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Sitting versus standing: Does the intradiscal pressure cause disc degeneration or low back pain?

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Abstract

Studies of lumbar intradiscal pressure (IDP) in standing and upright sitting have mostly reported higher pressures in sitting. It was assumed clinically that flexion of the lumbar spine in sitting relative to standing, caused higher IDP, disc degeneration or rupture, and low back pain. IDP indicates axial compressive load upon a non-degenerate disc, but provides little or no indication of shear, axial rotation or bending. This review is presented in two main parts. First, *in vivo* IDP data in standing and upright sitting for non-degenerate discs are comprehensively reviewed. As methodology, results and interpretations varied between IDP studies, *in vivo* studies measuring spinal shrinkage and spinal internal-fixator loads to infer axial compressive load to the discs are also reviewed. When data are considered together, it is clear that IDP is often similar in standing and sitting. Secondly, clinical assumptions related to IDP in sitting are considered in light of basic and epidemiologic studies. Current studies indicate that IDP in sitting is unlikely to pose a threat to non-degenerate discs, and sitting is no worse than standing for disc degeneration or low back pain incidence. If sitting is a greater threat for development of low back pain than standing, the mechanism is unlikely to be raised IDP.

Keywords: Intervertebral disc; Intradiscal pressure; Lumbar; Sitting; Low back pain

1. Introduction

Intradiscal pressure (IDP) is the hydrostatic pressure measured in the nucleus pulposus of a non-degenerate intervertebral disc. Standing and sitting are everyday activities, which has made the findings of posture and lumbar IDP studies immediately relevant and memorable. Much lower IDP has been reported in standing than in sitting. It has been reported that "in standing, the disc pressure is about 35% of the pressure in relaxed sitting without back support" (Andersson et al., 1975). Sitting on a horizontal surface involves lumbar flexion relative to standing (Makhsous et al., 2003; Scannell and McGill, 2003), and it has been assumed clinically that lumbar flexion was the cause of higher IDP, tensile stress to the annulus, disc degeneration and low back pain (Nachemson and Morris, 1964; Andersson and Ortengren, 1974; Andersson et al., 1974a,b; Nachemson, 1975). IDP data and related assumptions have formed a basis for clinical advice to advocate erect sitting postures for the prevention of spinal complaints (Brunswic, 1984; Pynt et al., 2002). However, some studies have reported near identical pressures "with the subject either sitting [relaxed without back support] or standing... the intradiscal pressure was approximately 300 kilopascals [0.30 MPa]" (Schultz et al., 1982). Similar pressures in standing and sitting would undermine a common clinical basis for advocating erect sitting postures, and encourage broader inquiry into aetiologies for pathology and pain at the lumbar spine.

This paper uses two approaches to examine IDP in upright sitting (relaxed without back support). First, *in vivo* IDP data for non-degenerate lumbar discs in standing and upright sitting are comprehensively reviewed, with consideration for measurement methodologies, results, and

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interpretations. Second, the assumed clinical links between IDP in sitting, disc degeneration and low back pain are considered through basic *in vitro* studies (IDP and degeneration), spinal imaging and epidemiologic studies (sitting, disc degeneration and low back pain). The biomechanical emphasis of this review is the axial compressive load affecting intervertebral discs, as this relates to IDP. Spinal pain can relate to other mechanisms of loading, load-bearing structures, neuro-muscular control, tissue physiology and psychosocial factors, but those aspects are beyond the scope of this review.

2. IDP measurement: development of methodology

Over the past thirty years, there has been a clear progression of the accuracy and sensitivity of *in vivo* IDP measurement. It is important to review these developments in methodology, to provide context for interpretation of results. With IDP measurement, the pioneering work of Nachemson, Morris, Elfstrom, Andersson and Ortengren in the 1960s and 1970s, helped to pave the way in the study of spinal biomechanics. The first pressure transducer used a polyethylene membrane (elastic tubing) over the tip of a hollow liquid-filled needle, connected to an electromanometer (Nachemson, 1963; Nachemson and Morris, 1964; Nachemson, 1965, 1966; Okushima, 1970). Research subjects for these studies had no apparent disc degeneration on radiograph or discography, but did suffer from low back pain. Early studies showed similar pressure readings with changes in transducer orientation, which provided evidence that the nucleus pulposus of a non-degenerate intervertebral disc could behave hydrostatically. Unfortunately, apparatus used in early studies had a number of limitations. The polyethylene membrane, covering the needle tip, had insufficient sensitivity for dynamic pressure measurement. The fluid-filled needle could also alter the measured pressures if it was bent more than 20°. Movement and changes in the research subject's posture could induce at least this degree of bend in the needle (Nachemson and Elfstrom, 1970). Table 1 shows a list of studies that reported IDP data from standing and sitting in non-degenerate discs (with population studied and transducer type).

In the 1970s, major advances in transducer technology and calibration increased accuracy, and produced results ~25–33% lower than earlier measures (as shown in Figs. 1 and 3). Calibration to body temperature, rather than room temperature, improved precision of recordings; and the liquid-filled needle was replaced with a piezoresistive semiconductor strain gauge (embedded in an epoxy resin, and covered with a pressure sensitive membrane, at the tip of a 0.8 mm diameter transducer needle) (Nachemson, 1975). It was reported that this new transducer needle could bend as much as 40° without influencing accuracy of pressure measurement (Nachemson and Elfstrom, 1970). Furthermore, research subjects with non-degenerate discs and no history or low back pain were used, but it is uncertain whether a history of back pain influenced IDP measurements. From 1983, there were no further studies of IDP with non-degenerate discs in standing and sitting until the late 1990s.

In 1999, two separate research teams reported IDP measurements. One team from Japan (Sato et al., 1999) used a similar design to the 1970s apparatus, but with the piezoresistive sensor positioned laterally in the transducer needle. In this way, the pressure was detected at a window in the guiding needle, rather than at the needle tip. Another team in Germany (Wilke et al., 1999) avoided the problem of having a needle *in situ*, by using telemetry to record from an implanted transducer measuring 1.5 mm diameter, by 7 mm in length. This implanted transducer had the advantage of transmitting data during a variety of daily activities over 24 h, compared with other studies that reported trials of ~15 s or less (Schultz et al., 1982).

3. Comparison of IDPs in standing and sitting

3.1. Method for comparison of studies

IDP results from all published studies with individual subject data from non-degenerate discs in standing and sitting were converted to megapascals (MPa), and are shown in Fig. 1. Broad error bars shown in Fig. 1 denote the range of data values for each column, except for one study with narrow error bars to denote the standard deviation (range data were not available). Two studies that did not report individual data were also included because they involved authors from earlier studies, and the lower pressures reported in these papers were an important finding. The

Table 1
Comparison of subjects and studies that report standing and upright sitting IDP in non-degenerate discs

Author	Year	L3-4, *n*	L4-5, *n*	Low back pain	Transducer type
Nachemson[a]	1964/1965	6	4	Yes	Liquid-filled
Okushima	1970	10	20	Yes	Liquid-filled
Nachemson and Elfstrom	1970	7		No	Piezoresistive
Andersson et al.	1974	4		No	Piezoresistive
Schultz et al.	1982	4		No	Piezoresistive
Sato et al.	1999		8	No	Piezoresistive, side-window
Wilke et al.	1999		1	No	Piezoresistive, implanted

[a] Data from the Nachemson and Morris (1964) and Nachemson (1965) papers are combined, as indicated by the author in 1965.

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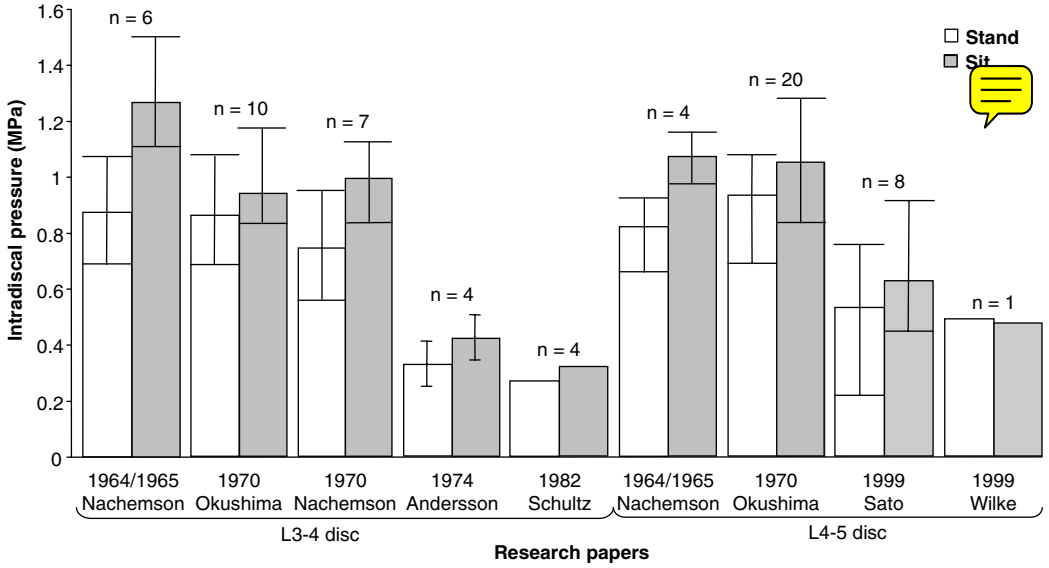

Fig. 1. IDP results in standing and sitting: 1964–1999. Mean IDP and number of subjects are shown for the L3-4 disc (Nachemson and Morris, 1964; Nachemson, 1965; Nachemson and Elfstrom, 1970; Andersson and Ortengren, 1974; Schultz et al., 1982) and the L4-5 disc (Nachemson and Morris, 1964; Nachemson, 1965; Okushima, 1970; Sato et al., 1999; Wilke et al., 1999) in standing and sitting. Ranges of IDP measured are shown in five studies (Nachemson and Morris, 1964; Nachemson, 1965; Nachemson and Elfstrom, 1970; Okushima, 1970; Sato et al., 1999). For Andersson and Ortengren (1974), mean IDPs were estimated by adding the normalisation pressure (0.508 MPa) to values measured from Fig. 6 in that paper, and error bars denote the SD (0.081 MPa). For Schultz et al. (1982), only the mean IDPs were reported. For Wilke et al. (1999), IDPs reported are mid-point values for data from the single subject. A trend to lower IDP, with smaller differences between the postures is seen with successive studies.

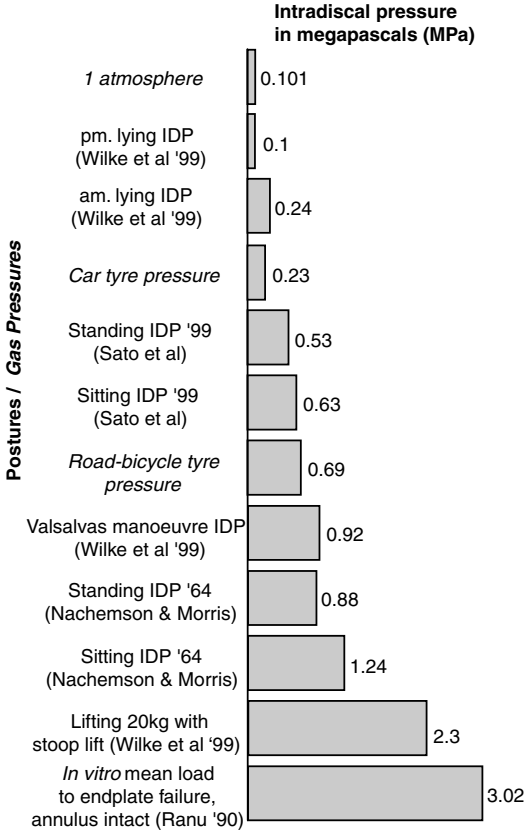

Fig. 2. Benchmarks for comparison of IDP. IDP results in standing and sitting from 1964 and 1999 are shown, along with a variety of postures, some common fluid pressures (in italics) and average *in vitro* compressive load to failure of the vertebral endplates (annulus commonly intact).

two studies in Fig. 1 without individual subject data were Andersson et al., 1974 (mean and SD values), and Schultz et al., 1982 (mean values only).

Although all data shown in Fig. 1 is from subjects with no signs of disc degeneration on spinal imaging (radiograph, discogram, fluoroscopy, or MRI, depending upon the study), in studies up to 1970 all subjects suffered from low back pain (Nachemson and Morris, 1964; Nachemson, 1965; Okushima, 1970). Data from subsequent studies were all from subjects with no history of low back pain (Table 1).

To assist interpretation of pressures in MPa, Fig. 2 shows early (Nachemson and Morris, 1964) and recent IDPs (Sato et al., 1999) relative to common fluid pressures. Pressure results in Figs. 1–3 from early studies of IDP (p_n) were multiplied by 0.098 to convert results from kg/cm^2 to MPa, (Nachemson and Morris, 1964; Nachemson, 1965; Okushima, 1970).

3.2. IDP results in standing and sitting

The earliest data, obtained with a polyethylene tipped liquid-filled tranducer, in subjects with a history of low back a pain, showed the mean pressure in sitting to be 1.24 MPa (11 atmospheres, shown in Fig. 1) (Nachemson, 1965). In contrast, more recent studies with piezoresistive transducers and subjects with no history of low back pain, have reported smaller pressures of 0.5–0.6 MPa (~5–6 atmospheres) for standing or sitting (Sato et al., 1999; Wilke et al., 1999). Aside from large variation in means between studies, there is also large variation between subjects (Okushima, 1970; Andersson et al., 1974a; Sato

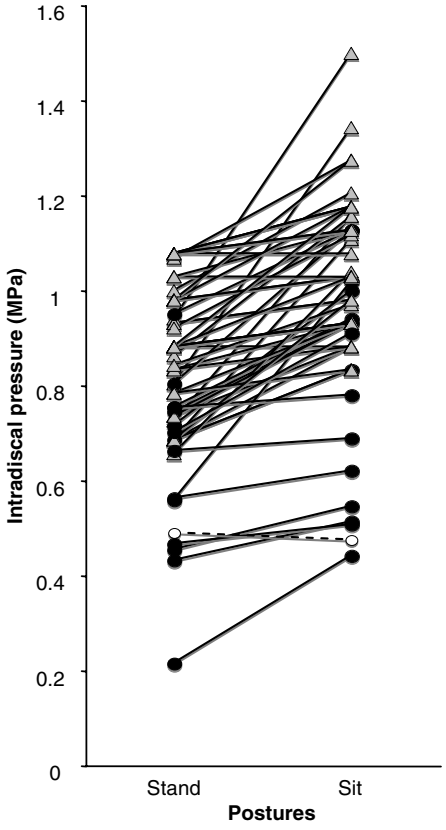

Fig. 3. Individual results for IDP in standing and sitting. Individual IDP data from Fig. 1 are shown (Nachemson and Morris, 1964; Nachemson, 1965; Nachemson and Elfstrom, 1970; Okushima, 1970; Sato et al., 1999; Wilke et al., 1999), from L3-4 and L4-5 intervertebral discs. ▲: Liquid-filled needle-transducer (subjects with low back pain). •: Piezoresistive needle-transducer (no history of low back pain). ○: Piezoresistive implanted-transducer (no history of low back pain). Needle-transducer measurements generally show an increase in IDP from standing to sitting, with large variation in IDP and differences between postures. Only the implanted-transducer shows a slight reduction in IDP from standing to sitting.

et al., 1999). The most recent needle-transducer study (Sato et al., 1999, shown in Fig. 1), demonstrated greater variation between subjects, than the difference between mean pressures for standing and sitting. The recent findings of lower mean pressures and large between-subject variation highlight a need to re-evaluate the interpretation of IDP results.

Despite large variability, measurements with needle-transducers showed lower pressures in standing than in sitting (Fig. 3) which implied a systematic effect of posture upon pressure, but there may be important confounders. For example, changes in posture may have resulted in muscular responses to the needle *in situ*, needle bend or needle-transducer movement relative to tissues. Furthermore, spinal flexion was not quantified with respect to the available range of motion. If the lumbar spine was sufficiently flexed in upright sitting, tension in posterior ligaments could have caused an increase in IDP measured.

In contrast, data from the implanted transducer in a single subject indicated marginally higher IDP in standing (0.49 MPa) than in sitting (0.47 MPa, Figs. 1 and 3) (Wilke et al., 1999). The implanted transducer presented advantages for data collection and accommodation of the subject to the apparatus; being a smaller apparatus that did not protrude from the disc, and *in situ* for recording over 24-h. Despite these advantages, results from a single-subject study must be considered with some caution. At present, the only alternative methods to infer IDP differences between standing and sitting are by studies of change in spinal height or load upon internal fixators.

3.3. Spinal height and load cell measures in standing and sitting

In vitro studies have shown a near-linear relationship between increased axial compressive load applied to an intervertebral disc and increased IDP (Nachemson, 1963; Berkson et al., 1979). *In vivo*, progressively higher pressures measured from lying, to standing, to lifting a weight, provide evidence that greater compressive load upon the disc is associated with greater IDP (Nachemson and Elfstrom, 1970; Schultz and Andersson, 1981). Several studies have sought to measure disc compression either indirectly (stadiometry) or directly (load-cell equipped spinal fixators). Although IDP has not been concurrently measured with disc compression *in vivo*, compression measures in standing and sitting permit inference as to which posture causes a higher IDP.

Stadiometry is the measurement of spinal height, and is used to infer the effect of disc compression in different activities. Activities with greater compressive load cause lesser disc hydration, and thus reduce spinal height (Eklund and Corlett, 1984; Kingma et al., 2000; Hutton et al., 2003). Stadiometry has the advantages of providing a non-invasive, intra-subject comparison of sustained disc compression (e.g. 30 min or more) with different postures. Subjects commonly lose more height in standing than they do in sitting (1–5 mm difference) (Althoff et al., 1992; Lievseth and Drerup, 1997), which infers that standing causes more disc compression than sitting.

Load cells within steel bars for surgically implanted spinal fixators have been used as a more direct measure of compression and flexion torque between two spinal segments (Rohlmann et al., 2001). Data from load-cell fixators have been reported for ten subjects, with spinal fixators applied to various levels from T11-L4 (data transmitted by telemetry). Again, sitting postures showed lower compression than upright standing, consistent with implanted-transducer disc pressure and stadiometry, but contrary to needle-transducer results.

Each experimental approach has limitations. Stadiometry of different postures has a small effect size, but relatively large variation between subjects and between trials for the same subject (Althoff et al., 1992; McGill et al., 1996; Lievseth and Drerup, 1997; van Deursen et al., 2005). The load-cell fixator study was a series of single cases, with

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fixators at a variety of spinal levels (Rohlmann et al., 2001). However, taken together these studies provide evidence in support of results from the implanted-transducer study, to show that sitting may be associated with less disc compression than standing.

3.4. Interpretation of pressure in standing and sitting: developments with methodology

Results obtained with IDP measurement appear to be greatly influenced by the experimental methodology. Consequently, there has been a parallel development of interpretations for the results, and how they inform rehabilitation and ergonomic advice. For example, initial *in vitro* results from 1963 showed similar pressures in extension or flexion of a loaded spinal segment (Nachemson, 1963). Subsequent *in vivo* studies reported a difference between pressures in standing (extension) or sitting (flexion) (Nachemson and Morris, 1964), which led the author to state, "it might appear unreasonable that the load decreases in shifting from sitting to standing" (Nachemson, 1966). At the time it was hypothesised that sitting might have caused higher muscle activity in psoas major, and thus higher IDP. It was also suggested that disc degeneration was a result of tensile fractures of the annulus fibrosus (Nachemson and Morris, 1964). Pressures in sitting of ~1.2 MPa were considered to support a mechanical theory of ruptures to the posterior annulus fibrosus, leading to irritation of free nerve endings in the posterior disc, thus contributing to low back pain (Nachemson and Morris, 1964).

In the 1970s, electromyography showed that muscle activity of psoas major did not explain IDP differences between standing to sitting. The new hypothesis was that lumbar flexion caused higher IDP than lumbar extension (Andersson et al., 1974a). Several papers reported a direct link between increased lumbar flexion and increased IDP in sitting (Andersson and Ortengren, 1974; Andersson et al., 1974a,b; Andersson et al., 1975; Nachemson, 1975). Based on the assumption that lower disc pressure was advantageous for reducing low back pain, advice followed the mechanical theory for disc ruptures causing chemical irritation and pain. 'Back Schools' recommended postures and movements that had been shown to involve lower disc pressures, including the 'take home' message for Physiotherapist's ergonomic advice that "straight standing is better than unsupported sitting" (Nachemson, 1975).

Notably, the same authors recognised that there was no direct evidence to link sitting and the aetiology of disc degeneration, mechanical ruptures, or pain (Nachemson, 1975). However, with the high incidence of disc degeneration, pain and loading the spine in sitting, correlation of these variables may have been interpreted as causality. The difficulty in determination of pain aetiology, and a lack of alternative disc degeneration paradigms may have strengthened assumptions that IDP in sitting could be adverse for disc health.

With progression of transducer technology and calibration, the lower pressures reported gave reason to question assumptions about disc health being affected by the IDP in sitting. One example was a lifting-task study, which moved away from labeling the IDP in sitting as threatening, with the statement that "it is clear that in the upright sitting and standing positions there are small differences [in pressure]" (Nachemson and Elfstrom, 1970). By the late 1970s, standing pressure was reported as low as 0.33 MPa (3 atmospheres) at the L3-4 disc (Andersson et al., 1978), and a 1982 lifting-study reported that pressures in sitting and standing were similar (Schultz et al., 1982).

Further challenging previous interpretation of the data, evidence has suggested that the position of spinal flexion or extension is a poor predictor of IDP. Lumbar flexed postures, such as slumped sitting with elbows resting on thighs, can demonstrate similar pressure to upright sitting (Andersson et al., 1974a) and lower pressure than standing (Wilke et al., 1999). Studies of lifting also measured IDP with flexed and straight spinal postures. Although some studies reported higher IDP in lumbar flexed (stoop lift) than straight spine (squat lift) strategies (Nachemson and Elfstrom, 1970; Wilke et al., 2001), others showed similar pressures for both alternatives (Andersson et al., 1976; Andersson et al., 1978). This variability suggests that the degree of spinal flexion or extension is a lesser determinant than other variables affecting IDP (e.g. individual variation in muscle activity, facet joint load or ligamentous tension for a given task).

In summary, pressures for upright standing and sitting appear to be ~0.5–0.6 MPa (~5–6 atmospheres) with inter-subject variation of ~0.2–0.3 MPa (±2–3 atmospheres), based on mean pressures from piezoresistive needle-transducer studies (Andersson et al., 1974a; Schultz et al., 1982; Sato et al., 1999). Contrary to results from needle-transducer studies, data from an implanted-transducer (Wilke et al., 1999), studies of spinal shrinkage and internal fixator loads (Althoff et al., 1992; Lievseth and Drerup, 1997; Rohlmann et al., 2001) infer that similar or marginally greater IDP is likely in standing than sitting. Thus, a pertinent question is whether IDP of 0.3–0.7 MPa in sitting poses a mechanical threat to the annulus fibrosus of a non-degenerate disc, an elevated risk of low back pain or both.

4. Does IDP in sitting pose a threat to the annulus fibrosus or low back pain?

4.1. Does IDP in sitting threaten or protect the annulus fibrosus in vitro?

In the 1960s and 1970s, horizontal force of IDP upon the annulus was proposed as the mechanical cause for tensile rupture of disc fibres (Nachemson and Morris, 1964; Nachemson, 1975). Three assumptions linked this tensile rupture model to the IDP of non-degenerate discs in sitting. First, it was theorised that an optimum IDP for intervertebral discs would be lower than that the IDP in sitting

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(Nachemson, 1975), despite little data on annulus cell physiology. Second, higher IDP was equated with higher risk of annulus rupture and low back pain, because tensile stress to annulus fibres was mainly equated with compressive load (Nachemson and Elfstrom, 1970), with less attention to loads in shear, axial rotation or bending. Thirdly, hydrostatic behavior of the nucleus pulposus indicated an even pressure per unit area at the annulus fibrosus and adjacent endplates (Nachemson and Elfstrom, 1970), but not the load distribution within the annulus. Advances with *in vitro* research methods have provided new insights into annulus response to IDP in sitting, biomechanics of disc rupture and load distribution within the annulus.

One aspect affecting annulus fibrosus integrity is the metabolic response of disc cells to compressive loading. Composition and structure of the annulus matrix are determined by the balance of protein synthesis and degradation. Cells harvested from the inner annulus fibrosus and nucleus pulposus undergo metabolic change in response to hydrostatic pressure *in vitro* (Handa et al., 1997). Pressure of 0.3 MPa applied for 2 h (the lower limit of 0.3–0.7 MPa in sitting or standing) can stimulate proteoglycan synthesis and inhibit degradation. In contrast, 0.10 MPa pressure (similar to the pressure in lying down) can inhibit synthesis and facilitate degradation. Pressures within normal physiological range appear to provide an essential mechanical stimulus for maintenance of the proteoglycan matrix, and consequent load bearing capacity of the intervertebral disc.

Investigation of disc biomechanics with axial compressive loading has indicated that tensile rupture of annulus fibres is unlikely with compression alone (Adams and Dolan, 1995; Fujita et al., 1997). An *in vitro* study of 43 isolated spinal motion segments showed that a mean repetitive load of 3076 N compression (range: 1500–6000 N) applied at 0.66 Hz for 4 h, resulted in trauma at the vertebral endplates or anterior vertebral bodies, but not the disc (Adams and Hutton, 1983). Twenty of the spinal motion segments were then loaded to failure, with the pressure increased by a further 3000 N/s. Only 3 of the 20 segments suffered a disc protrusion or prolapse. Failure most commonly occurred at the vertebral body (Adams and Hutton, 1983).

Similar results were found in a more recent study with eight *in vitro* spinal segments (Ranu, 1990). A mean critical compressive load of 4370 N (SD 1030) was associated with a mean IDP of 3.02 MPa (SD 0.76) for vertebral endplate failure. In summary, compressive loads ~10 times the magnitude of upper-body weight rarely cause annulus fibrosus failure in a non-degenerate disc. With axial compression alone, IDP of ~0.5–0.6 MPa in sitting appears trivial for non-degenerate discs, when compared with ~2.26–3.78 MPa required for endplate failure. Loading mechanisms other than simple axial compression must be involved in initiating disc degeneration.

Combined loading of spinal motion segments with compression (1,334 N), flexion (7°) and axial rotation (<3°) for 3–13 h, has been observed to cause disc failure in all 14 discs that were tested (Gordon et al., 1991). While this study aimed to simulate repetitive loading in a very flexed position, combined loading can also occur throughout range. Even in mid-range spinal positions, neuromuscular control is essential to prevent spinal motion segments from buckling in shear, axial rotation and/or bending (Crisco and Panjabi, 1992; Adams, 1995). The potential for these combined loads to occur, and evidence that combined loads can cause disc trauma, raise the question of how combined loads affect disc integrity differently to axial compression alone.

Structurally the annulus fibrosus is a composite material, with 20–40 layered sheets (lamellae) of collagen fibres in a proteoglycan matrix. Although pressure is relatively evenly distributed in the nucleus of a healthy disc (i.e. hydrostatic behaviour) (Sato et al., 1999), this even load distribution from nucleus to annulus does not indicate how load is distributed within the annulus fibrosus. Profilometry was developed as a progression from IDP measurement, to measure both IDP and annulus fibrosus stress profiles (McNally and Adams, 1992; McNally et al., 1992). Typically profilometry utilizes a piezoelectric transducer in a lateral window of the needle (similar to that used by Sato et al.) to measure changes in compressive load as the transducer is drawn across a diameter of an intervertebral disc (McNally and Adams, 1991). Non-degenerate discs loaded in a neutral position with axial compression, show uniform stress profiles at the nucleus pulposus and inner - middle annulus fibrosus layers (lower stress at outer annulus). With combined loading in compression plus antero-lateral bending, stress profiles in the annulus fibrosus were not uniform, and 10 of the 22 segments failed at the disc (McNally et al., 1993) (compared with 3 out of 20 with axial compression alone (Adams and Hutton, 1983)). All 10 discs that failed in combined loading showed concentrations of higher stress in the annulus fibrosus (especially the posterior annulus). Of 12 that failed at the vertebral endplate, only two demonstrated stress concentrations in the annulus. That is, combined loading can produce regions of higher stress in the posterior annulus (~1.5 × IDP), and these stress concentrations were strongly associated with disc prolapse (McNally et al., 1993).

Identification of these stress concentrations with combined loading, shed new light on disc pathomechanics. Ensuing studies of non-degenerate discs have demonstrated annulus stress concentrations as a consequence of reduced IDP, caused by trauma to vertebral endplates (0–80% reduction in IDP) (Adams et al., 2000a) or sustained compressive load (13–36% reduction in IDP with 3–6 h at 2–4 × upper body weight) (Adams et al., 1996). Degenerate discs commonly display this combination of endplate trauma, reduced IDP (relative to annulus stress) and stress concentrations in the annulus fibrosus. Instead of higher IDP being adverse for the disc, reduced IDP relative to concentrations of annulus stress appear more indicative of adverse loading.

In non-degenerate discs, horizontal stress applied by IDP acts to maintain the annulus lamellae in close apposition. If

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IDP is lower than compressive load to the annulus, inner and outer laminae can buckle and collapse away from middle laminae, causing failure of the inter-lamina matrix, cellular trauma and separation of the lamellae (Adams et al., 1994; Adams and Dolan, 1995; Bruehlmann et al., 2004). Separation and collapse of inner lamellae towards the nucleus is observed in early stages of disc degeneration *in vitro* (Seroussi et al., 1989), as well as formation of local and circumferential annulus tears in animal models (Iatridis and Gwynn, 2004). This breakdown or delamination of the disc's composite structure make it more vulnerable to subsequent loading. Trauma to vertebral endplates and interlaminar matrix will also affect disc nutrition, cell physiology and distribution of load to facet joints, but examination of these changes is beyond the scope of this IDP review.

In summary, health and integrity of the annulus fibrosus appears dependent upon many variables including hydrostatic pressure as a stimulus for matrix metabolism, IDP to support and distribute load to the annulus, and vertebral endplate integrity. Compression alone (measured via IDP) is unlikely to pose a threat to a non-degenerate disc, but combinations of shear, axial rotation or bending with compression may. Current research methods preclude prediction of relationships between common postures, loads and initiation of disc degeneration. Further tools and methods will be needed to predict the complex interactions between variables such as spinal physiology, an individual's spinal anatomy and neuro-motor control (Adams et al., 2000b; Natarajan et al., 2004).

4.2. Does sitting predict disc degeneration or low back pain?

Considering that IDP is of similar magnitude in standing and sitting, it is necessary to evaluate the clinical assumption that exposure to sitting poses a greater risk of disc degeneration and low back pain than exposure to standing. In 1975, an exploratory epidemiological study of people seeking acute medical care, was the first to report a link between sedentary occupations (sitting), reduced disc space on X-ray and acute low back pain (Kelsey, 1975; Kelsey and Hardy, 1975). Occupational motor vehicle driving was reported to have an even stronger relative-risk for reduced disc space and low back pain. The author cautioned that the methodology had limitations, such as the population studied, questionnaire design, response rate, and the large number of associations tested (Kelsey, 1975). The author also stressed that further investigations were needed to confirm or refute the association of sitting, disc degeneration and low back pain. A subsequent study with 45 pairs of identical twins (Battie et al., 2002) refuted the association. One individual from each of the pairs, had more than five times the lifetime exposure to driving a motor vehicle than their twin. No difference in lumbar disc degeneration on MRI, or incidence of low back pain occurred with prolonged exposure to vibration in sitting (Battie et al., 2002). Likewise, greater exposure to sitting at work was no worse for disc degeneration (Battie et al., 1995). Given that sitting commonly involves lumbar flexion relative to standing (Makhsous et al., 2003; Scannell and McGill, 2003) and recent epidemiologic studies (Battie et al., 1995, 2002), lumbar flexion associated with sitting appears to be no worse for disc health or LBP than relative extension in standing.

5. Conclusions and implications

Since the initial studies of IDP, new tools and methods have continued to advance understanding of pathomechanics and neuro-muscular control related to spinal degeneration and low back pain. Current evidence shows similar IDP in standing and upright sitting postures. *In vitro* biomechanics show that the axial compression in sitting is unlikely to pose a threat to non-degenerate discs. Epidemiology shows that sitting is no worse than standing for incidence of disc degeneration or low back pain. If sitting is a greater threat for development of low back pain than standing, the mechanism is unlikely to be raised IDP.

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Andrew Claus *MPhty (Manip)*, *BPhty (Hons)*, is completing PhD studies with Paul Hodges, Julie Hides and Lorimer Moseley advising for the project, using fine-wire EMG to examine spinal extensor muscles with sitting postures (The University of Queensland, School of Health and Rehabilitation Sciences, Australia). Physical interests include rowing, infantry training, road cycling, track & field, long-track speed skating, physical theatre and hiking, etc. but recent years have mostly involved standing and sitting. Future studies will continue to focus upon neuro-muscular coordination of the lumbar spine.

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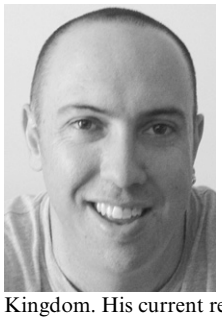

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