# Study of the Link between Ligament Laxity and the Occurrence of Idiopathic Scoliosis in Adolescents

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## Abstract

**Background:** The objective of our study is to investigate the relationship between ligament hyperlaxity and adolescent idiopathic scoliosis (AIS) within other risk factors. **Materials and Methods:** We conducted a matched case–control study in adolescents aged 9–15 years. We matched the age and sex of adolescents with AIS to their healthy controls. The AIS is defined by a Cobb angle of 10° and the ligament hyperlaxity is defined by a Beighton score ≥4. The two parameters were identified blindly. Other risk factors for AIS were analyzed. The multivariate analysis by simple logistic regression was carried out. **Results:** We analyzed 430 adolescents, among them 215 subjects with AIS were paired with 215 healthy controls. The sex ratio (female/male) is 1.46. Scoliotics showed a significantly higher rate of joint hyperlaxity than controls, $P < 0.003$ (61.4% vs. 46.5%) with an odds ratio (Orb) of 1.83 confidence interval (CI) (1.25–2.68). The multivariate analysis by logistic regression revealed three predictors of AIS which are ligament hyperlaxity ORa = 1.82 CI (1.23–2.69) $P < 0.003$, the existence of similar cases in the ORa family = 1.94 (1.24–3.03) $P < 0.002$ and a trunk growth spurt ≥4 cm/6 months ORa = 1.62 (1.09–2.4) $P < 0.02$. **Conclusion:** There is a potential relationship between ligament hyperlaxity and the occurrence of idiopathic scoliosis in adolescents. Two other predictors were detected, similar cases of scoliosis in the family and the trunk growth spurt.

**Keywords:** Adolescent idiopathic scoliosis, ligament hyperlaxity, risk factors, predictions elements

## Introduction

Adolescent idiopathic scoliosis (AIS) is a three-dimensional vertebral deformity. It combines a lateral curvature on the frontal plane >10° measured according to the Cobb method, vertebral rotation on the horizontal plane, and a disorder on the sagittal plane.[1-3] It is a pathology of growth that evolves until bone maturity.[4-7]

The Scoliosis Research Society has estimated the prevalence of idiopathic scoliosis 2%–3% in children under 16 with a curvature of 10° or more[8] and in between 0.3% and 0.5% for curvatures of 20° or more and in between 0.1% and 0.3% for curvatures of 30° or more (Weinstein, 1999).[9] Girls remain the most affected with a sex ratio of 10:1 for scoliosis of 20° and more.[10,11]

AIS cause remains unknown, several etiopathogenesis are evoked among them the abnormalities of the connective tissue particularly that of the collagen which is at the origin of the ligament hyperlaxity.[12-15] It is defined as a capacity for joint movement that exceeds normal limits. There are several nosological entities:[16]

Generalized joint hypermobility (GJH) can be pauciarticular (less than five joints involved) or polyarticular and concerns the small and large joints.[17-24]

Joint hypermobility syndrome (JHS) which is a syndrome in which GJH is associated with joint pain.[25]

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Benign joint hypermobility syndrome (BJHS), represents type 3 or the hypermobile form of Ehlers–Danlos Syndrome.[26]

The clinical diagnosis of GJH can be made according to different methods. We cite the Beighton score, the Bulbena score, and the Beighton associated with a 5-part questionnaire where the previous history of laxity in adolescents is evaluated.[27]

JHS is diagnosed using the Brighton score, which combines laxity, joint pain, tendinopathy, marfanoid syndrome and/or vascular fragility.[23]

BJHS is the hypermobile form of Ehlers–Danlos syndrome (type 3), it associates laxity, cracking with instability and joint dislocation, and visceral signs such as prolapse.[28]

To retain these clinical forms, it is first necessary to rule out inherited connective tissue disorders such as osteogenesis imperfecta, Marfan's disease, Ehlers–Danlos syndrome, Down syndrome, and Larsen's disease.[29]

The objective of our study is to investigate the relationship between ligament hyperlaxity and AIS aged between 9 and 15 years using logistic regression.

## Materials and Methods

We carried out a case–control study matched on age and sex, in the commune of Douera from March 2014 to March 2020.

The case is an adolescent with idiopathic scoliosis with a Cobb angle ≥10° associated with the presence of vertebral rotation, aged between 9 and 15 years old. He must not have any underlying pathology that could be the cause of this scoliosis.

The witness is a healthy adolescent between 9 and 15 years old who does not present any deformity whether vertebral or other. He must be the same age and gender as the case.

The inclusion criteria are adolescents aged between 9 and 15 years old, enrolled in the primary and middle cycle in public schools in the municipality of Douéra and adolescents followed at the level of our specialist consultation in vertebral deformities of Physical Medicine and Rehabilitation (PMR) randomly.

The criteria for noninclusion are adolescents presenting with vertebral deformities other than scoliosis, adolescents presenting with scoliosis other than idiopathic etiology. The investigation was incomplete.

According to the Kelsey Formula, on the Dean AG, Sullivan KM, Soe MM. OpenEpi: Open Source Epidemiologic Statistics for Public Health the Number of Necessary Subjects is 330 including 165 cases and 165 controls. We have gathered 430 subjects among them 215 cases and 215 controls.

The explanatory variables of the logistic regression are ligament hyperlaxity (the positive case must have a Beighton score ≥4), similar cases of AIS in the family, the weight of the school bag in kilograms, the practice of a sports activity, the consanguinity of the parents, the age of puberty, the weight, the body mass index (BMI), the growth spurt of the trunk is defined by the measurement of the sitting height every 6 months, this must be ≥4 cm, the distance between the fingers and the floor : Distance fingers ground (DFG) different or equal to 0, the footprint looking for a hollow foot, the bone maturity following the stages of Risser from 0 to 5.

We created a final model using the step-by-step descending procedure with the application of likelihood ratios. For each variable retained in the model, the adjusted odds ratios (ORs) and their 95% confidence intervals (CIs) were measured.

Data entry and analysis were performed using Statistical Package for the Social Sciences Chicago, Illinois, USA.

## Results

In our sample, there were no significant differences between the two case and control groups with respect to standing height, arm span, sitting height, weight and BMI, distance fingers ground, and school bag weight.

As for the age of puberty, we noticed that the scoliotics had presented an earlier puberty than the controls with a significant difference.

In our study, the age of puberty is equal to 12.66 ± 1.21 years in the overall sample. Scoliotics have a slightly earlier puberty age than nonscoliotics 12.47 years versus 12.87 years, with a significant difference ($P < 0.03$), which does not agree with other documented results. We found in the scoliotic group many more pubescent adolescents than in the control group (43.3% vs. 36.7%) without any significant difference ($P < 0.2$). Our finding remains a statistical event that must be verified later [Table 1].

Difference between the two groups [Table 2].

More than 50% of the subjects had a Risser = 0, this result was identical in the two groups [Table 3].

For the scoliotic group, the largest Cobb angle was located at the thoracic level; it is 16.13° ±10.96°, with a double curvature in half of the cases.

Age matching explains why the mean ages in the case and control groups are strictly the same with a $P = 1$. We observed a slight female predominance (sex ratio (female/male) =1.42). This is related to the nature of the pathology which is more common in girls. Matching on gender was well done ($P = 1$). The frequency of GJH in scoliotics in our study is 61.4%. This rate is significantly higher than that of the controls with a significant *P*. Adolescents with joint hyperlaxity are almost twice as likely to have idiopathic scoliosis as controls [Table 4].

The mean Beighton score in the scoliotic group was 4.05 ± 2.39, which was in favor of ligament hyperlaxity, while it was 3.16 ± 2.5 in the control group with a significant difference ($P < 10^{-3}$) [Table 5].

**Table 1: Results of anthropometric and clinical parameters**

| Sample characteristics | Mean±SD | | | *P* |
|---|---|---|---|---|
| | Total (*n*=430) | Positive scoliosis (*n*=215) | Negative scoliosis (*n*=215) | |
| Anthropometric parameters | | | | |
| Standing height (cm) | 150.26±12.83 | 151.14±12.11 | 149.38±13.49 | 0.15 |
| Arm span (cm) | 155.41±53.52 | 154.49±12.53 | 156.34±14.73 | 0.72 |
| Sitting height (cm) | 75.54±6.52 | 76.07±6.08 | 75.01±6.9 | 0.09 |
| Weight (kg) | 41.64±11.99 | 41.91±11.45 | 41.37±12.54 | 0.64 |
| BMI (kg/m$^2$) | 18.10±3.21 | 18.05±3.14 | 18.15±3.29 | 0.73 |
| Clinical parameters | | | | |
| Puberty age (years) | 12.66±1.21 | 12.87±1.09 | 12.47±1.28 | 0.03 |
| DFG (cm) | 11.30±10.54 | 11.14±10.47 | 11.47±10.63 | 0.74 |
| School bag weight (Kg) | 9.01±2.32 | 9.07±2.39 | 8.95±2.25 | 0.58 |

BMI: Body mass index, DFG: Distance fingers ground, SD: Standard deviation

**Table 2: Secondary sexual characteristics according to tanner stages**

| Tanner stages | Total (*n*=430), *n* (%) | Positive scoliosis (*n*=215), *n* (%) | Negative scoliosis (*n*=215), *n* (%) | *P* |
|---|---|---|---|---|
| S1P1 or T1P1 | 116 (27) | 52 (24.2) | 64 (29.8) | 0.35 |
| S2P2 or T2P2 | 81 (18.8) | 37 (17.2) | 44 (20.5) | |
| S3P3 or T3P3 | 53 (12.3) | 30 (14) | 23 (10.7) | |
| S4P4 or T4P4 | 15 (3.5) | 10 (4.6) | 5 (2.3) | |
| Puberty | 165 (38.4) | 86 (40) | 76 (36.7) | |

**Table 3: Bone maturity according to risser stages**

| Risser | Total (*n*=430), *n* (%) | Positive scoliosis (*n*=215), *n* (%) | Negative scoliosis (*n*=215), *n* (%) | *P* |
|---|---|---|---|---|
| 0 | 233 (54.2) | 113 (52.6) | 120 (55.8) | 0.83 |
| 1 | 42 (9.8) | 22 (10.3) | 20 (9.3) | |
| 2 | 60 (14) | 32 (14.9) | 28 (13) | |
| 3 | 65 (15) | 35 (16.2) | 30 (14) | |
| 4 | 30 (7) | 13 (6) | 17 (7.9) | |

**Table 4: Evaluation of the link between ligament hyperlaxity and the occurrence of adolescent idiopathic scoliosis**

| | Total (*n*=430), *n* (%) | Positive scoliosis (*n*=215), *n* (%) | Negative scoliosis (*n*=215), *n* (%) | OR | CI (95%) | *P* |
|---|---|---|---|---|---|---|
| GJH positive | 232 (54) | 132 (61.4) | 100 (46.5) | 1.83 | 1.25-2.68 | 0.003 |
| GJH negative | 198 (46) | 83 (38.6) | 115 (53.5) | | | |

OR: Odd ratio, CI: Conference interval, GJH: Generalized joint hypermobility

**Table 5: Comparison of average ligament hyperlaxity between the two case and control groups**

| | Mean±SD | | | *P* |
|---|---|---|---|---|
| | Total (*n*=430) | Positive scoliosis (*n*=215) | Negative scoliosis (*n*=215) | |
| Beighton score | 3.56±2.48 | 4.05±2.39 | 3.16±2.5 | 0.001 |

SD: Standard deviation

We introduced 11 other predictive risk factors for idiopathic scoliosis into the study.

The bivariate analysis of each risk factor (initial model) allowed us to conclude that there is no significant difference between the two groups concerning the risk factors studied, with the exception of similar cases in the family, and a growth spurt on the trunk ≥4 cm every 6 months.

Two variables were retained in the logistic regression model because their $P \leq 0.20$. These are puberty and playing sports.

An interaction was individualized between growth spurt and bone maturity by the Breslow index test ($P < 0.006$). Thus, the two variables of the interaction were kept in the model.

The variables: ligament hyperlaxity, similar cases of scoliosis in the scoliotic family, and growth spurt with an increase in height

on the trunk ≥4 cm every 6 months were retained in the final model. The adjusted ORs and their 95% CIs were measured:

Adolescents with similar cases of scoliosis in their families are almost twice as likely to develop idiopathic CI scoliosis (1.24–3.03).

Adolescents who had a growth spurt on the trunk ≥4 cm/6 months were 1.62 times more likely to develop idiopathic scoliosis than other adolescents.

Hypermobile adolescents are 1.83 times more likely to develop idiopathic scoliosis than nonhypermobiles [Table 6].

## Discussion

The frequency of ligament hyperlaxity in the general population in our sample of 430 adolescents aged between 9 and 15 years is 54%. This is similar to the other figures, emphasizing that this frequency is inversely proportional to the age of the children, as demonstrated by De Inocencio Arocena on a sample of 222 children aged between 4 and 14 published in 2004 by finding a frequency of 55% and Lamari on a sample of 1120 children aged between 4 and 7 years old, finding a percentage of 64.6%.[30]

Comparison of risk factors linked to the occurrence of AIS with other studies:

Regarding ligament hyperlaxity, our results OR = 1.82, confident interval (CI) (1.23–2.69) ($P < 0.003$) confirmed the link of GJH to AIS, which joins the data of other authors who used different scores. Czaprowski on adolescents aged between 9 and 18 years used the Beighton with the 5-part Questionnaire in an unpaired study carried out on a sample of 128 with an OR of 4.5, CI (2.01–10.13) ($P < 10^{-4}$) and the Beighton only in another paired study on a sample of 356 adolescents found an OR = 1.95, CI (1.12–3.38) ($P < 0.02$),[31,32] Binns, in his unpaired study of 500 Chinese adolescents with AIS, used the Marshall score, which looks at thumb mobility only. He found a decrease in the distance between the thumb and the forearm measured during passive opposition.[33] This encourages screening for AIS with possible follow-up in children with ligament hyperlaxity.

As for similar cases in the family, Lenz in his systematic review on the predictors of the progression of idiopathic scoliosis published in March 2021: "The cause of scoliosis seems to be based on several hereditary notions and this with the discoveries of the genetic aspects in the AIS."[34] Our study, which found an OR = 1.94, CI (1.24–3.3) $P < 0.002$, is in agreement with Watanabe's work, which concludes that the risk is 1.5 times higher for participants whose mothers had scoliosis[35] and the work of Grauers which reports that the chances of having a Cobb angle >40° if the patient had a family history are multiplied by 1.30 CI (1.05–1.6).[36]

It would be necessary to systematically examine the siblings of a scoliotic child who presents similar cases in his small and/or large family, as part of the screening.

The third risk factor found, which is the growth spurt, was analyzed by Ylikoski in 2005, who concluded that the progression of curves was notable if the growth rate ≥2 cm/year, the age between 9 and 13 years, bone ages between 9 and 14 years, Risser from 0 to 1 between 6 months and 2 years before menarche.[37] Chazono in 2012 published that the speed of growth spurt in healthy girls was equivalent to 8.3 cm/year in North Americans and 7 cm/year in the Taiwanese population when it was the speed of peak growth rate in scoliotic girls was 9.5 cm/year in the Americans and 8.5 cm/year in the Japanese, noting that the speed of the thrust on the trunk was high over a short period of growth.[38,39] This is in agreement with our growth spurt study on blood pressure ≥4 cm/6 months OR = 1.6 CI (1.09–2.34) $P < 0.02$.

## Conclusion

Our study evaluated the potential relationship between AIS and generalized joint hyperlaxity (GJH), by comparing in an age- and sex-matched case–control study, the scoliotic group to a nonscoliotic group.

To make an analysis of the literature allowed to put point out several risk factors of occurrence of the AIS having a different etiopathogenesis.

Through a logistic regression, these factors made it possible to confirm the potentiality of this relationship.

GJH increases the likelihood of the occurrence of AIS in adolescents aged between 9 and 15 years. It represents one of the risk factors predicting the onset of AIS.

The others being the presence of similar cases of idiopathic scoliosis in the subject's family and trunk growth spurt of 4 cm or more per 6 months.

No association was found for the other parameters studied.

**Table 6: Final model**

| Variables | Positive scoliosis ($n=215$), $n$ (%) | Negative scoliosis ($n=215$), $n$ (%) | OR raw | CI 95% | OR adjusted | CI 95% | $P$ |
|---|---|---|---|---|---|---|---|
| Interrogatory | | | | | | | |
| Family similar case | 72 (33.5) | 43 (20) | 2.01 | 1.3-3.12 | 1.94 | 1.24-3.03 | 0.002 |
| Physical examination | | | | | | | |
| Growth spurt ≥4 cm/6 months | 112 (52.1) | 87 (40.5) | 1.6 | 1.09-2.34 | 1.62 | 1.09-2.4 | 0.02 |
| GJH Beighton ≥4 | 132 (61.4) | 100 (46.5) | 1.83 | 1.25-2.68 | 1.82 | 1.23-2.69 | 0.003 |

OR: Odd ratio, CI: Conference interval, GJH: Generalized joint hypermobility

The question of GJH in its connection with idiopathic scoliosis seems clinically important because these children can be subjected to intensive physiotherapy according to various approaches ("schools"). These therapeutic activities include flexibility exercises, passive stretching, as well as proprioception. It seems crucial to assess the existence of GJH in these patients.

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## Conflicts of interest

There are no conflicts of interest.



## References

1. Green BN, Johnson C, Moreau W. Is physical activity contraindicated for individuals with scoliosis? A systematic literature review. J Chiropr Med 2009;8:25-37.
2. Beauséjour M, Roy-Beaudry M, Goulet L, Labelle H. Patient characteristics at the initial visit to a scoliosis clinic: A cross-sectional study in a community without school screening. Spine (Phila Pa 1976) 2007;32:1349-54.
3. Sabirin J, Bakri R, Buang SN, Abdullah AT, Shapie A. School scoliosis screening programme-a systematic review. Med J Malaysia 2010;65:261-7.
4. Bauknecht K. Scoliosis dance therapy: A worth-while addition to conservative scoliosis treatments? A pilot study evaluating the effect of a DVD led instruction on the wellbeing of scoliosis sufferers. Scoliosis 2012;7:P24.
5. Bialek M, M'hango A. «FITS» concept functional individual therapy of scoliosis. Stud Health Technol Inform 2008;135:250-61.
6. Rogala EJ, Drummond DS, Gurr J. Scoliosis: Incidence and natural history. A prospective epidemiological study. J Bone Joint Surg Am 1978;60:173-6.
7. Yaman O, Dalbayrak S. Idiopathic scoliosis. Turk Neurosurg 2014;24:646-57.
8. Altaf F, Gibson A, Dannawi Z, Noordeen H. Adolescent idiopathic scoliosis. BMJ 2013;346:f2508.
9. Ohtsuka Y, Yamagata M, Arai S, Kitahara H, Minami S. School screening for scoliosis by the Chiba University medical school screening program. Results of 1.24 million students over an 8-year period. Spine (Phila Pa 1976) 1988;13:1251-7.
10. Ondrašík M, Rybár I, Rus V, Bošák V. Joint hypermobility in primary mitral valve prolapse patients. Clin Rheumatol 1988;7:69-73.
11. Konieczny MR, Senyurt H, Krauspe R. Epidemiology of adolescent idiopathic scoliosis. J Child Orthop 2013;7:3-9.
12. Dayer R, Haumont T, Belaieff W, Lascombes P. Idiopathic scoliosis: Etiological concepts and hypotheses. J Child Orthop 2013;7:11-6.
13. Wai M, Jun W, Yee Y, Ho W, Bun N, Ping L, *et al*. A review of pinealectomy-induced melatonin-deficient animal models for the study of etiopathogenesis of adolescent idiopathic scoliosis. Int J Mol Sci 2014;15:16484-99.
14. Nachemson AL, Sahlstrand T. Etiologic factors in adolescent idiopathic scoliosis. Spine 1977;2:176-84.
15. Trobisch P, Suess O, Schwab F. Idiopathic scoliosis. Dtsch Arztebl Int 2010;107:875-83.
16. Sherry DD, Malleson PN. The idiopathic musculoskeletal pain syndromes in childhood. Rheum Dis Clin North Am 2002;28:669-85.
17. Grahame R. Joint hypermobility and genetic collagen disorders: Are they related? Arch Dis Child 1999;80:188-91.
18. Chevallier B, Benoist G, Jouneaux M, Stheneur C, Germain D. Syndromic hypermobility in children and adolescents. Arch Pédiatr 2014;21:212-4.
19. Jansson A, Saartok T, Werner S, Renström P. General joint laxity in 1845 Swedish school children of different ages: Age- and gender-specific distributions. Acta Paediatr 2004;93:1202-6.
20. Grahame R, Bird HA, Child A. The revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS). J Rheumatol 2000;27:1777-9.
21. Grahame R, Edwards JC, Pitcher D, Gabell A, Harvey W. A clinical and echocardiographic study of patients with the hypermobility syndrome. Ann Rheum Dis 1981;40:541-6.
22. Grahame R, Bird H. British consultant rheumatologists' perceptions about the hypermobility syndrome: A national survey. Rheumatology (Oxford) 2001;40:559-62.
23. Nyffeler R, Grobety T. Conservative rehabilitation of adults with hypermobility syndrome. Bachelor thesis: Haute Ecole de Santé Vaud; 2014.
24. Qvindesland A, Jónsson H. Articular hypermobility in Icelandic 12-year-olds. Rheumatology (Oxford) 1999;38:1014-6.
25. Haumont T. Postural instability in early adolescent idiopathic scoliosis. Doctoral dissertation, Nancy 1 (2010).
26. Remvig L, Jensen DV, Ward RC. Are diagnostic criteria for general joint hypermobility and benign joint hypermobility syndrome based on reproducible and valid tests? A review of the literature. J Rheumatol 2007;34:798-803.
27. Cheng JC, Chan PS, Hui PW. Joint laxity in children. J Pediatr Orthop 1991;11:752-6.
28. Beighton P, De Paepe A, Steinmann B, Tsipouras P, Wenstrup RJ. Ehlers-Danlos syndromes: Revised nosology, Villefranche, 1997. Ehlers-Danlos national foundation (USA) and Ehlers-Danlos support group (UK). Am J Med Genet 1998;77:31-7.
29. Tofts LJ, Elliott EJ, Munns C, Pacey V, Sillence DO. The differential diagnosis of children with joint hypermobility: A review of the literature. Pediatr Rheumatol Online J 2009;7:1.
30. Hakim AJ, Sahota A. Joint hypermobility and skin elasticity: The hereditary disorders of connective tissue. Clin Dermatol 2006;24:521-33.
31. Czaprowski D, Kotwicki T, Pawłowska P, Stoliński L. Joint hypermobility in children with idiopathic scoliosis: SOSORT award 2011 winner. Scoliosis 2011;6:22.
32. Czaprowski D. Generalised joint hypermobility in caucasian girls with idiopathic scoliosis: Relation with age, curve size, and curve pattern. ScientificWorldJournal 2014;2014:370134.
33. Binns M. Joint laxity in idiopathic adolescent scoliosis. J Bone Joint Surg Br 1988;70:420-2.
34. Lenz M, Oikonomidis S, Harland A, Fürnstahl P, Farshad M, Bredow J, *et al.* Scoliosis and prognosis-a systematic review regarding patient-specific and radiological predictive factors for curve progression. Eur Spine J 2021;30:1813-22.
35. Fadzan M, Bettany-Saltikov J. Etiological theories of adolescent idiopathic scoliosis: Past and present. Open Orthop J 2017;11:1466-89.
36. Reamy BV, Slakey JB. Adolescent idiopathic scoliosis: Review and current concepts. Am Fam Physician 2001;64:111-6.
37. Ylikoski M. Growth and progression of adolescent idiopathic scoliosis in girls. J Pediatr Orthop B 2005;14:320-4.
38. Chazono M, Soshi S, Kida Y, Hashimoto K, Inoue T, Nakamura Y, *et al.* Height velocity curves in female patients with idiopathic scoliosis. Res Spinal Deform 2012;176:202-5.
39. Ramirez-Moreno J. The pathogenesis of idiopathic scoliosis and the Mézières method Bibliographic review of the literature. Spain: International University of Catalonia (Barcelona); 2014.