Review article

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# Clinical biomechanics of the spine in three unsolved problems. A brief analytical review

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Chronic pathology of the spine, especially its forms, such as degenerative disc disease (DDD), is one of the most common in the human population and a marker for a person. Even though this pathology lacks the burden of mortality, its existence and consequences worsen the quality of life. Hypotheses of the high prevalence of DDD often appeal to a person's upright gait and the function of the spine as a movable vertical support, which means a permanent significant axial load of the intervertebral discs (IVDs). Therefore, finding out the magnitude of such a load, its dependence on the body's position in space, and types of motor activity is an essential practical task of the biomechanics of the spine as a separate interdisciplinary direction of biomedical research.

Despite all the efforts and significant activity during the 70s and 80s of the last century, the central questions of clinical biomechanics of the spine still need to be explored. It is visible from the state of development of three "legendary" problems – elucidation of intradiscal pressure against the background of usual types of physical activity, the role of sitting in the promotion of DDD of the lumbar region, and determination of the role of intra-abdominal pressure in reducing the axial load of this region of the spine. For example, the results of the investigations can state that assessment of intradiscal pressure against the background of human behavioral activity has so far been the focus of a disproportionately small number of works, which, due to the weakness of the accompanying visualization and the technical unreliability of the sensors did not obtain a sufficient empirical base for statistically significant conclusions. Therefore, the urgent task of the future is developing and using a more accurate, reliable, miniature, and durable intradiscal pressure monitoring technique, which would make it possible to evaluate this parameter on large samples of volunteers with conditionally intact IVD and against the background of pathology. In this regard, the assumptions about the role of sitting in the development of DDD of the lumbar spine remain unverified.

Similarly, the research on the phenomenon of intra-abdominal pressure needs to determine under what conditions and mechanisms this factor can affect the magnitude of the axial load on the lumbar spine. Also, constructing more insightful models of the biomechanics of the spine is only possible with expanding ideas about the composition, vascularization, and innervation of the IVD, biology, and pathology of IVD cells. The practical outcome of all these studies is delineation of the most dangerous types of motor activity in the promotion of DDD, which will bring us closer to understanding the drivers of DDD and thus improving the means of preventing and treating this ubiquitous pathology.

**Keywords***: vertebral column; spine; intervertebral discs; degenerative disc disease; biomechanics of the spine; axial load; intradiscal pressure; intra-abdominal pressure*

## Introduction

The term "intervertebral discs" (IVDs) is familiar to any adult due to the pathology with the conventional name "degenerative disc disease" [1]: despite the lack of clear causal correlation between clinical and pathomorphological aspects [1, 2], its prevalence, at least in terms of back pain, must be enormous, since about 10% of the world's population suffers from this type of pain [1]. Nevertheless, our understanding of the pathogenesis of DDD is still insignificant [1-10], and our ideas about the biomechanics of the human spine are dissonantly naive against the background of the rapid development of bionics and robotics. Such a conclusion is easily confirmed by considering three problems: 1) elucidation of the magnitude of axial load on the spine against the background of human behavioural activity; 2) the influence of prolonged sitting on the state of the lumbar spine; 3) the role of intra-abdominal pressure in reducing the axial load aims to clarify the situation regarding these issues.

The analytical review is devoted to clarifying the situation with these issues.

***Intervertebral discs***

The human spine is a unique structure combining strong anti-gravity resistance with significant flexibility. This combination is expressed structurally: the spine is a chain of alternately located rigid (vertebrae) and elastic (IVD) links. Both anatomical components have their own spectrum of pathology (although some types of pathology are common), and their proportion in the total height of the spine are believed to be in a ratio of 3:1 (in favor of the vertebrae). It is known that in the cervical spine, the height of the IVD in a person is ~2/5 of the vertebral body height, in the thoracic spine - ~1/5, in the lumbar spine - ~1/3 [11], and the human spine contains 6 cervical, 12 thoracic and 6 lumbar vertebrae [11].

Three parts are distinguished in the structure of the IVD - a thick fibrous ring covering the nucleus pulposus, and the so-called cartilaginous endplates at the border between the IVD and the bodies of two adjacent vertebrae [2, 6, 9, 11, 12]. The main (bio)chemical components of IVD are water, proteoglycans (aggrecan, biglycan, versican, decorin, lumican, fibromodulin), collagen and a number of other matrix proteins [3, 6, 12]. The average water content in mature IVD along the spinal axis is almost the same - 72–73% [13]. The difference in water content between the nucleus pulposus and the fibrous ring reaches a maximum up to 30 years of age, and disappears at the end of life [12,14]. The water content is closely related to the proportion of proteoglycans, it is the largest in the nucleus pulposus [15].

In healthy individuals, the fibrous ring, in addition to water, contains type I and III collagen (to a lesser extent type II), proteoglycans, and a small amount of elastin and other intercellular matrix elements [3,12]. Towards the nucleus pulposus, the content of proteoglycans, water, and type II collagen in the substance of the fibrous ring increases, while the content of type I collagen decreases [12]. The most hydrated part of the fibrous ring is the dorsal part [14].

Spatially, the fibrous ring consists of interconnected concentric layers (lamellae) of different thicknesses, only half of them are circularly closed [3, 12]. The direction of the strength fibers of type I collagen in the lamellae is different [12]. They are the ones that determine the resistance of the ring to stretching — the greatest, by the way, with simultaneous stretching along the entire perimeter [12]. Type II collagen, binding to proteoglycans, creates a water-retaining matrix ensuring the ring's resistance to compression [3,12].

Mature nucleus pulposus occupies about half of the volume of the IVD, consists of water (80–85%), proteoglycans (35–65% of the weight of dry residue), collagen (mainly type II, as well as types VI, IX, and XI), a small amount of elastin and other non-collagenous proteins [3, 12, 14, 16]. Aggrecan is the main proteoglycan and osmotic agent of the nucleus pulposus, an important inhibitor of the growth of blood vessels and nerves [3]. With age, the concentration of aggrecan in the nucleus pulposus decreases, but it increases in the inner part of the fibrous ring, therefore, in this period of life, this part of the fibrous ring is the most resistant to compression [3].

Each of the two (cranial and caudal) cartilaginous endplates is a thin (~0.6 mm) layer of hyaline cartilage tissue bordering the corresponding bony endplate [12]. They contain about 60% of water, type II collagen and proteoglycans, distributed across the width of the plate in varying ratios [12]. These parts of the IVD are capable of retaining water under a significant axial load, providing stability of the environment for nutrients diffusion into the IVD [12]. The plates likely do not possess linear elastic properties. [12].

With age, tissue stiffness of the fibrous ring increases, but due to the accumulation of small structural defects, it generally softens and weakens against the background of a decrease in the hydration of the nucleus pulposus, thinning of the IVD and weakening of the intervertebral ligaments - therefore, in general, stiffness when bending the spine with age and the development of the degenerative process decreases [12]. However, due to the osteophyte overgrowth, the range spinal motion does not increase, but decreases, and the proportion of direct transmission of the axial load by the fibrous ring increases [12].

Despite several technical challenges, it has been established that the nucleus pulposus remains an avascular part of the IVD throughout human life, the cartilaginous endplates and the fibrous ring have a branched blood network at the beginning of life, which subsequently decreases and increases again in the cartilaginous endplates and in the inner layers of the fibrous ring at against the background of their damage [17]. Thus, the main way nutrient supply to the cells of the central part of the IVD is passive selective diffusion from the vertebral bodies, better vascularized ligaments and outer parts of the fibrous ring [3, 17].

The main way of extracting energy in the IVD is anaerobic glycolysis with the formation of lactic acid [3], therefore, in the layer of the nucleus pulposus characterized by low oxygen level, a low pH level and a high lactate content are found. Since the vascularization, oxygen and glucose levels in the fibrous ring are higher than in the nucleus pulposus, the cell density is highest near the outer edge and lowest in the central part of the IVD [3].

Innervation of the lumbar IVD is carried out from three sources [18, 19]: 1) direct terminals of the anterior branch of the spinal nerve, 2) direct terminals of the autonomic connective branch, 3) terminals of the synuvertebral nerve. Usually, nerve fibers accompany the vessels of the IVD [18]. The main source of innervation of the IVD is the synuvertebral nerve (recurrent nerve of von Luschka), formed by the fibers of the anterior branch of the spinal nerve and the gray communicating branch [20, 21], responsible for sensory, proprioceptive, and sympathetic innervation of various parts of the fibrous ring [20], especially the posterior one [21] .

At the micro level, the following are found in the thickness of the IVD: 1) sensory nerve fibers spatially unrelated to the blood vessels of the IVD, 2) perivascular nerve fibers, 3) mechanoreceptors [19]. The fibrous ring is innervated most intensively. In conditionally intact IVD, nerve elements are detected mainly in the outer third, and with age or against the background of the IVD pathology, also in the inner two thirds of the ring and in areas of its damage [19]. Nerve elements are absent in

the thickness of the conditionally intact nucleus pulposus, their appearance is associated only with IVD pathology [19]. The endplates contain nerve fibers, the number of which also increases in the presence of DDD [19].

***General scheme of IVD function***

A smaller portion of the axial load is transmitted from the cranial to the caudal vertebra directly by the fibrous ring, while the larger portion is transmitted by a more complex mechanism involving the elastic "ball" of the nucleus pulposus. It is likely that the role of these two mechanisms of axial load transmission at various points of IVD is different (see [22]), and the pressure distribution in different parts of the nucleus pulposus (see below) largely depends on the level of its location, the presence of degenerative changes in it, the extent and axial load history [23]. It is also important that, at least in bovine IVDs, at high levels of axial load, the pressure in the nucleus pulposus does not affect load transmission through the IVD or its absorption of kinetic energy, and the fibrous ring, and not the nucleus pulposus, may play a crucial role in performing these functions [12].

In order for part of the axial load to be transmitted from vertebra to vertebra through the gelatinous "ball", this "ball" must have a good fibrous ring wall, which, when stretched, allows for rapid height variation (flattening) within a small range without loss of volume. With such an arrangement, the supporting function of the spine is maintained without compromising mobility. However, another, equally important problem that is solved in this way is "amortisation" in the sense of "channeling" a portion of the kinetic energy of the upper part of the body into the potential energy of elastic stretching of the fibrous ring, and then gradually into thermal energy. In the absence of such a means of "depositing" and slowly "channeling" the kinetic energy of the upper part of the body, it would immediately be transformed into energy sufficient to break the intermolecular or intramolecular bonds of the spinal substance, i.e. into the breaking and kinetic energy of the "scattering" of vertebral fragments, and then – into their (debris) thermal energy. Consequently, the "amortisation" mechanism actually reduces the power of transforming the kinetic energy of downward movement of the upper part of the body into the thermal energy of the support, protecting it (the support) from destruction. Since the redistribution of molecular deformations caused by a mechanical factor requires time, in the absence of this time, i.e., during rapid deformation, displacements occur between the focus of the deformation and its inert (in the sense of inertial) environment, which are out of bounds from the point of view of the stability of intramolecular and intermolecular bonds connections, i.e. destruction. In other words, if the mechanical deformity at this point increased gradually, it would have "managed" to induce small elastic deformities of the surrounding area, the wave of which would spread throughout the substance, having accumulated the mechanical energy of the primary external impact into the potential energy of elastic deformity of the substance. If the velocity of kinetic energy transfer at the point of contact with the substance is such that its movement in space is far ahead of the elastic movement of the local environment, then extra-boundary displacements and destruction occur between the point and the environment: the point of contact "detaches" and behaves independently within the substance regardless of interactions, which ensured its elastic connection with the substance. Therefore, the "channeling", albeit incomplete, of part of the kinetic energy of the impact of the cranial vertebra on the caudal one into the energy of the elastic deformity of the fibrous ring saves the contact zone of neighboring vertebrae from destruction. The energy accumulated in the elastic deformity of the stretched fibrous ring is returned in the form of kinetic energy of the upper part of the body ("elastic upward reflection"), kinetic energy of the more caudal vertebra, and thermal energy of the entire spinal segment. It is evident that the rate of extinction of such axial oscillations of the spine during a sudden axial jerk is high enough, i.e., the energy accumulated in the elastic deformity of the ring is discharged intensively, but not so intensively as to cause the vertebral and the IVD damage. This is the meaning of just described damping system of axial jerks, i.e. peaks of the axial load of the spine.

***Pathophysiology of DDD***

Given the conceptual ambiguity of the term "DDD" [1], this pathology of the IVD should be considered multifactorial. Factors and immediate causes contributing to its development include genetic background, age, characteristics of axial loading, obesity, various metabolic disorders, smoking, and even inflammatory processes within the IVD triggered by specific commensal flora in humans. [1, 4, 5, 8, 9].

The pathophysiological course of DDD involves the transformation of the intercellular matrix of the IVD:

1) increase in the content and alteration of the spatial distribution of type I collagen;

2) decrease in the content, alteration of the structure and spatial distribution of type II collagen;

3) decrease in the content and alteration of the structure of aggrecan in the nucleus pulposus [3, 6, 24].

This leads to a reduction in the volume of the nucleus pulposus, as well as to a decrease in the mechanical stability and elasticity of the fibrous ring [3, 6].

From a molecular point of view, disturbances in the metabolic activity and survival of the IVD cells, which are regulated by a number of growth factors, an increase in angiogenic activity and a decrease in the metabolic permeability of the cartilaginous endplates of the IVD, a change in the activity of the intercellular matrix remodeling apparatus, and development of a long-term local inflammatory process with all factor and by its cellular participants, secondary hyperinnervation of the IVD and sensitization of the nociception apparatus with diffuse spread of sensitization factors to adjacent segments, as well as morphological and functional changes in the brain against the background of chronic pain, are important for the formation of chronic pain [1, 3, 6, 7, 9, 10, 17, 20, 21, 25]. Furthermore, the long course of DDD leads to a decrease in the height of the IVD, hypertrophy of the surfaces of the facet joints and their capsules, a reduction in the diameter of the intervertebral foramina and compression of their vascular and neural contents, i.e., resulting in radicular symptoms [6, 21].

However, a decrease in the strength of the fibrous ring can lead to another, frequent and more dramatic version of the course of DDD: in case of sudden large radial destruction of the ring, part of the nucleus pulposus moves into the formed passage and, having gone beyond the boundaries of the ring or formally remaining within its boundaries, forms a hernia of the IVD, which compresses the located adjacent neural structures, causing, in addition to radicular pain, other neurological symptoms such as local sensory disturbances, motor dysfunction and autonomic dysregulation.

***Mechanical factor in the development of DDD***

In the scholarly and popular scientific literature, an important (if not determining) factor in the development of DDD is considered to be "mechanical" - the peculiarities of the axial load of the spine during human motor activity, when the state of the muscular apparatus of the trunk and the ligamentous apparatus of the spine does not correspond to this activity [5, 6, 8, 9]. Therefore, it is not surprising that finding out the range of values of axial load is a primary task in almost all studies of the biomechanics of the lumbar spine. Since direct measurement of the axial load would require replacing the entire IVD with an alternative support system equipped with appropriate sensors (a design resembling this concept can be found in — [26–30]), it is advisable to assess this parameter indirectly: by measuring the pressure in the center of the IVD using a puncture-installed miniature sensor and multiplying it by calculated area of IVD. The primary practical outcome of such studies should be the establishment of body positions in space and types of human behavioral activity, at which the intradiscal pressure is maximum, and these positions and activities are the most dangerous in terms of triggering the degenerative process in the IVD.

Currently, the methodology for measuring intradiscal pressure has been tested both *ex vivo*, on explants of spinal motion segments [31], and *in vivo* [32]. The results of *in vivo* study of pressure in the lumbar and lower thoracic IVD are given in a number of well-known and frequently cited papers: A. Nachemson and J.M. Morris (1964) [33], A. Nachemson (1965) [34, 35], A. Nachemson (1966) [36], A. Nachemson and G. Elfstrom (1970) [37], H. Okushima (1970) [38], A. Nachemson and G. Elfström (1973) [39], A. Nachemson (1975) [40], A. Nachemson (1981) [41], A. Schultz (1982) [42], B.J.G. Andersson et al. (1974) [43–46] (see also [47]), K. Sato et al. (1999) [48], H.-J. Wilke et al. (1999) [49], H.-J. Wilke et al. (2001) [50], D.J. Polga et al. (2004) [51], I. Takahashi et al. (2006) [52]. The pioneering work carried out by A. Nachemson provided an understanding of the range of values of axial loading on the lumbar spine during normal human behavioral activity. Research by other groups generally confirmed these findings, although A. Nachemson later had to take into account technical shortcomings of his method and adjust the obtained results [53].

It is now understood that limitations of these studies include a small sample, the lack of a standard list of positions in which measurements were made, and a number of engineering and technical difficulties [51, 52, 54, 55]. Therefore, a qualitative comparison and generalization of data obtained is impossible, and the reliability of practical conclusions is questionable. For example, M. Dreischarf et al. (2016) [53] cite all the available data on pressure in the IVD in the lying supine, lying prone, and lying on side positions, as well as in standing position (probably refers to the standing position freely) and unsupported sitting (without specifying the position of the hands). Regarding the pressure in the lower lumbar IVD[1], we have the following figures:

- for the supine position, the pressure in the IVD $L_3$-$L_4$ – ~0,14 MPa [37], in the IVD $L_4$-$L_5$ – ~0,1 MPa [49], ranging generally from ~100 to ~140 kPa;
- for the prone position, the pressure in the IVD $L_3$-$L_4$ – ~0,13 MPa [37], in the IVD $L_4$-$L_5$ – 0,11 MPa [49] or 0,09 MPa [48], generally ranging from ~90 to ~130 kPa;
- for the lateral position, the pressure in the IVD $L_3$-$L_4$ – ~0,33 MPa [33], in the IVD $L_4$-$L_5$ – ~0,28 MPa [33] or 0,12 MPa [49] or 0,15 MPa [48], generally ranging from ~120 to ~330 kPa;
- for the unsupported sitting position, the pressure in the IVD $L_3$-$L_4$ – ~1,0 MPa [33], ~0,9 MPa (possibly, [40]), ~0,77 MPa [37], ~0,7 MPa [38], ~0,32 MPa [42], the pressure in the IVD $L_4$-$L_5$ – ~0,87 MPa [33], ~0,84 MPa (possibly, [40]), ~0,81 MPa [38], ~0,46 MPa [49], ~0,62 MPa [48], generally ranging from ~320 kPa to ~1 MPa;
- for the standing position, the pressure in the IVD $L_3$-$L_4$ – ~0,62 MPa [33], ~0,44 MPa [37], ~0,58 MPa [38], ~0,33 MPa (possibly, [45]), ~0,27 MPa [42], the pressure in the IVD $L_4$-$L_5$ – ~0,59 MPa [33], ~0,48 MPa (possibly, [40]), ~0,64 MPa [38], ~0,49 MPa [49], ~0,54 MPa [48], ~0,35 MPa [52], generally ranging from ~270 to ~640 kPa.

According to the generalization of N. Newell et al. (2017) the pressure range in the nucleus pulposus of the healthy lumbar IVD is 91-539 kPa in supine positions, 500-870 kPa in standing positions and 460-1330 kPa in sitting positions. The maximum pressure in the nucleus pulposus of the lumbar IVD recorded in studies of this kind – 2300 kPa was observed during lifting a load weighing 20 kg with straight legs, bending forward with a "rounded back" [49], which is the reason for excluding this method of lifting loads.

Rare but interesting results of pressure registration in the lumbar IVD against the background of lifting a very heavy weight are known. For example, when weightlifter performs a "clean+jerk" [56] with barbell weighing more than 150 kg, at the level of the lower thoracic IVD, an axial load of more than 9500 N may occur [57], and therefore, at the cross-sectional area of the lower lumbar IVD is about 22 $cm^2$ [58] the intradiscal pressure should be 4.3 MPa. When performing a deadlift of 335 kg, the calculated axial load on the lumbar IVD should be 36,400 N [59], i.e., with the indicated cross-sectional area of the lower lumbar IVD, the intradisc pressure should be 18 MPa. This is probably not the limit, since the current deadlift record is 501 kg ("elephant bar deadlift"; H. Björnsson) [60] or 549 kg when pulling a barbell 15 inches from the floor ("hummer tire deadlift"; O. Novikov) [61], or 580 kg when pulling a barbell, the bar of which

[1] Some data are provided based on the diagrams available in the article by M. Dreischarf et al. [53], with reference to the original source.

is at rest at a height of 18 inches from the floor ("silver dollar deadlift"; R. Heinla) [61].

For comparison, here are some known pressure values in nature or in everyday life [62]:

- earth's atmospheric pressure above the sea surface – ~101.3 kPa;
- the pressure in the tyres of passenger cars during normal operation or at rest is 180-250 kPa higher than the atmospheric pressure;
- pressure between the contacting surfaces of human teeth at average biting force - 1.1 MPa;
- water pressure near the remains of the Titanic is 40 MPa;
- water pressure at a depth of more than 6.5 km – >69 MPa;
- the maximum pressure in the chamber when firing a gun is 70–280 MPa;
- water pressure at the bottom of the Mariana Trench is 110 MPa.

Therefore, the probability of the existence of colossal values of intradiscal pressure in humans is quite real and at the same time improbable due to the much smaller range of mechanical stability of the vertebra. This situation requires an explanation, at least through the development of "conscious" mechanisms of natural lumbar spine load relieving [63–65 etc.], which we will further explore.

### *Self-loading, or the price of elegant bipedalism*

The greatest axial load on the lower lumbar vertebra is associated with lifting a load in a standing position with the body bent forward and legs straight. The reason for this is the enormous "additional loading" of the spine by the pull of the back extensors, as evidenced by the very first and, in our opinion, generally relevant biomechanical model of the spine - the "cantilever model" [63, 65–68, etc.]. The force vector of lifting the trunk (together with the head and neck) from the forward-leaning position is perpendicular and directed backward from the surface of the back. It is formed due to the presence of a lever "counter-arm" - a certain distance between the conventional middle of the largest, caudal thickness of the mass of the erector spinae muscles (primarily *m. erector spinae*) and the posterior surface of the sacrum. Due to the elegance and slenderness of the human anatomy at the $L_5$ level, the thickness of the *m. erector spinae* in an adult average man is only about 5 cm [69], that is, half of it is about 2.5 cm. With such an insignificant "counter-arm", a sufficient magnitude of the lifting vector of the trunk leaning forward is formed by the enormous tension of the back extensor muscles, and the main part of the result of this tension is accounted for by the vector component directed caudally along the spine. In other words, the back extensor muscles forcefully "additionally load" the spine, compressing it along the axis, in order to hold, lift or extend the trunk from a forward-leaning position. If these muscles were attached to the pointed back spine of the sacrum with a length of, for example, 20–30 cm, and preferably 50 cm, there would not be such a colossal "additional loading" of the spine. However, our slenderness and fitness in such a case would be nothing to dream about, and a person would resemble at best a stegosaurus. So, elegance comes at a price.

A similar situation with extensor "additional loading" occurs when holding a large weight asymmetrically. Carrying a 20-kilogram box with one lowered arm is accompanied by a pressure in the $L_4$-$L_5$ IVD of about 1 MPa, while carrying two such boxes (total weight of 40 kg) symmetrically in two lowered arms is 0.9 MPa [50]. Therefore, when carrying one box, the contralateral spinal flexors "additionally load" it by 100 kPa more than holding a similar box with the contralateral arm. For comparison: when lifting a 20 kg box from the ground between the legs with the back as straight as possible and knees bent and spread to the sides, the pressure in a similar vertebra was 1.7 MPa, when lifting the same box from the ground with a bent forward "rounded back" and straight legs - 2.3 MPa, when lifting two similar boxes from the ground (each box - with one hand) with a squat between them - 2.1 MPa [50].

### *Sitting and intradiscal pressure*

One of the "legendary" issues associated with this array of data is the negative impact of prolonged sitting on lumbar spine health, since the pressure in the lumbar IVD seems apparently greater in this body position, than when standing upright [54, 55, 70]. However, such a belief remains hypothetical. Thus, A. Claus et al. (2008) [54], after analyzing the available data, came to the conclusion that the pressure in the lumbar IVD when standing and sitting unsupported seemed similar, and if sitting is indeed an important factor in the development of low back pain, it is not due to the mechanism of increasing intradiscal pressure. It should be noted that the authors, while comparing the primary empirical data, did not take into account such an important point as the position of the arms while sitting, which varied across different studies of intradiscal pressure. Another overlooked factor is the condition of the volunteers' backs in the sitting position - stretched or relaxed. Finally, in our opinion, the fact that A. Claus et al. included in the analysis the results of intradiscal pressure measurement using manometers incorporated into the system of rigid internal fixation of the spine, established for medical reasons [26–30]. Nevertheless, in general, according to the observation of A. Claus et al. (2008) [54], the pressure in the lumbar IVD during standing and sitting is in the range of ~0.5–0.6 MPa, while the interindividual variation of this indicator is ~0.2–0.3 MPa. Under such conditions (statistical analysis of this heterogeneous material is impossible – *V.M.*) it is clear that the difference in the values of the evaluated parameter for the two specified body positions is probably insignificant. Also, according to A. Claus et al. (2008) [54], epidemiological data do not demonstrate a convincing association between prolonged sitting and the manifestation of low back pain. This finding is generally consistent with the results of a recent meta-analysis by D. De Carvalho et al. (2020) [70]: substituting sitting work for standing work does not reduce the risk of low back pain manifestation. However, we did not find in the work of D. De Carvalho et al. taking into account the possibility of leaning with elbows or forearms on a high stand while working in a standing position, and such a maneuver can significantly reduce the axial load on the spine.

A recent study by J.-Q. Li et al. (2022) [55] adds uncertainty: according to the meta-analysis of empirical

results obtained before the 1990s, sitting appears to be associated with higher pressure in the lumbar intervertebral discs than standing, but for discs with signs of degeneration, as well as for studies conducted after 1990, there is no significant difference in the evaluated parameter between the two positions. As in the work of A. Claus et al. (2008) [54], we were unable to ascertain whether J.-Q. Li et al. paid attention to the state of the back while sitting on a stool and to the position of the volunteers' arms in the papers they analyzed. In addition, the authors, like A. Claus et al. (2008) [54], involved in the analysis the results of intradiscal pressure measurement with manometers incorporated into the system of rigid internal fixation of the spine [26–30]. Finally, J.-Q. Li et al., referring to the results of T.A. Beach et al. (2005) [71], concluded that regardless of which of the compared body positions is accompanied by higher intradiscal pressure values, prolonged stay in any of them is not recommended due to the possible development of lower back pain or (we may add – *V.M.*) of the degenerative process in the lumbar spine.

Given all of the above, the question arises: are professional drivers and representatives of other professions more prone to the manifestation of DDD? And also, how can the significant sexual dimorphism in lumbar lordosis [72] be related to the gender-specific epidemiology of low back pain [1]?

***Intra-abdominal pressure***

The issue of increased pressure in the lumbar IVD while unsupported sitting and hand support compared to standing is closely related to another "legendary" problem in clinical biomechanics of the lumbar spine – the "load relieving" effect of the strained belly, or, to be precise, an abdominal cavity filled with internal organs.

R.M. Aspden (1987) [65] associates the origin of the idea that the abdominal cavity filled with viscera, under the condition of active compression from everywhere, turns into an elastic body that can transfer a certain part of the weight of the supradiaphragmatic half of the body to the pelvic bones, bypassing the lumbar spine, with the related ideas formulated 100 years ago by A. Keith (1923) [73]. The idea of the " load relieving role" of the tense abdominal cavity was (and still is) supported by a number of authors [63, 64, 67, 74, etc.]. Various measurements of intra-abdominal pressure allow estimating the physiological value of this indicator at the level of ~0.24 kPa in the supine position [75] to ~27 kPa during the Valsalva maneuver [76], and during weightlifting procedures with air retention – from ~11 kPa against the background of bench press to ~45 kPa while the deadlift of the barbell weighing 305 kg [64, 77]. Maximal abdominal straining by a trained weightlifter leads to an increase in intra-abdominal pressure up to ~56 kPa [64]. If the area of the diaphragm effective for generating the "load relieving action" of intra-abdominal pressure in this case was ~300 $cm^2$ (see [66, 78]), according to the simplest calculation, the value of the cranially directed "load relieving action" of this factor should be ~1700 N. Moreover, the use of an athletic belt during heavy lifting is likely to increase intra-abdominal pressure [67, 79, 80].

Some authors [65, 66, 78, 81, etc.] criticize the hypothesis of the "load relieving effect" of intra-abdominal pressure, noting that the tension of the abdominal wall muscles, attached at one end to the costal arch and the other to the upper border of the pelvis, not only increases the axial load on the lumbar spine, but also creates an additional bending moment, which must be compensated for by more tension of the back extensor muscles, which "additionally loads" the lumbar spine. Other arguments against the hypothesis of the "load relieving effect" of intra-abdominal pressure are the dangerous effect of intense coughing and sneezing on the (damaged - *V.M.*) lumbar spine [66], as well as the incomplete temporary overlap of the period of increased intra-abdominal pressure with the duration of the lifting process [65]. In this regard, alternative views on the role of intra-abdominal pressure in the biomechanics of the lumbar spine have been proposed:

1) a tense abdominal cavity creates a convex surface for the spine to "stretch" against, thus relieve load of the spine [65];

2) increased intra-abdominal pressure stabilizes the entire abdomino-lumbar part of the body and reduces the likelihood of excessive displacements and injuries of the IVD [74, 81–84];

3) limitation of bending in the lumbar spine reduces the necessary tension of the extensor muscles and their additional loading of the spine [67].

In our opinion, an increase in intra-abdominal pressure in certain situations is important for reducing the load on the lumbar spine, because no alternative version that would receive further mechanical and physical development has been proposed, and some authors reasonably consider the mechanism of intra-abdominal pressure generation by the tension of muscles that do not cause an "additional loading effect" – the transverse abdominal muscle [63, 85] and diaphragm [84, 86]. The activity of these muscles against the background of motor acts better correlates temporally with the value of intra-abdominal pressure [84].

***Narrow pathways of spinal biomechanics***

The issue of calculating the actual load on the lumbar IVD in a static position and in dynamic, considering all determining and influencing factors, remains unresolved. Attempts to solve it are ongoing [50, 53, 68, 78, 87–102], but, in our opinion, they refer to very simplified situations and involve a still too short list of initial conditions and factors. The reason is rather limited ideas about the structure and mechanical parameters of the components of a typical motor segment of the spine, an even greater simplification of these ideas when described by mathematical means, and the low capacity of computing devices that calculate the necessary biomechanical parameters on the basis of the created models. Nevertheless, it is quite obvious that the increase in intra-abdominal pressure in real conditions is caused by various combinations of the activity of the muscles surrounding this cavity - the diaphragm, a number of muscles of the abdominal wall, ventral muscles of the lumbar spine and pelvic diaphragm muscles. Which of these combinations and to what extent results in "additional loading" or "load relieving" of the lumbar spine is unknown. Therefore, how the "load relieving", "additional loading", "stabilizing"

and possible other effects of intra-abdominal pressure are combined against the background of a certain motor or late-tonic activity will probably not be revealed soon.

By the way, if the "load relieving" effect of intra-abdominal pressure against the background of normal human activity exists, then in a sitting position freely and unsupported sitting, this factor should be eliminated. Probably, in the sitting position, not only the configuration of the spine and thorax changes, but also the volume of lung ventilation is significantly reduced due to a decrease in the tone of the diaphragm: this is obvious, since any loud phonation, for example, singing, is most effectively performed in a standing position with a straight back. In addition, in the sitting position, due to the straightening of the lumbar lordosis, the costal arch approaches the upper edge of the pelvis. Apparently, this straightening of the lumbar lordosis is the result of the spine acquiring the most ergonomic position, i.e. one requiring the least muscle activity, while sitting. Due to the reduced distance between the upper and lower attachment points of the rectus and oblique muscles of the anterior abdominal wall, its significant tension is impossible: in a sitting position without a stretched back, the anterior of abdominal wall is usually relaxed. It is also evident that in such a situation, the volume of the abdominal cavity must decrease, which is compensated by the movement of part of its contents forward, in the direction of the relaxed anterior abdominal wall and upwards, i.e. into the lower part of the chest, due to the relaxation of the diaphragm. Under such conditions, a significant increase in intra-abdominal pressure is possible only when straining after a deep breath and with a straight back — that is, it is significantly hindered. Therefore, under normal conditions, outside of known viscero-somatic acts, during sitting at rest, the "load relieving" effect of intra-abdominal pressure should be absent, which theoretically could be associated with an increase in the axial load on the lumbar spine, provided there is unsupported sitting and/or elbows support on one's own knees in such a spatial position.

***Diurnal changes in the IVD hydration***

It is worth mentioning the daily changes in the hydration of the IVD [103, 104], and consequently, the volume and height of the IVD [49, 103, 104]: during prolonged axial load (until the end of the working day), hydration and height of the IVD decrease, while during prolonged rest in the supine position (typically at night) - increase. P. Leatt et al. (1986) [106] report a daily fluctuation in height, likely associated with changes in spinal height, i.e., IVD height, of approximately 14.4 mm. According to A.R. Tyrrell et al. (1985) [105], such fluctuations amount to ~19.3 mm (1.1% of growth), with 54% of height loss occurring during the first hour after morning awakening and regular vertical behavioral activity, and 70% of the height loss being recovered within the first hour regular nighttime sleep. H.-J. Wilke et al. (1999) [49], during a single observation of a volunteer, found that the pressure in the $L_4$-$L_5$ IVD increases during regular 7-hour sleep from ~0.1 to ~0.24 MPa. The authors note that this is consistent with the concept of a higher likelihood of lumbar IVD prolapse during morning physical activity [107], since during the first three hours after overnight sleep, forward bending of the trunk requires greater mechanical efforts and therefore results in a greater stress effect on the IVD [108, fig.6, fig.7]. Consequently, maximizing lumbar flexion removal from morning physical activity may reduce the manifestation of lower back pain [109, 110].

These data are consistent with experimental observations demonstrating that multiple pulsatile axial loading of a mature lumbar IVD of a domestic goat of equal force with a frequency of 1 Hz is accompanied by a gradual decrease in intradiscal pressure against the background of each subsequent pulsatile peak of a similar load and a decrease in the IVD height [111]. In other words, the axial preload of the IVD may reduce the increase in intradisc pressure during the subsequent axial load.

At the same time, the pattern of hydration changes in various parts of the lower lumbar IVD against the background of vertical activity remains unclear J. Kraemer et al. (1985) [14] on a model of prolonged axial load of the lower lumbar IVD of a person (although without specifying the biomedical condition of the studied IVD) found a greater loss of water by the fibrous ring (11%) than by the nucleus pulposus (8%). However, according to magnetic resonance imaging, the T2 signal characteristics from the fibrous ring of the lumbar IVD increase after daytime exercise indicating an increase (rather than a decrease) in its hydration against the background of the expected decrease in the hydration of the nucleus pulposus [103].

***Conclusions***

Despite all efforts and peak activity during the 1970s-1980s, the major issues in clinical spine biomechanics remain unresolved. It can be noted that the study of intradiscal pressure against the background of human behavioral activity has been and continues to be disproportionately underrepresented in research. Unfortunately, due to the limitations in visualization and technical reliability, these studies have not been able to accumulate a sufficient empirical base for statistically significant conclusions. Therefore, a current challenge is the development and use of more accurate, reliable, and miniature techniques for monitoring intradiscal pressure, which would allow the assessment of this parameter in large samples of volunteers with conditionally intact IVD and in the presence of pathology. Additionally, measuring intradiscal pressure should ideally be combined with recording the activity of muscles important for "additional loading" of the IVD. Similarly, studies on the phenomenon of intra-abdominal pressure require improvement to clarify under what conditions and by what mechanisms this factor may affect the magnitude of axial spine load.

The practical result of such research should be the identification of the most dangerous types of human motor activity for provoking and developing DDD. Furthermore, there is hope for expanding our understanding of the biochemical composition, vascularization, and innervation of IVD, as well as the biology and pathology of IVD cells, which would enable the construction of more insightful models of spine biomechanics. The solution of these problems, in our view, will significantly approach the understanding of the key drivers of DDD pathogenesis, and consequently, to improving the means of prevention and treatment of this widespread pathology.

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*Conflict of interest*

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