls. Further, the motion was not increased around the corresponding primary axis of rotation but increased in coupled rotations. For example, during flexion and extension rotation primarily occurs about the mediolateral axis; in patients with LIVD degeneration, increased motion during flexion and extension was seen in the anteroposterior axis, whilst rotation about the mediolateral axis did not increase. At the adjacent level, rotation about the primary axis was decreased whilst

coupled motion was increased. Li et al., (2011) suggests the observed hypermobility may increase facet joint compressive forces and articular cartilage degeneration.

Wang at al., (2011) also observed that LIVD degeneration in lower lumbar spine was associated with changes in L2-L4 biomechanics. Using combined dual fluoroscopic imaging and MRI, Wang et al., (2011) investigated the effect of LIVD degeneration and discogenic low back pain between L4 to S1 on L2 to L4 in vivo, during maximal lumbar spine rotation, lateral flexion, flexion, and extension motion. Wang et al., (2011) included 10 patients (51.8±13.1 years (mean)) with LIVD degeneration and 8 age, height and weight matched healthy controls (54.4±3.5 years (mean)). LIVD degeneration was graded using Pfirrmann classification. Wang et al., (2011) observed that in patients with LIVD degeneration at L4-S1, during full range lateral flexion, rotation, flexion, and extension demonstrated larger shear (approximately 25-40% higher) and tensile deformations (up to 23%) at L3/L4 and L2/3 compared to healthy controls. Wang et al., (2011) defined areas of minimal deformation as LIVD areas that experience less than 5% deformation during end ranges of spinal motion. A lower percentage of the LIVD area that experiences less than 5% deformation suggests less mechanical resistance to tensile (tension and compression) and shear forces. In the LIVD degeneration group, at L3/L4 and L2/L3 there was significantly less areas of minimal disc deformation compared to controls ($P<0.05$), except with left rotation. Therefore, there is an association of L4-S1 LIVD degeneration with increased LIVD tensile and shear deformation at L3/L4

and L2/L3. As the study was retrospective it is unknown whether L4-S1 LIVD degeneration was causative of higher deformations at L2-L4, or vice versa, higher lumbar spine LIVD deformation caused the LIVD degeneration and discogenic pain at L4-S1.

Contrary to Li et al., (2022) Lee et al., (2015) and Wang et al., (2011) did not find compensatory increased range of motion at lumbar spine motion segments adjacent to a degenerated LIVD. This contradictory finding may be due to Lee et al., (2015) using static observation via MRI to assess range of motion, whereas Galbusera et al., (2021) used dynamic dual fluoroscopy which allowed for the dynamic observation of increased range of motion about a secondary axis of rotation. Increased motion was observed at L1/L2 in all subjects with L4/L5 or L5/S1 grade 5 LIVD degeneration. However, significant reductions in whole lumbar motion were observed in patients with Grade V LIVD with <5 degrees of angular motion at L4/L5 or L5/S1 LIVD indicating that the lumbar spine was not able to fully compensate to make up for the loss of motion.

Degenerated LIVDs have also been associated with altered biomechanical responses during functional activities. Coppock et al., (2023) investigated the relationship between T1rho relaxation times and in vivo LIVD deformation from a treadmill walking stress test in 18 asymptomatic subjects. T1rho relaxation times were used to assess LIVD composition due to its sensitivity to detect proteoglycan and water content. Coppock et al., (2023) measured LIVD deformation from exercise stress tests in vivo by analysing the percentage change in LIVD height with pre and post exercise MRI. LIVD degeneration was

assessed using Sampling Perfection with Application optimised Contrasts using difference flip angle Evolution MRI and graded using a modified eight-point Pfirrmann scale (Coppock et al., 2023). Subjects were rested for 45 minutes in supine prior to MRI to minimise diurnal LIVD height changes, the subjects then preformed a 30-minute treadmill walk at a constant speed which was normalised to the participants dominant leg length, this resulted in average speed of 1.5 m/s and a range of 3.1-3.7mph. In degenerated LIVDs (Pfirrmann grades: III-VIII) NP T1rho relaxation times were significantly lower compared to non-degenerated LIVDs (Pfirrmann grades I-II) ($P<0.0001$). The deformation across all LIVDs was -6±4.0% (mean) and there was an inverse relationship between LIVD deformation and LIVD NP T1rho relaxation times. When LIVD level and subject BMI were controlled for, increased compressive deformation of the LIVD was significantly associated with lower NP T1rho relaxation times ($P=0.005$). Therefore, a relationship between LIVD composition as reflected by T1rho relaxation times and LIVD mechanical deformation from treadmill walking was observed (Coppock et al., 2023).

3.2 LIVD Degeneration and Intradiscal Pressure

An in vivo study found that LIVD degeneration is associated with significantly reduced LIVD intradiscal pressure compared to normal LIVDs (Sato, Kikuchi, and Yonezawa, (1999) as demonstrated in Figure 6 (Sato, Kikuchi, and Yonezawa, 1999, p. 2472).

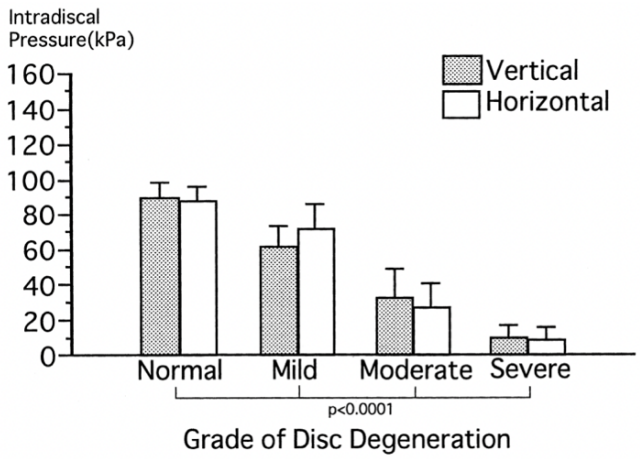

Figure 6. LIVD intradiscal pressure (measured vertically and horizontally) and progression of LIVD degeneration grade in prone posture, reproduced from Sato, Kikuchi, and Yonezawa, (1999, p. 2472).

3.3 LIVD Degeneration and Axial Strain

Meadows et al., (2023) performed an in vivo MRI based measurement of LIVD mechanics in supine (AM and PM for diurnal variation), flexed, and extended lumbar spine positions. The researchers used vertebral motion and LIVD deformation to quantify LIVD axial strain. The study population was healthy with no history of low back pain (n=16, 8=females, age= 25.±2.6 years (mean) 23-31 years (range). In the flexed MRI LIVDs with lower NP T2 times were significantly correlated with higher axial strain in posterior or anterior regions, wedge angle change and anterior to posterior shear displacement ($p<0.05$), however in the extended posture MRI NP T2 times were not significantly correlated ($p>0.49$) with the aforementioned characteristics.

Johansson et al., (2022) performed a supine lumbar spine MRI analysis of 36 subjects (18 females, 24 low back pain patients, 12 healthy controls, age = 38 years (mean), 25-69 years (range)), comparing the effects of non-loaded and

axial-loaded on the LIVD. Backward elimination analysis of LIVD deformation under axial load with LIVD characteristics of Pfirrmann grade, disc height, tilt and disc angle found that higher Pfirrmann grades were correlated with higher LIVD compression deformation.

3.4 LIVD Degeneration and the Intervertebral foramen

LIVD degeneration is associated with changes in intervertebral foramen biomechanics. Occult foraminal stenosis is a term coined by Splendiani et al., (2014) to describe a dynamic intervertebral neural foraminal stenosis that is not observed on normal supine MRI but is present on an upright weight bearing MRI. In a study of 160 patients with mono-radicular symptoms (age=56 (average), 40-65 years (range)), Splendiani et al., (2014) found no cases of occult foraminal stenosis in non-degenerated LIVDs. However, 61 cases were observed exclusively on the upright weightbearing MRI and in all cases LIVD degeneration was present at the same level.

LIVD degeneration has also been shown to affect the intervertebral foramen during changes in upright posture. In an MRI and fluoroscopy biomechanical assessment of low back pain patients with L4-S1 LIVD degeneration and lower extremity radicular symptoms and a control group. Cha et al., (2017) investigated the effect of LIVD degeneration on the Intervertebral foramina during standing flexion and extension. Compared to controls LIVD degeneration was associated with significantly smaller intervertebral neural foramina (>30%) in supine, upright flexed and extended postures at both the involved and the adjacent L3/L4

segment (>30%) ($p<0.05$). Cha et al., (2017) suggest that the phenomena of the adjacent segment reductions in intervertebral neural foramina is unexplained.

4. LIVD load management

4.1 Acute effect of exercise

Yanagisawa et al., (2021a) investigated the effect of half and parallel depth barbell back squats on the LIVD. The subjects performed 40 repetitions (8 repetitions, 5 sets, at 80% 1 repetition maximum with 90 seconds rest). Water movement as indicated by ADC values decreased significantly post parallel squats at L4/L5 and L5/S1 ($p<0.1$), this was not observed post half squats. Further, the decreases in L5/S1 ADC were significantly greater at L5/S1 in the parallel compared to the half squat ($p<.05$). The authors found that significantly higher degrees of lumbar flexion were associated with the bottom position of the parallel squat compared to the half squat ($p<.01$). Increased lumbar flexion imparts more compressive force on the LIVD, the researchers propose this as a reason for the greater reduction in ADC.

Similar findings have been observed from deadlifting. Yanagisawa et al., (2021b) investigated the effect of deadlifting on LIVD ADC with MRI. The subjects performed 30 deadlifts (5 sets of 6 repetitions at 70% of 1 repetition maximum with 90 seconds rest). All LIVDs demonstrated a significant reduction in ADC ($p<0.01$), with the largest reductions being observed at L5/S1.

Running has also been associated with acute changes in LIVD morphology. Dimitriadis et al., (2011) investigate the effect of 1 hour of running on the LIVD in

15 male and 10 female long distance runner ages 23 to 69 years who regularly ran for 1 hour or 10km. Post 1 hour of running on MRI all LIVDs showed a significant reduction in height (p=0.001). The mean LIVD height reduction after 1 hour of running was 5.17 ± 5.8 mm (*P* = 0.001).

4.2 LIVD Recovery – Spinal Unloading

Posture effects LIVD pressure, a systematic review with meta-analysis of studies investigating in vivo LIVD pressure and posture found that sitting induces significantly higher LIVD pressure compared to standing (p<0.01) (Li et al., 2022). In vivo LIVD pressure studies have also found that compared to standing and sitting, prone and sideling postures place significantly less pressure on the LIVD (Sato, Kikuchi, and Yonezawa 1999; Wilke et al., 1999). Spinal unloading refers to postures and positions that minimise LIVD compressive forces with the purpose of promoting LIVD rehydration and elongation (Kumanchik, 2014).

Stelzeneder et al., (2012) investigated the effect of spinal unloading, using a 38-minute supine posture protocol. Post spinal unloading, LIVD height significantly increased in the central region (p=<.001) and posterior region (p=.002). T2-times significantly decreased in the anterior NP (2.7ms, p=<.05), and increased in the AF (+1.6ms, p=<.05) suggesting a redistribution of LIVD water content from the anterior to posterior regions (Stelzeneder et al., 2012). These results were only significant when the intervention took place in the afternoon compared to the morning, this is likely due to the diurnal pattern of LIVD hydration and the LIVD pressure already being high in the morning.

Utilising a pre-test, post-test crossover design, Owens et al., (2009) investigated the effect of 2 spinal unloading positions on spine height post loaded sitting. Stadiometry was used to measure changes in subject height, a proxy for LIVD height. The spinal unloading positions, prone lumbar hyperextension and supine trunk flexion were held for 10 minutes. The researchers observed a significant difference in spine height between post 5 minutes of loaded sitting (mean, 895.74 mm; SD, 59.59) and 10 minutes of prone hyperextension (mean, 898.84 mm; SD, 59.55) ($P<.0001$). A significant difference was also observed in spine height between post 5 minutes of loaded sitting and 10 minutes of supine flexion (mean, 895.85 mm; SD, 59.19) compared to (mean, 899.05 mm; SD, 59.09) respectively ($P<.0001$).The supine hyperextension position was associated with 3.11 mm (SD, 2.81 mm) gain in vertebral height, and the prone flexion position associated with 3.19 mm (SD, 3.00 mm), there was not a significant difference between the positions ($P = .927$).

Following a loading program of seated miliary presses (3 sets of 8 reps at 60% at 1 repetition maximum) designed to impart compressive forces to the LIVDs, Rodacki et al., (2008) compared the effect of a static spinal unloading position to abdominal exercises to restore spinal height. Post military press, a significant loss of height as measured by stadiometer ($P>0.05$). Three different spinal unloading interventions were tested, of which there was a significant difference in stature recovery between the interventions. The static unloading posture Fowlers position was used, the upper body was supported at an incline of 30° with the hips flexed and supported to 90 degrees, which was maintained for 4.5 minutes.

Regular abdominal crunches with knees flexed to 90° (ABS1), and abdominal crunch on a 45° incline bench with feet supported (ABS2) which were performed with 3 sets of 30 reps, performed at 1.5 second concentric and eccentric phase, with 30 seconds rest between sets. Compared to Fowlers position, ABS1 and ABS2 were associated with a significant recovery in post exercise height ($p<0.05$). There was no significant difference between ABS1 and ABS2, but ABS1 tended to have a greater effect on height recovery ($p=0.07$). Fowlers position also had a significant effect of recovering height, but to a lesser extent than ABS1 and ABS2 ($P<0.05$).

4.3 LIVD Interventional Studies

Khanzadeh, Mahdavinejad, and Borhani, (2020), conducted a quasi-experiment study of male office staff to investigate 2 different core exercise programs on L4/L5 and L5/S1 LIVD herniation with unilateral or bilateral lower extremity radicular pain. The cohort were split into a conventional core training (n=13, age: 43.4±8.6 years) and a suspension core training group (n=11, age: 37.2±5.3 years). Pre and post intervention MRI was used to assess LIVD height and herniation index. Khanzadeh, Mahdavinejad, and Borhani, (2020), did not observe a significant difference in LIVD height or herniation index post intervention, however, at L4/L5 the suspension core stability group and at L5/S1 the conventional core stability showed a marginal improvement in LIVD height. In both groups at L4/L5 and L5/S1 LIVD herniation index tended to reduce indicating a reduction in herniation, however this did not approach significance.

In a prospective randomised control study, Owen et al., (2020) investigated the effect of exercise on LIVD health. In both groups the researchers included participants aged 24-45 years (range) with non-specific chronic low back pain and a mean Pfirrmann grade of 2.3 (0.5 SD). The exercise groups program included aerobic and resistance exercises for 2, 1 hour sessions per week for the first 3 months, and 1-2 sessions per week for the last 3 months. The exercise emphasised axial loading e.g., squat, deadlift, trunk flexion and trunk extension. In comparison the control group in the first 3 months received 12, 30-minute physiotherapy led manual therapy and core motor control sessions e.g., non-weightbearing exercises stimulating the multifidus, pelvic floor and transverse abdominus. During the second 3 months the control group received 2 sessions. In both the exercise and control group, markers of LIVD health: LIVD T2-time, rate of LIVD expansion in short duration lying, and ADC value did not significantly improve. However, there were some within group positive trends in the markers of LIVD health. The control group experienced positive LIVD changes in the first 3 months of the intervention, whilst in the last 3 months of the program these benefits tended to regress back to baseline levels. The control group had a significant positive change in average LIVD height between base line and a 3-month MRI 1.3% increase ($p=0.035$), and a positive change in T2-times in the NP however this did not approach significance. In contrast the exercise group tended to show reductions in NP T2-times. The difference between the first versus the second 3 months was a reduction in delivered sessions. This may suggest that in this cohort of subjects the exercise groups program was not as suitable as the control groups program for creating positive LIVD changes.

The benefits in the control group of Owen et al., (2020) observed, albeit non-significant may have been partly due to the application of manual therapy. Mitchell et al., 2017 performed a systematic review of the physiological effects of physical therapy interventions on LIVDs. The study included in vitro and animal studies which were excluded for the purpose of this review. The included in-vivo literature demonstrated that lumbar spine traction can increase LIVD height, increase water content of degenerated LIVDS, and Lumbar spine mobilisation can increase ADC of degenerated LIVD. A single Lumbar spine high velocity manipulation intervention and a 10-minute treatment of low velocity lumbar spine mobilisation can positively influence LIVD ADC. Increased ADC is a mechanism by which the LIVD receives nutrients and rids metabolites, suggesting a positive shot term effect.

4.4. LIVD Loading followed by unloading protocols

Sustained mechanical loading has been found to significantly reduce transport of small solutes into the centre of the LIVD (Arun et al., 2009). Using non-ionic contrast Gadoteridol, Arun et al., (2009) investigated the effects of mechanical loading on diffusion of small solutes in vivo. Initially subjects acquired postcontrast MRI in a supine unloaded position at 1.5, 3, 4.5, 6 and 7.5 hours, during this process the subjects remained in bed rest. One month after this initial process, in supine subjects were loaded with 50% body weight to simulate standing. Postcontrast MRI were acquired at 1.5, 3 and 4.5 hours whilst loaded, and 1.5 and 3 hours after unloading. During this entire period subjects remained

in bed rest. Compared to the initial unloaded MRI, the loaded MRI observed reductions in LIVD solute transport at 1.5 hours of loading which continued up to decreased to the 4.5 hour MRI. In the loaded MRI LIVD diffusion had started to recover at 6 hours, and by 7.5 hours diffusion matched the rate seen at 7.5 hours in the unloaded conditions. Arun et al., (2009) suggest that sustained loads to the LIVD reduce diffusion and can therefore create relative nutritional deficiency and hypoxia which may predispose the LIVD to degeneration.

In a dynamic exercise stress test, Chokan et al., (2016) found that LIVD T2 values significantly decreased ($p<0.01$), but significantly increased with 30 minutes of supine rest ($p<0.01$). T2 values pre-exercise and post-rest were not significantly different, indicating that 30 minutes of supine rest allowed for nearly full recovery of LIVD T2 values.

5. Conclusion

5.1 Introduction

This literature review aimed to understand how load affects pathology and positive adaptive changes in the LIVD in vivo, with specific considerations to athletic populations. The following objectives were used to investigate this aim:

1. Critically appraise studies that investigate the effect of chronic load on the LIVD in vivo, in athletic populations.

2. Review the effects of LIVD degeneration and injury on lumbar spine biomechanics in vivo.

3. Investigate research on LIVD load management.

5.2 Research objectives: Summary of Findings and Conclusions

5.3 Objective 1: Critically appraise studies that investigate the effect of chronic load on the LIVD in vivo, in athletic populations

Weightlifting

In elite adolescents and adults, and former elite athletes, weightlifting is associated with lower NP T1ρ values, and increased prevalence of LIVD degeneration.

Cricket Fast bowling

Cricket fast bowling is associated with higher Pfirrmann grades and a high prevalence of LIVD degeneration, in both adolescent amateur and adult professional players. Technique change from a mixed to either front or side on bowling technique, which creates less lumbar axial rotation, flexion, extension and lateral flexion is associated with a lower prevalence of LIVD degeneration in cricket fast bowlers.

Cycling

High volume cycling >150km per week for 5+ years is associated with greater LIVD height, LIVD height to vertebral body ratio and longer LIVD T2-times compared to controls. Thus, cycling is associated with improved LIVD glycosaminoglycan content and hydration.

Running

Running 20-50+km per week for 5+ years is associated with higher LIVD T2-times, LIVD height relative to vertebral body height and lower LIVD Pfirrmann scores compared to controls. This suggests that the LIVDs are relatively hypertrophied with better hydration and glycosaminoglycan content, a positive adaptation. Further, slower running pace may have a greater benefit in the ranges of 1.5-2.5 meter per second, 5.4-9 kilometres per hour.

Conclusion 1

Chronic low level cyclic loading from running or cycling is associated with improved LIVD health, whereas sports that place high load, with larger ranges of lumbar spine motion are associated with a higher prevalence of LIVD degeneration.

5.4 Objective 2 - Review the effects of LIVD degeneration and injury on lumbar spine biomechanics in vivo

Effect of Posture on LIVD Herniation

Compared to supine posture, standing posture is associated with increased LIVD herniation size. Higher grades of LIVD degeneration as scored by Pfirrmann classification is associated with increased LIVD bulging in upright axially loaded MRI. Healthy LIVDs are associated with predictable movement; an anterior direction with lumbar spine extension and a posterior movement with flexion, however LIVD movement is less predictable in degenerated LIVDs.

Mechanical deformation

Lower T1rho relaxation times are associated with increased LIVD compressive mechanical deformation. Higher grades of LIVD degeneration are also associated with same and adjacent level increases in shear and tensile deformations. Under axial load, lower NP T2-times and higher Pfirrmann grades are associated with higher LIVD axial strain.

LIVD Degeneration and Lumbar Spine Motion

Lumbar spine segmental angular motion tends to decrease, and segmental translation tends to increase in degenerated LIVDs. LIVD degeneration is associated with increasingly disperse axis of rotation and an increase in coupled motion which is associated with higher facet joint loading. This is observed at the same and adjacent level of LIVD degeneration.

LIVD Degeneration and the intervertebral foramen

LIVD degeneration is associated with reductions in intervertebral foramen size, in supine, upright flexed and extended postures at the same, and adjacent level and with dynamic intervertebral foraminal stenosis in standing versus supine posture.

Conclusion 2

LIVD degeneration is associated with a reduction in segmental angular range of motion, increased segmental translation, altered axis of rotation and reduced vertebral intervertebral size at same and adjacent levels.

5.5 Objective 3 - Investigate research on LIVD load management-

Acute effects of exercise

Squatting and deadlifting is associated with acute reductions in LIVD ADC values, lower squat depth is associated increased lumbar flexion range of motion and more significant reductions in ADC values. Running is associated with acute reductions in LIVD height.

LIVD Recovery - Spinal Unloading

Posture effects intra LIVD pressure, with pressure increasing from prone and side-lying postures to standing and highest pressure being associated with sitting. Following axial spine loading, prone lumbar hyperextension or supine with trunk flexion for 10 minutes is associated with increased LIVD height. Further compared to Fowlers position abdominal crunches have significant effect at increasing LIVD height post axial loading of the spine.

LIVD Interventional Studies

Interventional studies have investigated the effect of exercise on LIVD health, with no significant findings reported. However positive trends in markers of LIVD health were seen in a group that received gentle core exercises and manual therapy. Manual therapy interventions are associated with acute positive changes in the LIVD; spinal traction is associated with acute increased in LIVD height, and high velocity manipulation and mobilisation are associated with acutely increased LIVD ADC.

LIVD loading followed by unloading protocols

LIVD exercise stress tests have found post exercise reductions in LIVD T2-times, which recover back to near baseline levels following 30 minutes of supine rest.

Conclusion 3

Exercise stress is associated with an acute reduction in LIVD height and ADC values. Supine postures are associated with a reduction in LIVD intradiscal pressure and can be used post exercise to acutely increase LIVD height and LIVD T2-times.

5.6 Limitations

Due to a lack of prospective cohort studies the objective 1 conclusion is based upon cross sectional observation studies, thus survival bias is a possible confounding factor to this conclusion. Therefore, randomised controlled trials and long-term prospective cohort studies are needed to further establish this conclusion. Further the researcher was limited by time constraints of being in full time clinical practice, therefore sports with numerous available studies were excluded such as Gymnastics and American Football from review, had they been included a different conclusion may have been established.

5.7 Research Recommendation

To the researcher's knowledge there has not been an interventional study investigating the effect of exercise in combination with a regular spinal unloading protocol on long term LIVD health. Therefore, interventional studies utilising a spinal unloading protocol post training, to investigate whether this helps to reduce LIVD degeneration would be helpful for sports and exercise professionals in the load management of the LIVD. A second research recommendation is an interventional study investigating an exercise protocol consisting of slow long-distance running or long-distance cycling with a post-exercise spinal unloading protocol on LIVD health.

5.8 Research Conclusion

Chronic low level cyclic loading from running and cycling is associated with better LIVD health, whereas sports that place high load with larger and faster ranges of motion are associated with a higher prevalence of early LIVD degeneration. LIVD degeneration is associated altered lumbar spine biomechanics at the same and adjacent levels. Subsequent to the lumbar spine being axially loaded from exercises stress, spinal unloading postures for 10 or more minutes have an acute, positive effect on LIVD health parameters. The implication of this to sports and exercise medicine are that integrating a 10+ minute unloading protocol post axial spine loading may support LIVD injury prevention, rehabilitation, and load management. Further research is required to investigate whether spinal unloading positions can be utilised within load management strategies to reduce early LIVD degeneration associated with sport.

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