

Review Article

Scoliosis in Adolescence - A Revision

Minghelli B*

School of Health Jean Piaget Algarve, Research in Education and Community Intervention (RECI) - Piaget Institute, Portugal

*Corresponding author: Minghelli B, School of Health Jean Piaget Algarve, Research in Education and Community Intervention (RECI) - Piaget Institute, Silves, Portugal

Received: January 17, 2017; Accepted: February 13, 2017; Published: February 21, 2017

Abstract

Scoliosis is defined as a lateral deviation in the frontal plane of the body at an angle greater than 10° obtained on radiography by the Cobb method and may be accompanied by vertebral rotation, deformity in the sagittal plane and changes in the ribs. Several studies have shown a low prevalence of scoliosis in adolescents, with values between 2% to 4%. The ideal age for scoliosis screening is still under debate. The classification of scoliosis considers the following indicators: magnitude, location and direction of curvature, age group and etiology. Scoliosis isn't a single disease, but a common final pathway of a set of disorders, and there isn't still accepted scientific theory for the cause of adolescent scoliosis. This review will discuss the possible etiological origins of scoliosis and aspects related to its screening, and presents results on the prevalence of scoliosis in several studies that used different methods of detecting this deformity.

Keywords: Scoliosis; Adolescent; Etiology; Prevalence

Scoliosis Definition

The scoliosis term has Greek origin and means "curvature" [1,2]. Scoliosis is simply a descriptive term and not the consequence of an accurate diagnosis and is defined as a lateral deviation in the frontal plane of the body at an angle greater than 10° obtained on radiography by the Cobb method [3-5]. This deformity includes a vertebral body rotation, sagittal plane deformity and alterations in the ribs [5-7].

The classification of scoliosis considers the following indicators: magnitude, location and direction of curvature, age group and etiology [1].

Regarding the magnitude, scoliosis is classified as mild, moderate or severe, depending on the degree of involvement, and is also classified as primary or secondary. The primary curvatures correspond to those of larger magnitudes and involve a greater component of vertebral rotation and present a greater rigidity to the attempt of rectification. The secondary curvatures don't present important structuring components and are smaller in magnitude [1].

Scoliosis is described by the convexity location of the curve in the frontal plane and by the position of the apex of this curve. The four most common curvature patterns are remarkably consistent: more than 90% of the simple curvatures in the thoracic region of the spine are located to the right, 80% of the thoraco-lumbar curves are to the right, more than 70% of the simple curves in the lumbar regions are to the left and 90% of the "S" curves (double curves) are located to the right in the dorsal region and to the left in the lumbar region [7].

According to the onset period, scoliosis can be subdivided into three groups: infantile, with onset before age 3; juvenile, between 3 and 9 years of age; and in the adolescent, in individuals with ages equal or superior to 10 years until the end of the growth period, 18 years [3,4,7,8].

Regarding the classification of scoliosis according to etiology, it can be divided into structural and non-structural. There are several classifications of these types of scoliosis. The classification of scoliosis

according to Robert Winter (1987) [1] will be described below.

Structural scoliosis includes idiopathic, neuromuscular, congenital, neurofibromatosis, mesenchymal diseases, rheumatic diseases, trauma, extra-spinal contractures, osteochondrodystrofas, bone infections, metabolic diseases related to the lumbosacral joint and tumors [1].

Non-structural or functional scoliosis includes postural, hysterical, nervous, inflammatory irritation, asymmetry of the length of the lower limbs and contractures in the region of the hip joint [1].

The "non-structural" or "functional" scoliosis encompasses the temporary and reversible lateral curvatures that occur naturally in response to an asymmetric posture. In this type of scoliosis the spine return to a linear configuration when the patient adopts dorsal decubitus or tilts to the side, and can be completely corrected by, for example, a shoe insole to balance a length discrepancy of a lower limb. Structural scoliosis is associated with a loss of flexibility in one or more segments of the spine and doesn't yield with the adoption of other postures such as lateral flexion and dorsal decubitus [1,9,10]; Scoliosis is defined as structural when some part of the curvature is fixed, and is always accompanied by rotation of the vertebral body [7].

Functional scoliosis may be a consequence of poor posture, and structural scoliosis is seen as a genetically based disorder, the result of which is largely impervious to environmental influences [9,10].

Functional scoliosis may result from postural imbalance due to pain, muscle spasms, or other factors that may evolve over time. Functional scoliosis can progress to structural scoliosis, where it is believed that postural imbalance, alone, can cause severe structural scoliosis with vertebral rotation, as well as coinage of the vertebrae and intervertebral discs [8].

Etiology of Scoliosis

Scoliosis isn't a single disease, but a common final pathway of a

set of disorders [7,11-13]. The etiology of most scoliosis is unknown and since 1922 these patients have been diagnosed with idiopathic scoliosis [3]. Idiopathic scoliosis is a structural curvature of unknown cause [7,14,15].

Throughout the 18th and 19th centuries, it was believed that scoliosis was caused by the body's postural positioning. The etiology of idiopathic scoliosis is attributed to a wide variety of conditions ranging from inadequate posture to malnutrition [6].

There is still no single accepted scientific theory for the cause of adolescent idiopathic scoliosis. Several authors argue that adolescent idiopathic scoliosis can be caused by genetic factors [3,6-8,11,12,16-18], asymmetries of bone growth, bone mass, abnormalities of the neuromuscular system [3,7,8,16-20], the content of collagen in the intervertebral disc, proteoglycans and metabolism of calcium and mechanical factors [3,6,7,13,16,17]. Additionally, the possibility of environmental factors being involved in its pathogenesis isn't excluded [3,7,9,11,16,17].

Almost all individuals presenting with adolescent idiopathic scoliosis have a positive familial history [18]. Data of studies had suggest possible chromosomal regions related to the etiology of adolescent idiopathic scoliosis, with a genetic link in families with multiple affected members. There is a theory of genes responsible for the synthesis of proteins for the composition of human tissues, which act in supporting the spine, and probably cause adolescent idiopathic scoliosis [8].

Vertical human posture requires continuous, precise, and complex coordination between the central nervous system and several of bones, muscles, cartilage, and other soft tissues. Therefore, any disease, injury or mutation that results in failure to mount or deterioration of any component can promote the development of scoliosis. In this way, scoliosis can develop in association with postural imbalance due to genetic defects, as well as the presence of pain resulting from trauma or surgery. Prolonged postural imbalance may result, over time, in the establishment of an asymmetric continuous state of load relative to the axis of the spine, resulting in the development of a deformed spinal curvature [9].

Grivas study [21] found a significant correlation between trunk asymmetry in the thoracic region and the brain lateralization function expressed through the manual domain ($p < 0.038$). 8,245 students, aged 6 to 18 years, were evaluated through the use of the scoliometer, of which 91% were right-handed students. The results obtained in this study point to the possible role of the cerebral cortex, the dominant hemisphere of the brain, in determining the surface morphology of the thoracic region, which may be involved in the etiology of scoliosis.

The asymmetric load applied to the vertebral axis is the main driving force for the development and progression of a spinal deformity: once a curvature develops, an uneven compression in the vertebrae results in uneven growth, which in turn contributes to the progression of deformity [9]. In this way, biomechanical factors can affect the alignment of the spine so that they are often involved in the pathogenesis of idiopathic scoliosis. The mechanical properties of the spinal tissues, alignment of the spine, unequally distributed loads (either through forces or displacement), and the way the spine is supported can lead to the development of scoliosis [13].

Scoliosis can also develop due to weakness of the abdominal muscles, which isn't able to properly support the spine. Increased extensibility of soft tissues and joint laxity suggestive of reduced muscle or ligament rigidity may be important risk factors for the progression of scoliosis, but there is little evidence that it's a significant etiological factor [13].

Scoliosis can occur as a result of posture or other developmental disorders [10]. However, according to the Scoliosis Research Society [14] and Spine Society of Australia [11] scoliosis doesn't occur by carrying excessive weights in school backpacks, by assuming wrong postures when sleeping and standing, by spending many hours watching television or by difference in limb length lower. However, the Zheng et al. study identified that the use of single-shoulder bags, and more time using a computer were associated with presence of scoliosis [22].

A possible reduction of spinal cord - to - vertebral column length ratios can be involved with pathogenesis of adolescent idiopathic scoliosis, suggesting a disproportional growth between the skeletal and the neural systems which may promote stretching -tethering forces between the cranial and caudal extremities of the spine, leading to the initiation and progression of thoracic scoliosis [18].

The pathogenesis of idiopathic scoliosis has also been recently related to Body Mass Index (BMI) [10,18,23], although this isn't well established, such as the relation between BMI and trunk asymmetry [17,23,24].

Grivas et al. [25] study showed that there wasn't evidence that obesity is a factor leading to postural changes in the trunk, and a low BMI was associated with the presence of severe asymmetries of the trunk in healthy adolescents, suggesting that the consequence of the asymmetry of the trunk would be related to the low levels of leptin, where the hypothalamus could influence the asymmetric growth of the trunk.

In agreement with these results (and other similar studies), a new pathogenic interpretation called the osteo-neural theory that relates the Autonomic Nervous System (Sympathetic division) to the formation/resorption and bone growth arises. This theory involves the imbalance between the Somatic Nervous System and the Autonomic Nervous System, with implications in the spine, and it's suggested that severe asymmetries of the trunk are caused by a specific genetic factor that increases the sensitivity of the hypothalamus to leptin. This increase leads to a low concentration of circulating leptin levels, which are associated with a low BMI [25].

This functional imbalance of the hypothalamus is expressed through the increase of the activity of the Sympathetic Nervous System which, through neuroendocrine mechanisms, leads to an early increase in the peak of growth velocity, to an excessive growth of the skeleton, to an asymmetry in the length of the skeleton, especially in the regions of the ribs, vertebrae and hip bones, leading to severe asymmetries, besides promoting the development of a low BMI; that is, the effects of the Sympathetic Nervous System can cause changes in the spine, including asymmetries, which affects the neuroendocrine effects on the growth of the adolescent's spine. This action of the Sympathetic Nervous System overlaps the action of the Somatic System that cannot activate the postural mechanisms

to control these asymmetric forces that affect the trunk, thus leading to asymmetries [17,18,24-27]. However, there is still no evidence that BMI can be related to the pathogenesis of asymmetry of the trunk, such as scoliosis; therefore, further studies are needed to prove this fact [17,25-27].

Yong et al. [28] found that girls with low body weight (BMI <18.5 kg/m²) had a higher risk of developing adolescent idiopathic scoliosis (OR = 1.5; 95% CI: 1.2 -1.8, p = 0.001) for girls whose weight was in the healthy range and range of overweight.

There is also a tendency to increase the number of adolescents with idiopathic scoliosis in the overweight category [29,30].

Minghelli et al. [31] study evaluated 966 students, and these dates showed that regarding the nutritional status, the majority of students classified with scoliosis showed normal weight, however it was verified a high prevalence of students with thinness (7.1%) compared to those classified as overweight (1.7%) and obesity (1.9%), but not achieving statistical significance.

Bruce et al. [30] study evaluated 427 adolescents with idiopathic scoliosis and found that female patients who presented a Cobb angle greater than 50° were older and had a significantly higher BMI than those with curves smaller than 50° (p= 0.0557). The reasons for these results can be attributed to the difficulty of detection of the curve by the presence of subcutaneous fat, influence of endocrine factors, as well as premature puberty caused by an increase in fat mass [30].

Another factor thought to be related to the development of idiopathic scoliosis is the age of menarche; this factor varies in different geographic latitudes [32] and it's thought that the late age of menarche is associated with a higher prevalence of adolescent idiopathic scoliosis [32,33].

The relation of sex and age to height gain and the speed of vertebral growth is a relationship of extreme importance, presenting two periods of rapid growth, being a first period from birth to three years of age and the second period of growth that occurs during adolescence. The velocity of vertebral growth is directly related to the development of sexual characters, where the highest peak occurs approximately one year after the development of the breasts and the pubic hair in the female sex (between 11 and 12 years) and the pubic, axillary and facial hair in the male sex (between 14 and 15 years old). After one year of this period, menarche will occur in girls, and the growth in length of the spinal column will be completed after another 2 years [1].

Idiopathic scoliosis is associated with the pubertal growth spurt and its progression decreases upon completion of skeletal maturity. The age at onset of menarche is indicative of the girls' remaining peak growth. The late onset of menstruation is correlated with delayed skeletal maturity and implies that there is a potential for the progression of a scoliotic curve [32-34].

High levels of melatonin before menarche can be considered as a possible factor for the onset of idiopathic scoliosis, since it changes the length growth in a period of vulnerability of the spine [6,12,13,17,32,35].

The prevalence of adolescent idiopathic scoliosis varies according

to the different latitudes and has higher values in the Northern countries and lower values in countries near the Equator. One possible explanation for the geographic influence on the pathogenesis of idiopathic scoliosis is in the latitude that differentiates sunlight, influencing the secretion of melatonin that modifies the age of menarche, the latter associated with the prevalence of idiopathic scoliosis [32].

Sexual development occurs earlier in the tropics than in temperate zones. It has been reported that climate has little or no individual effect on menarche [32,36]. The retinal response to ambient light mediates a wider list of neuroendocrine effects, including control of puberty, ovulation, and a large number of daily rhythms [32].

The production of melatonin is stimulated by darkness. The lack of light in the retina through the optic nerve and the preganglionic sympathetic fibers of the superior cervical ganglion reach the center of vision and then through the postganglionic fibers stimulate the pineal gland and causes the release of noradrenaline through of the sympathetic system. Norepinephrine mediates the entry of tryptophan into the pineal gland and controls the activity of many enzymes, especially Hydroxyindole-O-Methyltransferase (HIOMT) which is important for the synthesis of melatonin. The concentration of the pineal gland of HIOMT reduces during the day and increases during the night, as a result of ambient light conditions. Melatonin acts indirectly on the gonads, reducing the secretion of gonadotrophins and, especially, the luteinizing hormone that stimulates ovulation. In this way darkness leads to an increase in the production of melatonin and the clarity of light causes a reduction in melatonin production [32,36].

The role of melatonin deficiency in the pathogenesis of adolescent idiopathic scoliosis was proposed by Machida et al. [6] who in their study administered melatonin in chickens and verified that in chickens submitted to melatonin infusion there was prevention of scoliosis, concluding that a defect in melatonin production could be related to the etiology of idiopathic scoliosis in humans. However, the results observed in animals cannot be associated with humans, since the action of melatonin in humans seems to differ from that of animals. Results of a prospective clinical Machida et al. [37] study suggested that melatonin deficiency could play a role in the prognosis of adolescent idiopathic scoliosis, with a possible preventive effect of melatonin supplements on curve progression. The study by Sadat-Ali et al. [38] evaluated melatonin levels in 20 adolescents with idiopathic scoliosis and found that these levels were significantly lower in these individuals than in the control group, suggesting that serum melatonin levels may contribute to pathogenesis of idiopathic scoliosis.

Calmodulin can also be a molecule involved in an etiology of scoliosis, as a second messenger of melatonin and because of its effects on muscle contractility; the increased calmodulin levels in platelets have been shown to be associated with the progression of idiopathic scoliosis [18].

Mao et al. [33] study found that 10% of the 6,376 healthy girls had menarche before 11.38 years and approximately 90% had menstruated before 13.88 years. Of the 2,196 girls with scoliosis, less than 10% started menstruation before 11.27 years and about 90% menstruated

with 14.38 years of age. The mean age of menarche in girls with adolescent idiopathic scoliosis was significantly later (12.83 ± 1.22 years) compared to healthy girls (12.63 ± 0.98 years) ($p < 0.001$). The proportion of girls who started menstruation after 14 years of age was significantly higher in girls with idiopathic scoliosis in the adolescent than in the healthy group (16.3 versus 8.1%, $p < 0.001$). In addition, girls with idiopathic adolescent scoliosis with a Cobb angle greater than 60° started menstruation with a mean of 13.25 years which was significantly later compared to girls with Cobb angle less than 40° (12.81 years, $p < 0.05$).

The study by Grivas et al. [39] didn't find a statistically significant difference between the age of menarche in girls with and without scoliosis. The mean age of menarche of girls with scoliosis was 11.98 ± 1.49 years, ranging from 7.7 to 16.72 years. 28 girls with scoliosis hadn't yet had menarche. The late onset of menarche or non menarche was observed in 4 girls with scoliosis.

Yong et al. [28] study, held in Singapore, found that the mean age of menarche was 11.5 ± 0.96 years, with 55.9% (1,806) of girls reporting menarche up to 12 years of age. Of the female students who reached menarche, 762 (42.2%) had scoliosis. The risk of developing scoliosis for girls who had menarche before 13 years of age was 1.5 (95% CI: 1.1-1.9, $p = 0.003$). In this study, and considering Malaysian and Chinese ethnicities, ethnicity also showed to be a weak factor in the univariate model ($p = 0.049$), showing that the Malays had a lower risk of adolescent idiopathic scoliosis compared to the Chinese.

Scoliosis Screening

The Scoliosis Research Society, American Academy of Orthopedic Surgeons, The Pediatric Orthopedic Society of North America, and the American Academy of Paediatrics have the favorable opinion about scoliosis screening in schools, while the Canadian Task Force on the Periodic Health Examination, the British Orthopedic Association and British Scoliosis Society don't recommend these screenings. Objections to school scoliosis screening are largely based on the low rate of scoliosis prevalence, the inverse relationship of sensitivity and specificity in the screening process, high false positive rates, high inter-observer variances, and the costs involved with excesses of examinations. The challenge, therefore, for scoliosis screening programs is to decrease sensitivity to an acceptable rate of false-positive results and increase specificity in order to reduce over examination, thus minimizing costs to patients and society [40].

The ideal age for scoliosis screening is still under debate. Screening has been generally performed between the ages of 10 and 14 years. Ideally, the screening should be performed on girls before the onset of menstruation and 1-2 years later for boys [40].

The American Academy of Orthopedic Surgeons recommends screening for girls ages 11 to 13 and boys 13 to 14 years old. The American Academy of Paediatrics recommends performing the Adams test as a screening test at the ages of 10, 12, 14, and 16 years [40,41].

According to Grivas et al. [41] the screening is justified, since it allows detecting mild and reversible spinal curvatures and treating them conservatively, before progressing to spinal deformities, with a potential risk to causing symptoms throughout life.

A systematic review aimed to assess the efficacy and cost-effectiveness of school screening programs for scoliosis. A total of 248 studies were analyzed, 117 abstracts were selected and 28 articles were included in the results of this review. A reasonable level of evidence has been found suggesting that the school screening program for scoliosis is safe to contribute to the early detection of scoliosis and reduction of surgical acts. In addition, there was also evidence to suggest that these screenings are effective in relation to cost-effective. School screening was recommended only for a high-risk group, including 12-year-old girls. However, according to the study by Labelle et al. [42], there is scientific evidence to support the importance of scoliosis screening in relation to the efficacy, clinical, program and efficacy of treatment; however, according to the latter study there is insufficient evidence in relation to cost-effective.

There is moderate evidence that scoliosis screening allows early detection and referral of patients, and scant scientific evidence suggests that patients with scoliosis who were detected early are less likely to undergo surgery when compared to not yet screened, however these results haven't yet been fully clarified [42].

Prevalence of Scoliosis

Adolescent scoliosis corresponds to 80% of idiopathic scoliosis [7,14,15]. According to Asher and Burton [3], adolescent idiopathic scoliosis is a likely systemic condition of unknown cause, affecting about 2.5% of the population. For Reamy and Slakey [4] this type of scoliosis is present in 2% to 4% of individuals aged 10 to 16 years. For Weinstein et al. [12] the prevalence of this type of scoliosis is present in 1% to 3% of adolescents between the ages of 10 and 16 years.

The proportion of idiopathic scoliosis among boys and girls with 10° curves is similar, but this ratio increases with the severity of the curve ie, for curves between 10° and 20° the girl-boy ratio is 2:1, while for curves greater than 30° , this ratio is 10 girls for each boy (10:1). Scoliosis in girls tends to progress more commonly when compared to boys [4,5,7,14].

In the specific case of adolescent idiopathic scoliosis, the time of greatest risk for the progression of curvature occurs in the puberty period, where bone growth occurs very rapidly [13-14]. The curvature identified before the menarche period is more likely to progress (66%) than those detected after menarche (33%) [7].

The curves of adolescent idiopathic scoliosis can be located in either direction, but the right and left lumbar curves are the most common patterns. A pattern of double curvature is also common with the thoracic components on the right and lumbar on the left. The cervical region is not affected by adolescent idiopathic scoliosis [43].

There are several explanations related to the higher prevalence of thoracic curves of adolescent idiopathic scoliosis with convexity to the right, including the positioning of the thoracic and abdominal organs, such as the heart, the larger size of the right lung, the diaphragm and the aorta [44].

The study by Adobor et al. [40] evaluated 4,000 Norwegian adolescents aged 12 years and found that 60 (1.5%) students had values greater than 7° with the scoliometer, of which 39 (65%) girls and 21 (35%) boys; 22 (0.55%) students had confirmation of scoliosis with radiographs (Cobb angle greater than 10°), of which 16 (73%)

were female and 6 (27%) were male; 38 (0.95%) students had normal spinal curvatures on X-ray examination (false-positive). All girls with scoliosis were postmenarche.

The study by Grivas et al. [45] evaluated 3,301 children, aged between 3 and 9 years, and found that 25.8% and 28.9% of the boys and 23% and 27.2% of the girls presented mild asymmetry (values of the scoliometer between 1° to 6°) of the trunk in the thoracic and lumbar region, respectively. Severe asymmetry (values equal to or above 7°) was observed in 0.9%, 1.9% and 2.5% of boys and in 1%, 2% and 2.3% of girls in the thoracic, thoracolumbar and lumbar, respectively. The highest prevalence of asymmetry was identified in boys aged 8 to 9 years and in girls between 6 and 9 years.

Grivas et al. [46] evaluated 2,071 children and adolescents, aged 5 to 18 years, using the scoliometer and verified a prevalence of mild trunk asymmetry in 32.9% of the boys and in 34.9% of girls. The values greater than or equal to 7° were observed in 3.23% of the boys and in 3.92% of the girls. The asymmetries located on the right and in the thoracolumbar region were the most prevalent.

Minghelli et al. [31] evaluated 966 adolescents in southern Portugal, aged between 10 and 16 years, with the scoliometer and the results showed presence of scoliosis in 41 (4.2%) students (values equal to or greater than 7° with scoliometer).

Wong et al. [47] evaluated 72,699 students and verified scoliosis prevalence (by radiographs) of 0.02% of boys and 0.05% of girls between 6 and 7 years of age, 0.15% of boys and 0.24% of girls from 9 to 10 years of age, 0.21% of boys and 1.37% of girls between 11 and 12 years of age, and 0.66% and 2.22% for boys and girls between 13 and 14 years of age, respectively. The thoracolumbar curves were the most common curve (40.1%), followed by thoracic curves (33.3%), double/triple curves (18.7%) and lumbar curves (7.9%).

Yong et al. [28] study revealed prevalence of idiopathic scoliosis of 0.27%, 0.64%, 1.58%, 2.22% and 2.49% by radiographs in 93,626 girls with 9, 10, 11, 12 and 13 years of age, respectively, showing a trend of increasing prevalence rates with increasing age. There was a significant increase in adolescent idiopathic scoliosis prevalence rates for girls aged 10 to 11 years compared to 9 years of age (OR = 1.7; 95% CI: 1.1-2.4, p=0.01). There was also a significant increase in prevalence rates among girls 12 to 13 years of age (OR = 2.2; 95% CI: 1.4-3.3, p=0.001). Of the 1,118 girls with scoliosis, 21.4% had a thoracolumbar curve on the left, 14.9% thoracolumbar on the right, 13.9% thoracic on the right, 3.5% thoracic on the left, 2.1% left lumbar and 0.4% to the right lumbar.

Soucacos et al. [48] evaluated 82,901 adolescents, and 4,185 students presented a positive result in the Adams test; these adolescents were submitted to radiography, with the presence of scoliosis in only 1,436 (1.7%) students.

Data of Zheng et al. [22] study showed that 282 (2.6%) of 11,024 individuals, age between 6 and 13 years, were found to be positive as a result of angle of trunk inclination, but of the 128 individuals who were screened positive only 11 (0.22%) had a diagnoses of scoliosis confirmed by Cobbangles greater than 10° (8.6%). The highest prevalence was found in girls aged 12-13 years.

Detsch et al. [49] performed the postural analysis in individuals

aged 14 to 18 years, which the photographic record was used in a posturograph, and the results obtained revealed a prevalence of 66% of students with lateral alterations. The study by Minghelli et al. [50] also performed the postural analysis by photographs in 75 individuals aged between 10 and 18 years and the results revealed that 86.7% of individuals presented trunk rotation.

As previously mentioned, the existence of asymmetries and/or trunk rotation may not correspond to the actual presence of scoliosis [5,14,51,52]. This fact may explain the differences between the high prevalence of factors indicative of scoliosis referred to in the latter studies and data from other studies that revealed that adolescent idiopathic scoliosis is present in approximately 2% to 4% of most populations. This disparity of values can be explained by the nature of the measurement instruments used in the studies. However, the values observed in the previous studies may be indicative of the presence of some physiological or structural lateral deviation [3,4,7].

Conclusion

Scoliosis is a lateral deviation in the frontal plane of the body at an angle greater than 10° obtained on radiography by the Cobb method, that include a vertebral body rotation, sagittal plane deformity and alterations in the ribs. This deformity can be classified by magnitude, location and direction of curvature, age group and etiology. Despite the fact that several studies showed numerous potential etiologies for idiopathic scoliosis, the primary etiology of idiopathic scoliosis remains unknown.

Although presenting a low prevalence in adolescents, scoliosis should be the subject of more scientific research in order to better define the etiological and aggravating factors, to prevent the appearance of other conditions, such as possibility of pain, social function and self-image which can be reduced in this type of patients. Untreated adolescent idiopathic scoliosis, seen in the long term includes curve progression, back pain, cardiorespiratory problems, and psychosocial problems.

References

1. Hebert S, Xavier R, Gomes Pardini A, Tarcísio E P de Barros Filho. *Ortopedia e Traumatologia: Princípios e prática*. São Paulo. Artmed. 2003.
2. About Scoliosis: Symptoms, causes, treatment. *Scoliosis Australia*.
3. Asher M, Burton D. Adolescent idiopathic scoliosis: natural history and long term treatment effects. *Scoliosis*. 2006; 1: 2.
4. Reamy B, Slakey J. Adolescent idiopathic scoliosis: review and currents concepts. *Am Fam Physician*. 2001; 64: 111-116.
5. Bunnell W. Selective screening for scoliosis. *Clinical Orthopaedics and Related Research*. 2005; 434: 40-45.
6. Machida M. Cause of Idiopathic Scoliosis. *Spine*. 1999; 24: 2576-2583.
7. Rinsky L, Gamble J. Adolescent idiopathic scoliosis. *West J Med*. 1988; 148: 182-191.
8. Wajchenberg M, Astur N, Kanas M, Martins D. Adolescent idiopathic scoliosis: current concepts on neurological and muscular etiologies. *Scoliosis Spinal Disord*. 2016; 11: 4.
9. Hawes M, O'Brien J. The transformation of spinal curvature into spinal deformity: pathological processes and implications for treatment. *Scoliosis*. 2006; 1: 3.
10. Anderson S. Spinal curves and scoliosis. *Radiologic Technology*. 2007; 79: 44-65.

11. Burwell R, Freeman B, Dangerfield P, Aujla R, Cole A, Kirby A, et al. Etiologic Theories of Idiopathic Scoliosis: Enantiomorph Disorder Concept of Bilateral Symmetry, Physically-created Growth Conflicts and Possible Prevention. *Stud Health Technol Inform.* 2006; 123: 391-397.
12. Weinstein S, Dolan L, Cheng J, Danielsson A, Morcuende J. Adolescent idiopathic scoliosis. *Lancet.* 2008; 371: 1527-1537.
13. Lowe T, Edgar M, Margulies J, Miller N, Raso V, Reinker K, et al. Etiology of Idiopathic Scoliosis: Current Trends in Research. *J Bone Joint Surg Am.* 2000; 82: 1157-1168.
14. Scoliosis Research Society.
15. Weiss H, Weiss G, Petermann F. Incidence of curvature progression in idiopathic scoliosis patients treated with Scoliosis In-Patient Rehabilitation (SIR): an age and sex-matched controlled study. *Pediatr Rehab.* 2003; 6: 23-30.
16. Burwell R. Aetiology of idiopathic scoliosis: current concepts. *Pediatric Rehabilitation.* 2003; 6: 137-170.
17. Burwell R, Dangerfield P, Freeman B. Concepts on the pathogenesis of adolescent idiopathic scoliosis. Bone growth and mass, vertebral column, spinal cord, brain, skull, extra-spinal left-right skeletal length asymmetries, disproportions and molecular pathogenesis. *Stud Health Technol Inform.* 2008; 135: 3-52.
18. Dayer R, Haumont T, Belaieff W1, Lascombes P. Idiopathic scoliosis: etiological concepts and hypotheses. *J Child Orthop.* 2013; 7: 11-16.
19. Sèze M, Cugy E. Pathogenesis of idiopathic scoliosis: A review. *Annals of Physical and Rehabilitation Medicine.* 2012; 55: 128-138.
20. Day G, Frawley K, Phillips G, McPhee I, Labrom R, Askin G, et al. The vertebral body growth plate in scoliosis: a primary disturbance of growth? *Scoliosis.* 2008; 3: 3.
21. Grivas T, Vasiliadis E, Polyzois V, Mouzakis V. Trunk asymmetry and handedness in 8245 school children. *Pediatric Rehabilitation.* 2006; 9: 259-266.
22. Zheng Y, Wu X, Dang Y, Yang Y, Reinhardt JD, Dang Y. Prevalence and determinants of idiopathic scoliosis in primary school children in Beitang district, Wuxi, China. *J Rehabil Med.* 2016; 48: 547-553.
23. Vlaski E, Stavric K, Isjanovska R, Seckova L, Kimovska M. Overweight hypothesis in asthma and eczema in young adolescents. *Allergol Immunopathol.* 2006; 34: 199-205.
24. Burwell R, Aujla R, Kirby S, Dangerfield P, Moulton A, Cole A, et al. Body mass index of girls in health influences menarche and skeletal maturation: a leptin-sympathetic nervous system focus on the trunk with hypothalamic asymmetric dysfunction in the pathogenesis of adolescent idiopathic scoliosis? *Stud Health Technol Inform.* 2008; 140: 9-21.
25. Grivas T, Burwell R, Mihas C, Vasiliadis E, Triantafyllopoulos G, Kaspiris A. Relatively lower body mass index is associated with an excess of severe truncal asymmetry in healthy adolescents: Do white adipose tissue, leptin, hypothalamus and sympathetic nervous system influence truncal growth asymmetry? *Scoliosis.* 2009; 4: 13.
26. Burwell R, Dangerfield P, Freeman B. Etiologic theories of idiopathic scoliosis. Somatic nervous system and the NOTOM escalator concept as one component in the pathogenesis of adolescent idiopathic scoliosis. *Stud Health Technol Inform.* 2008; 140: 208-217.
27. Burwell R, Aujla R, Grevitt M, Dangerfield P, Moulton A, Randell T, et al. Pathogenesis of adolescent idiopathic scoliosis in girls - a double neuro-osteous theory involving disharmony between two nervous systems, somatic and autonomic expressed in the spine and trunk: possible dependency on sympathetic nervous system and hormones with implications for medical therapy. *Scoliosis.* 2009; 4: 24.
28. Yong F, Wong H, Chow K. Prevalence of adolescent idiopathic scoliosis among female school children in Singapore. *Ann Acad Med Singapore.* 2009; 38: 1056-1063.
29. Sucato D, Lubicky J, Sarwark J. BMI is changing in children and adolescents presenting for scoliosis surgery. The Prospective Pediatric Scoliosis Database. Scoliosis Research Society 43rd Annual Meeting and Course. USA. 2008.
30. Bruce B, Talwalkar V, Iwinski H, Walker J, Milbrandt T. Does obesity hide adolescent idiopathic scoliosis? Scoliosis Research Society 43rd Annual Meeting and Course. 2008.
31. Minghelli B, Nunes C, Oliveira R. Prevalence of scoliosis in southern Portugal Adolescents. *Ped Endocrinol Rev.* 2014; 11: 374-382.
32. Grivas T, Vasiliadis E, Mouzakis V, Mihas C, Koufopoulos G. Association between adolescent idiopathic scoliosis prevalence and age at menarche in different geographic latitudes. *Scoliosis.* 2006; 1: 9.
33. Mao S, Jiang J, Sun X, Zhao Q, Qian B, Liu Z, et al. Timing of menarche in Chinese girls with and without adolescent idiopathic scoliosis: current results and review of the literature. *Eur Spine J.* 2011; 20: 260-265.
34. Goldberg C, Dowling F, Fogarty E. Adolescent idiopathic scoliosis: is rising growth rate the triggering factor in progression? *Eur Spine J.* 1993; 2: 29-36.
35. Girardo M, Bettini N, Dema E, Cervellati S. The role of melatonin in the pathogenesis of adolescent idiopathic scoliosis (AIS). *Eur Spine J.* 2011; 20: 68-74.
36. Saar E, Shalev C, Dalai I, Sod-Moriah U. Age at menarche: the influence of environmental conditions. *Int J Biometeorol.* 1988; 32: 33-35.
37. Machida M, Dubouset J, et al. Serum melatonin levels in adolescent idiopathic scoliosis prediction and prevention for curve progression-a prospective study. *J Pineal Res.* 2009; 46: 344-348.
38. Sadat-Ali M, Al-Habdan I, Al-Othman A. Scoliose idiopathique de l'adolescence: la mélatonine est-elle une cause? *Revue du rhumatisme.* 2000; 67: 73-76.
39. Grivas T, Samelis P, Pappa AS, Stavlas P, Polyzois D. Menarche in scoliotic and nonscoliotic Mediterranean girls. Is there any relation between menarche and laterality of scoliotic curves? *Stud Health Technol Inform.* 2002; 88: 30-36.
40. Adobor R, Rimeslatten S, Steen H, Brox J. School screening and point prevalence of adolescent idiopathic scoliosis in 4000 Norwegian children aged 12 years. *Scoliosis.* 2011; 6: 23.
41. Grivas T, Wade M, Negrini S, O'Brien J, Maruyama T, Hawes M, et al. SOSORT consensus paper: school screening for scoliosis. Where are we today? *Scoliosis.* 2007; 2: 17.
42. Labelle H, Richards S, Kleuver M, Grivas T, Luk K, Wong H, et al. Screening for adolescent idiopathic scoliosis: an information statement by the scoliosis research society international task force. *Scoliosis.* 2013; 8: 17.
43. About Scoliosis: Symptoms, causes, treatment. *Scoliosis Australia.*
44. Burwell RG, Dangerfield PH, Freeman BJ, Aujla RK, Cole AA, Kirby AS, et al. Etiologic theories of idiopathic scoliosis: the breaking of bilateral symmetry in relation to left-right asymmetry of internal organs, right thoracic adolescent idiopathic scoliosis (AIS) and vertebrate evolution. *Stud Health Technol Inform.* 2006; 123: 385-390.
45. Grivas T, Vasiliadis E, Mihas C, Triantafyllopoulos G, Kaspiris A. Trunk asymmetry in juveniles. *Scoliosis* 2008; 3: 13.
46. Grivas T, Vasiliadis ES, Koufopoulos G, Segos D, Triantafyllopoulos G, Mouzakis V. Study of trunk asymmetry in normal children and adolescents. *Scoliosis.* 2006; 1: 1-8
47. Wong H, Hui J, Rajan U, Chia H. Idiopathic scoliosis in Singapore schoolchildren: a prevalence study 15 years into the screening program. *Spine.* 2005; 30: 1188-1196.
48. Soucacos P, Soucacos P, Zacharis K, et al. School-screening for scoliosis: a prospective epidemiological study in Northwestern and Central Greece. *J Bone Joint Surg.* 1997; 79: 1498-1503.
49. Detsch C, Luz A, Candotti C, Oliveira D, Lazon F, Guimarães L, et al. Prevalência de alterações posturais em escolares do ensino médio em uma cidade no Sul do Brasil. *Rev Panam Salud Publica.* 2007; 21: 231-238.

50. Minghelli B, Costa J, Faria H, Serro F. Avaliação postural: método de detecção precoce de alterações posturais em alunos da Escola Básica de Silves, região do Algarve, Portugal. *Revista Fisio Brasil*. 2012; 108: 16-25.
51. Morrissy R. School screening for scoliosis. *Spine*. 1999; 24: 2584-2591.
52. Bassewitz H, Herkowitz H. The spine. Functional anatomy of the spine. Editors. Placzek J, Boyce D. *Orthopaedic Physical Therapy Secrets*. Philadelphia: Hanley & Belfus. 2001.