Journal of Orthopaedic Translation 22 (2020) 142

Contents lists available at ScienceDirect

Journal of Orthopaedic Translation

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LETTER TO THE EDITOR

Letter to the editor concerning "Imbalanced development of anterior and posterior thorax is a causative factor triggering scoliosis" by Chen et al., *Journal of Orthopaedic Translation*, 2019, https://doi.org/10.1016/j.jot.2018.12.001

Dear Editor,

With great interest, we have read "Imbalanced development of anterior and posterior thorax is a causative factor triggering scoliosis" by Chen et al. [1] and would like to compliment the authors on their article. In this article, the authors described a theory about the origin of idiopathic scoliosis, a topic that has received much interest over a long period of time. Patients with scoliosis were found to have a reduced thoracic anteroposterior length ratio, resulting from the relatively longer thoracic vertebra and relatively shorter sternum. In addition, artificially reducing the thoracic anteroposterior length ratio by surgically destroying the sternal growth plates induces scoliosis in quadrupedal mice and even a more increased scoliosis curvature in bipedal mice. Consistently, mice with deficiency of fibroblast growth factor receptor 3, which had severe scoliosis, had a reduced thoracic anteroposterior length ratio. The authors interpreted these findings as an imbalanced growth between the thoracic vertebral column and the sternum that may also play a role in the pathogenesis of idiopathic scoliosis. Their hypothesis was that the gradually reduced relative length of the sternum induced scoliosis.

Besides the reduced length of the sternum, the well-known so-called "relative anterior overgrowth" of the spine is described as well in the introduction section as a result of loss of coupling between endochondral ossification and intramembranous ossification during the growth spurt [1]. It is well known that the anterior part of the spine is longer than the posterior part in idiopathic scoliosis; this has been called relative anterior spinal overgrowth and has led to the assumption that idiopathic scoliosis may be a problem of active overgrowth of the anterior osseous structures of the spine, as well as a possible growth discrepancy of the spinal cord as compared with the vertebrae. However, we recently described that relative anterior spinal overgrowth is observed only in the primary and compensatory curves of idiopathic scoliosis, whereas the junctional zones do not exhibit this growth discrepancy [2]. It is thus definitely not a generalized growth disturbance. Furthermore, the discs were found to contribute three times more to this anterior overgrowth than the vertebral bodies; the bony structures (vertebral bodies) showed an anterior–posterior ratio that is basically similar to nonscoliotic controls [2–5]. Finally, we found that in scoliosis with known origin, as neuromuscular scoliosis, this same phenomenon of additional anterior spinal length exists, also mainly in the disc [4]. These facts suggest that the observed anterior–posterior length discrepancy is not the cause of idiopathic scoliosis and that it is more a generalized passive phenomenon that occurs in all types of scoliosis, than active bony growth. Thus, length measurements along the anterior side of the spine in scoliosis will always result in a longer spinal column than in controls, but the authors do not mention the relative contributions of the disc as compared with the bone. However, we congratulate the authors on their article and their contribution to this interesting discussion.

Conflict of Interest

The authors have no conflicts of interest to disclose in relation to this article.

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https://doi.org/10.1016/j.jot.2019.07.008
Received 5 June 2019
Available online 27 August 2019