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Are adolescent idiopathic scoliosis and ankylosing spondylitis counter-opposing conditions? A hypothesis on biomechanical contributions predisposing to these spinal disorders

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ABSTRACT

Human spinal biomechanics are profoundly complex and not well understood, especially in terms of the dynamic spine function. Translation of biomechanics to disease is difficult, particularly since cause must be separated from effect. Primary dynamics predisposing to the onset of chronic spinal disorders, e.g., adolescent idiopathic scoliosis (AIS) or ankylosing spondylitis (AS), must clearly be differentiated from secondary alterations.

This commentary addresses primary biomechanics that may predispose to these idiopathic diseases. A novel hypothesis is proposed, based upon inferences regarding their contrasting muscular dynamics. The hypothesis postulates opposing inherent muscle tonicity in AIS versus AS. Converse degrees of spinal stability may predispose to the respective curvature deformities of AIS and the enthesopathy lesions of AS. One condition is suspected to counter-oppose the other, within a polymorphic spectrum of spinal stability.

The challenges of causation studies in AIS and AS

The onset risks and severity gradients of polymorphic disorders, such as adolescent idiopathic scoliosis (AIS) and ankylosing spondylitis (AS), result from complex interactions among multifactorial determinants (1-7). Biomechanical variations which initially cause deviations from normal physiological control may differ from secondary pathological processes which supervene in the fully expressed disorders (1, 3).

Further difficulties encountered in investigations of causation are the overlapping boundary limits between the spectrum of clinical features in these diseases and the ranges of normal phenotypes. The Scoliosis Research Society has defined scoliosis as a lateral curvature of the spine of greater than 10 degrees, as measured by the Cobb method on a standing coronal plane radiograph (3, 8, 9). Such degrees of curvature have been reported in about 1 to 3 percent of otherwise healthy adolescents (8, 10). Lesser degrees of curvature occur more frequently and are considered to be "normal" developmental variations (8, 11). The spinal deformities in scoliosis are three-dimensional however, and the complex patterns challenge two-dimensional roentgenographic classification (12, 13).

Scoliosis (G. Skōliosis, a "crookedness") was first described by Hippocrates (14). Clinically, scoliosis is a descriptive term used to describe a structural (i.e., fixed) deformity of the spine, characterized by lateral curvature and rotation of involved vertebral bodies to the convex side. However, coronal plane (lateral) curve flexibility occurs in AIS (15). The curves may be significantly reduced by eliminating the effects of gravity, by side-bending the spine actively when supine, exerting head-pelvis traction (14,15), or by employing a bending-brace (2,16), as well as ultimately reducing curves by surgical correction (17). A non-structural (postural) scoliosis curve corrects on side-bending or traction films (18). Marfan syndrome is an example of secondary, usually lumbar, scoliosis (1, 19).

The difficulty in applying complex biomechanical concepts of spine instability to the etiology of AIS lies in the lack of a demonstrable link between spine buckling and scoliotic deformity. Buckling is the loss of structural stability causing a momentary deformation, while AIS is a chronically evolving deformity. Nevertheless, scoliotic deformity could be the expression of passive accommodation to many repeated instability events stemming from insufficient muscular support.

The essential role of muscles in stabilizing the spine and controlling its dynamic functions are unquestioned (20-28). Nevertheless, such normal intrinsic supportive mechanisms are conventionally interpreted within a static configuration and variously described in biomechanical terms as stiffness (i.e., resistance to deformation and displacement), stability (i.e., the ability to return to the equilibrium state), forces (i.e., restraining versus displacing movements), or other phenomena, alone or in combination (20).

In normal balanced posture, muscular support of the spine is often described

physiologically in terms of coordinated reflex activation (25, 29-31), concentric, eccentric, or isometric strength (32, 33), length dependent passive elastic energy (22, 34), thixotropic properties (35-37) or tonicity (7, 29-31, 35, 38).

Such complex mechanisms cannot be expected to be accurately expressed by any single biomechanical or biological term. Nevertheless, the clinical term axial muscular tone (tonicity) is used for purposes of simplification and unification of the hypothesis that bridges the contrasting biomechanics of AIS versus AS, within the one dimension of spinal stability. Muscle tone (and tension) connotes stiffness or tightness of the muscle and its moving part (7, 30, 31, 35, 38).

Without the essential supportive role of muscles, the load which can be placed upon an isolated osteoligamentous thoracolumbar spine before buckling is 20N (about 4.5 lbs) of compressive force (i.e., less than the weight of the head), which is its critical buckling load (39). Under balanced posture and light loading conditions, it has not yet been determined if the critical column stability is mainly provided by the thixotropic properties of muscle stiffness, i.e., unaccompanied by EMG amplitude changes (31, 35), or by low, continuous muscle activation of synergist and antagonist groups (25). Muscles act as “guy wires” and also contribute compression, thereby stiffening the column in all degrees of freedom and enhancing stability (37, 40).

The human spinal system has an exceedingly complex, evolutionary, multi-segmental architecture (41). Recently, principles of “biotensegrity” have been proposed to explain spinal structure and functions, which posit that forces operate primarily via continuous networks of tensional tissues (e.g., axial muscles and ligaments) and secondarily incorporate compressional loading into local islands, e.g., the bony elements of the vertebral column (7, 41).

Optimal or sufficient spinal structural stability of course is desirable for all normal tasks (42). Lesser stability permits hypermobility, but possibly at a greater risk of buckling behavior (37), as may occur in AIS. In contrast, greater spinal stability induces increased stiffness and may predispose to excessive tensional stresses, particularly at attachment sites, as may occur in AS (7). Clinically, a spine may be considered stable when it is maintained in an optimal state of equilibrium by its restraining structures during varied functions (28,42). Biomechanically, it is assessed by its degrees of stiffness,mainly under momentary, minor perturbations and static conditions (43).

Flexibility of the spine has been quantitatively analyzed in AIS (15). Ranges of motion of the back (44) and subjective symptoms of tightness (4, 7, 45) are routinely evaluated in AS. Rotation of the thoracolumbar spine was the clinical test of spinal mobility in AS that best correlated with the duration of disease (44). However, biomechanical measures of stability, in terms of quantitating forces required to disturb spinal equilibrium, have not been performed in either condition.

Complex biomechanics of truncal musculature

Muscle stiffness is related to muscle force. Intrinsic muscle stiffness is a mechanical property which stabilizes the balanced spine in response to a variety of small perturbations, without the need for active motor control adjustment (34, 43). In the balanced upright posture under static conditions, as little as 2% of the maximum contraction effort on average is sufficient from all trunk muscles to maintain a stable spine (25). A reduction in either muscle tonus or inherent neuromotor activity will result in spinal instability and buckling. Larger spinal perturbations elicit muscle stretch reflexes; such responses further stabilize or stiffen the spine and prevent its buckling failure (46-48). However, such reflexive muscle activation and de-activation must still conform to a coordinated recruitment of muscle forces; otherwise, structural instability could also occur (37). Thus, sufficient intrinsic muscle tonus as well as the appropriate active contractile reflex responses regulated by an intact motor control system are the necessary mechanisms to satisfy normal spinal stability (49).

Muscle tone (or tension) is essential for maintaining body posture (7, 30, 31, 41). Although it is influenced by the stretch reflex, little is known about the full processes controlling this mysterious aspect of human physiology (29-31, 35, 38). Overall tone results both from elastic muscular contractile properties, which do not require motor unit action potentials (MUAPs), and from electrogenic [i.e., electromyographic (EMG)-active] contractions, which may be either voluntary or involuntary (i.e., either reflex responses or "spasms") (30, 31). Also, giant protein molecules (titin/connection and nebulin) contribute viscoelastic properties to intrinsic muscular tone (50).

Intrinsic muscle tone and joint mobility are complex and associated physiological traits. Clinicians currently recognize individuals who have hypermobility syndrome (HMS) (51), also referred to as joint or ligamentous laxity. Importantly, muscular hypotonicity has been demonstrated in hypermobile women (52). Studies of joint laxity or soft-tissue extensibility have been reported in AIS (53-55) and in one adult case of AS with hypermobility (56). No comparative data on hypermobility were found in AIS versus juvenile AS (JAS) patients.

Peripheral joint mobility was found to vary considerably in the population, depending upon age, gender, and the side of the body involved (57). Like the peripheral joints, the normal range of spinal mobility in lateral bending was found to be greater in females than males (58). Physiological ranges of muscular tonicity, especially of the trunk, are most difficult to quantitate accurately in humans (38, 59). Such limitations greatly complicate scientific investigations of muscular tonicity in AIS, AS, or other rheumatic disorders. Like many other common physiological traits, such polymorphic characteristics are suspected to manifest in nature in relatively normal or lognormal frequency distributions (8, 10, 57, 58).

Features implying contrasting alterations of spinal stability in AIS versus AS

The most common form of idiopathic

Table I. Clinical features of AIS: Host and biomechanical influences.

- AIS composes circa 85% of all idiopathic scoliosis (IS) in childhood.
- A younger onset of idiopathic scoliosis heralds greater spinal deformity.
- With younger age in childhood, both the severity of new onset IS and the degrees of hypermobility in the population increase, implying a relationship to muscle tonicity.
- The spurt of spinal growth during puberty increases the risk of developing AIS.
- Idiopathic scoliosis rarely develops following the stage of skeletal maturation.
- Femaleness is significantly associated with greater spinal deformity and progression.
- The primary curvature is mainly thoracic (80%), and less often lumbar (20%).
- The primary thoracic curvature is typically convex to the right side in both sexes, and is influenced by handedness.

scoliosis (IS) is AIS (Table I), which is detected between the age of 10 years and the stage of skeletal maturity (17, 18). It is estimated to comprise almost 90% of all childhood cases (1,9, 17, 18, 60). In AIS, major curve(s) occur far more commonly in the thoracic (circa 80%) than the lumbar (circa 20%) spine, and occasionally in both. The thoracic curvatures in AIS are predominantly (80+ percent) convex to the right (1, 60-62), unlike the equal dominance of right and left curvatures seen in paralytic scoliosis (60, 63).

With respect to our hypothesis, secondary scoliosis often results from numerous myopathic or neuromuscular deficiencies that provide insufficient support to the spine (1-3, 9, 18, 29, 60, 63, 64). Entities such as poliomyelitis, cerebral palsy, muscular dystrophy, spinal cord injuries, hypotonia, or dystonia often cause secondary spinal curvatures (1-3, 9, 18, 29, 60, 63, 64). Occult intraspinal pathologies or neuroanatomical abnormalities have been detected on magnetic resonance imaging (MRI) in 20-25% of juvenile "idiopathic" scoliosis (JIS) patients (65, 66). The authors indicated that MRI should be routine in the evaluation of patients with scoliosis of "juvenile onset", i.e., curvatures detected between ages 3 and 10 years. Such reports suggest that muscular hypotonicity or other biomechanical deficiencies of spinal support, either inherent or due to myopathic diseases, may predispose to scoliosis.

In the literature, little or no attention has been given to the possibility that inherent polymorphic spinal muscular insufficiency may be a primary predisposing factor to the onset of AIS. However, one group did mention symmetrical spinal muscular weakness as a theoretical cause of asymmetrical deflections resulting from instability (22). Another biomechanical group (67) stated that rotational and translational instability, alone or in combination, always occur in scoliosis patients, but made no reference to the causation of IS.

The overriding concept proposed in the literature on the muscular biomechanics of AIS is muscular asymmetries contributing to buckling, or a neuromotor imbalance resulting in primary deforming forces or otherwise causing dysfunctional control (1, 3, 11, 68-72). A review of current concepts by the Scoliosis Research Society Etiology Committee (3) stated, “There is no strong evidence implicating any particular biomechanical factor in the etiology of idiopathic scoliosis”.

In contrast to rare studies of predisposing risk mechanisms, considerable research has been done on biomechanical (3,72), clinical (2,3,17), electromyographic (29), and histochemical or pathological (1,3,68, 69,71) aspects of muscle once scoliosis has presented and progressed. Unfortunately, such results probably do not reflect the primary alterations of axial musculature that predisposed to the initiation of the deformity (1, 3). Rather, they are believed to result from secondary muscular responses to the altered biomechanics of the spinal deformities (1,3, 71). Furthermore, AIS is a developmental disorder. A critical insufficiency that may have initially predisposed to instability at a critical period of musculoskeletal immaturity and axial growth, e.g., during the pubertal spurt in AIS (2, 70, 73, 74), may not necessarily persist after further maturation and muscle strengthening (17).

Musculature, particularly truncal, is significantly more developed (74, 75) and stronger (74, 76) in adolescent and adult males than females. In adolescents with mild lateral curvatures, e.g., 10 degrees or less, the female to male (F:M) sex ratio is equal. However, with either lateral curvatures of 30 degrees or greater or in those persons with progressive disease, girls predominate over boys in a ratio of four-fold or greater (1,2,10,12), which is the direct opposite of AS (4).

Despite their deformity, most patients with AIS live normal lives, and usually function without limitations or pain (2, 62, 72), unlike patients with AS (7, 77). Those who reach skeletal maturity with thoracic curves of less than 30 degrees are unlikely to worsen as adults (1, 2, 62), unlike the progressing course of radiographic spinal involvement in juvenile or adult AS (77). The younger the onset age, the more advanced and progressive is the course of IS (1, 62). Available data imply that younger onset ages result from a greater constitutional deficiency of axial muscular support of the spine, as in many diseases with a hereditary predisposition. In addition, the relative protection of males only begins to express in older JIS cases,and is fully evident in adolescent onsets (2, 17, 18).

The significant influence of younger onset age upon severity is also noted in the period prior to, versus following, the pubertal growth spurt of AIS patients (73,74,78). The pubertal spinal growth spurt of adolescents, i.e., during a population mean age of circa 12 in girls and 14 in boys (74), is an independent risk factor from gender for the development of AIS (2, 70, 73). Perhaps the more rapid axial elongation of the spine during that growth phase may result in a relative instability of muscular support (70) ? A smaller curve (i.e., 10-19 degrees) in a more mature adolescent has a very low (1.6%) probability of progression, whereas a larger curve (i.e., 20-29 degrees) in a less mature adolescent has a high probability (68%) of progressing (17, 78). Only infrequently does AIS begin after the stage of skele-

tal maturation (2,17), unlike the risk of developing AS (4).

As indicated, the thoracic curvature in AIS is characteristically convexed to the right (1, 2, 17, 61). Left thoracic patterns are less likely to occur in IS than in secondary scoliosis from underlying neuromotor disease (63, 79). In 254 girls with IS, the direction of the convexity significantly correlated with preferred handedness (61). Analogously, adolescent athletes who engaged in stressful sports involving predominately one arm were reported to have had minor curvatures convexed to the respective side (80-82). Such associations of curve laterality with handedness suggest that biomechanical and behavioral factors contribute to the pattern of spinal deformity (61, 80-82). A pilot study utilized progressive resistive torso rotation strengthening as a treatment for AIS (83). Curve reduction was noted in 16 of 20 patients, and no increase in severity was found in any patient during a twice-weekly, 4-month exercise regimen. Such data are preliminary and uncontrolled, but may infer insufficiency of torso rotational strength as a predisposing factor in AIS.

The etiologies of AIS (1-3, 84) and AS (4-7) remain unproven, although their clinical epidemiology and disease courses are well described. Such theories cannot be reviewed critically in this commentary. Although, the genetic (1, 3, 5, 6, 84) and host (2, 4, 6, 7, 17) contributions to these diseases clearly differ, they both centrally involve the spinal system. Accordingly, biomechanical alterations were suspected to be primary predisposing factors for these spinal disorders.

A novel hypothesis was recently reported that inherent axial muscular hypertonicity contributes to AS, based upon its characteristic and differentiating features from other rheumatic disorders (see Table I in ref. 7). Such a primary diathesis was proposed to exert increased tensional strains on the attachments of ligaments, tendons, or joint capsules on bone within the axial skeleton and at peripheral joints (i.e., enthesopathy). Adolescent idiopathic scoliosis, was then perceived to be a possible example of an acquired spinal condition caused by inherent relative insufficiency of the muscular support of the spine, by virtue of its clinical and epidemiologic features (Table I). The contrasting features of AIS versus AS are summarized in Table II. If the concept is true, then AIS and AS would be expected to rarely occur conjointly.

Literature search to test a counter-opposing occurrence of AIS and AS

A search of the English and non-English literature was conducted in the MEDLINE database (1966-February 2002). Search parameters were set to find the co-existence of scoliosis, hypermobility conditions, and Marfan syndrome (MFS) reported with either AS or other HLA-B27-related spondyloarthropathy (SPA). The two non-scoliosis disorders that were searched for in coexistence with AS or SPA, i.e., hypermobility conditions and MFS, were selected for reasons analogous to that of scoliosis. They too were suspected to have negative associations with the HLA-B27-related disorders. Rheumatoid arthritis (RA) was included in the search as a control inflammatory rheumatic disease for AS and SPA, in order to compare its frequency of coexistence with scoliosis and the other specified hypermobility disorders.

The medical subject headings searched were: Scoliosis (including the idiopathic and secondary forms); Marfan Syndrome; HLA-B27 Antigen; Reiter's Disease; Spondylitis, Ankylosing; and Arthritis, Rheumatoid (including juvenile). Hypermobility and hyperlaxity, buckling, instability, tonicity, sacroiliitis, and spondyloarthropathy were searched as keywords.

Only five reports were found of the coexistence of either scoliosis (85, 86) or hypermobility conditions (56, 87, 88) with AS or SPA disorders as opposed to a total of 17 articles retrieved of these conditions having coexisted with juvenile rheumatoid arthritis (JRA) or RA (Table III). A total of 20 additional reports were found of AS coexisting with RA, i.e., a concurrence of the two inflammatory rheumatic disorders, although of different genetic predispositions (references not cited). The relatively high frequency of the latter citations suggest that AS and RA are not counter-occurring disorders. Neither was a formal attempt made to enumerate the frequent reports of scoliosis co-existing with various hypermobility syndromes or MFS (1, 19,54,55). Such frequent reports endorse the concept that insufficient spinal support (i.e., either ligamentous or muscular) predisposes to scoliosis (1, 19).

Two case reports were retrieved of AS patients with the mention of coexistent scoliosis (85,86). In contrast,three survey publications (89-91) and two case reports (92, 93) were found on coexist-

Table II. Contrasting features of adolescent idiopathic scoliosis and ankylosing spondylitis.

- About 90% of all idiopathic scoliosis has an adolescent onset versus 10% of AS.
- In more advanced cases of AIS, the female to male (F:M) sex ratio is 4 to 1 or greater, which is the direct opposite of AS.
- In milder or moderate cases of AIS, the deformity does not typically advance radiographically during adulthood, compared with continued radiographic progression in juvenile and adult AS.
- Back pain is not a typical feature of uncomplicated AIS, unlike AS.
- Tarsal or hip joint arthropathy is not increased in AIS, in contrast to juvenile AS.
- Spinal involvement typically begins in the more mobile and unstable thoracolumbar spine in AIS versus the more stable and compression-bearing lumbosacral spine in AS.
- Strengthening exercises improve symptoms and maintain or reduce curve deformity in AIS, whereas stretching exercises are symptomatically beneficial in AS.
- Rotational hypermobility is characteristic of AIS, but decreased rotational ability significantly correlates with the duration of AS.
- Increased muscular tension is noted on the convex versus concave side of AIS curvatures, but neo-osteogenesis does not occur, unlike the syndesmophyte formation typical in AS.
- Unlike AIS, tissue injury is typical in AS, presumably contributed to by compressional forces or tensional stresses (or other biomechanical variables) which exceed tissue tolerance.
- Handedness is significantly correlated with the thoracic convexity in AIS, suggesting spinal instability, but is independent of patterns of pathology in juvenile AS.

Table III. Numbers of reports of specified coexisting disorders found in a literature search, from 1966 to 2002.*

	HLA-B27-related disorders*		
Axial hypotonicity or hypermobility syndromes*	Ankylosing spondylitis†	Other spondylo-arthropathy††	JRA/RA†
Scoliosis	2	0	5
Hypermobility syndrome	1	1	8 #
Marfan syndrome§	1	0	4 #
Total	4	1	17

* The search focused primarily on the coexistence of scoliosis, hypermobility syndrome, or Marfan syndrome with ankylosing spondylitis (AS) or other spondyloarthropathy (SPA). Juvenile or adult RA were included as control conditions to the HLA-B27-related disorders.
†The coexistence of AS with JRA or RA was reported in 20 articles (references not cited).
††Includes Reiter's syndrome, sacroiliitis, and other HLA-B27-related SPA.
§The suggestion of Marfan syndrome was provided by Dr. Dennis McGonagle.
#References not cited (the search was focused primarily upon scoliosis).

ing JRA or RA with scoliosis. The first article on AS was a case report of a 27-year-old male patient with a clinical diagnosis of HLA-B27-negative ankylosing spondylitis who had radiographic documentation of syndesmophytes and variegated sacroiliitis (85). He was mentioned to have right-sided leg length shortening of about 1 cm, a slanted pelvis, and a gentle left convex T12, L1, and L2 scoliosis (85). It is likely that the lumbar scoliosis was a secondary compensation mechanism (1,9,17,60) and not due to AIS. The second article on AS was the case report of an 18-year-old woman who had destructive, multilevel diskovertebral lesions mimicking infectious disease, bilateral sacroiliitis on computed tomography, and the presence of HLA-B27 antigen (86). During followup, she developed a slight thoracic scoliosis (86), probably secondary to the preceding extensive diskovertebral lesions and not attributable to AIS (1,9,17,60).

A single case report was retrieved on AS and coexistent Marfan syndrome (88). The 46-year-old male patient had a late onset of spinal symptoms at age 42 years and radiographic findings consistent with his diagnosis of HLA-B27-postive AS, in addition to typical physical traits of MFS (88). The authors noted the co-existence of peripheral joint hypermobility due to the ligamentous hyperlaxity of MFS, but the reduction of both axial skeletal mobility and chest expansion related to AS (88). They commented upon the interesting association of these two pathologies. In a report of two cases of seronegative SPA and coexisting hypermobility syndrome, this author also raised the issue: "Do pathological opposites cancel each other out ?" (56).

Scoliosis was reported to coexist with either JRA or juvenile chronic arthritis (JCA) more commonly than with adult RA (89-93). Scoliosis was believed to occur more commonly in JCA or JRA than in the population at large (89-93). In a study of 124 JRA patients, 11 (8.9%) were found to have scoliosis with a curve of over 20 degrees (89). In another study of 320 JRA patients (90), scoliosis of the thoracic or lumbar spine was detected in 17 (5.3%) patients, a frequency which was believed to be considerably higher than in the normal population. AIS was not studied specifically, and the reports on JCA or JRA (89-93) suggested that the scoliosis was mainly secondary, rather than a primary idiopathic diathesis.

The search turned up two reports of histocompatibility determinants in idiopathic scoliosis that were compared to normal control populations (94, 95). Both indicated relative frequencies of HLA-B27 in scoliosis patients similar to the respective control populations.

Biomechanical concepts in support of the hypothesis (Table IV)

As early as 1907, Feiss (96) considered mechanical factors as the cause of scoliosis. He used a physical model of the thoracolumbar spine and leather straps to simulate muscle forces. Later in 1932,Carey (97) developed a more elaborate physical model to demonstrate that all combinations of lateral curvature and axial rotation deformities can result from imbalances of both the superficial and the deep intrinsic muscles of the spine. He concluded that scoliosis is not a specific disease entity, but rather a “spinal sign of imbalance of muscle, bone growth, and the motor system of the spine”. Both models represent progressive thought and recognized the totality of bone-muscle interactions. Interestingly, much later when analytical methods became more sophisticated, many investigators abandoned the muscles in their biomechanical studies of scoliosis.

Many modeling studies that investigated spine buckling configurations in

Table IV. A summary of the relevance of muscles to spinal mechanics.

- Buckling is the loss of structural stability causing a momentary deformation, which may not necessarily cause an acute injury.
- Inherent muscle “stiffness” or “tone” is the main stabilizer of balanced spinal postures and is the most metabolically efficient.
- Trunk muscles, under the control of the central nervous system,are the most important structures providing stability to the spine.
- Appropriate muscle recruitment patterns are necessary to stabilize the spine and prevent injury in response to sudden loadings. Such responses are reflexive in nature and under the control of the central nervous system.
- Impaired motor control permits spine instability.
- All truncal muscles contribute to spinal stability and their relative importance is highly context-dependent, i.e., postures, loads, and load directions.
- Buckled configurations of the spine are complex three-dimensional shapes, which are also highly context-dependent, and may not be predicted by the primary deficiency.
- A great degree of spinal muscular insufficiency exists in primary and secondary neuromuscular disorders.

comparison to the deformities of scoliosis had not considered muscles that stabilize the spine (98-100). Biomechanical modeling of the three-dimensional geometric deformation of the spine in AIS without muscles is incorrect with respect to the complex theories of structural buckling (70, 101). The relatively simple Euler theory for beams and columns was often inappropriately applied to the structurally more complex spine (102-104). Therefore, the buckled shapes derived in these studies are irrelevant, whether or not they corresponded with the clinically observed curves.

The entire spinal system (bones, ligaments, and muscles) would have a very complex first mode of buckling (i.e., the shape of buckling which occurs at the lowest critical load). Such buckling (23) is largely determined by all of the trunk muscle forces, external loading schemes, and spine postures. In addition, the spine has normal kyphotic and lordotic curvatures (and their supporting ligaments-muscles), articulations within facet joints, and disk mechanics. That complex structure contributes to three-dimensional coupled rotations in the spine, which can be further modified by muscle forces (105, 106). The contribution of each muscle to spine stability is highly context dependent (107). Accordingly, buckled shapes can differ under varied circumstances and will depend upon the directions of external loads, their magnitude, and the initial spine posture. These complexities do not include the effects of the thoracic cage, which provide an additional stabilization to the thoracic spine (20, 108).

Deficiency in motor control of the spine may permit frequently repetitive un-stabilizing events, leading to various buckling configurations and the initiation of deformities, as seen in IS. The scoliotic deformity of AIS could be considered as the expression of passive accommodation to many repeated instability events with secondary tissue responses. However, any specific buckling configuration (i.e., the level or sidedness of curves) may not be determined fully by any underlying spinal instability predisposing to AIS. Rather, deformities are likely influenced by the complex structure and loading schemes. Nevertheless, axial muscles and their motor control should again become the most relevant focal points of future investigations into the initiating risk factors for acquiring AIS.

Future research challenges

The proposed hypothesis may not prove to be valid. Nor may the concept of axial muscular tonicity meaningfully clarify complex inter-relationships between the multiple factors that play a role in AIS and AS. Nevertheless, the biomechanics concepts involved are novel and directly relate to current research on spinal stability and its clinical ramifications. Some examples may be offered of controlled clinical, epidemiological, genetic, radiographic, and biomechanical studies of AIS and AS to explore the hypothesis.

One radiographic survey of the lumbar spine in Greenlanders versus Danes found sacroiliitis in 18 versus 3 percent, respectively, whereas scoliosis was less frequent in Greenlanders than in Danes (109). Controlled age-and sex-adjusted clinical, geoepidemiological, and radiological studies are obviously needed to determine if AIS and AS are counter-opposing disorders. Hereditary factors are widely accepted to play a role in both conditions (1,3,5, 6, 84). Critical collaborative comparisons of genomes from AIS versus AS patients (and families) may reveal diversities which could clarify the basis of the proposed polymorphism or more specific mechanisms in each condition. In one study (110), no consistent difference was found in the maximum voluntary trunk strength in attempted flexion, extension, and bilateral bending in 93 girls with mild AIS compared to 109 girls with structurally normal spines. A proper test of the current hypothesis would require direct measurements of axial stiffness versus mobility, strength, and other muscular components vital to the postural and dynamic support of the spine (44,83,111) in AIS versus AS. Longitudinal measurements during the early stages of AIS and prospective studies of genetically susceptible children could help to discriminate cause from effect relations.

The stiffness of axial rotation of the lower human spine was measured using a “Balans” chair, and by rhythmic torques generated automatically at the resonant frequency (59). Parameters relevant to back function (i.e. stiffness, inertia, and damping factors) were evaluated non-invasively. Such measurements are relevant to objective and symptomatic low back tightness in AS. Analogous techniques directed toward the thoracolumbar spine would be relevant in AIS. Other non-invasive methods of estimating trunk stiffness involve quick force release perturbations to the trunk (47) and direct measurements on a low friction platform (37). The former method includes the muscle reflex response, while the latter focuses on passive trunk stiffness.

Profoundly complex mechanisms control biomechanical vis-à-vis inflammatory processes in AS and SPA disorders (112). Critical studies need to determine which processes may predominate in the pre-clinical, early, and later clinical phases of these diseases (45). Unlike AS or SPA, however, AIS is not characterized by inflammatory activation. Whether or not HLA-B27 (113) predisposes AS patients to the biomechanical activation of inflammation (112) will also require critical investigations.

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