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Muscle Biology and the Pathogenesis of AIS: Insights from Electrophysiology, Imaging, Histology, and Molecular Genetics

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Editorial

Studies have indicated that patients with idiopathic scoliosis (IS) commonly present with an imbalance of the paraspinal muscles regarding the orientation, length, thickness, volume, cross sectional area, and fiber type composition [1–4]. However, whether paraspinal muscle imbalance is the cause or the consequence of spinal deformities in IS, remains unclear [4].

Generally speaking, studies that tried to answer this question can be collected under 4 titles: electrophysiological studies [5–11], imaging muscle studies e.g 3-dimensional magnetic resonance imaging (MRI) or ultrasonography [12,13], muscle histopathologic studies [1,11–18] and recently molecular genetic studies [19–21]. Muscle biopsies showed that in adolescent idiopathic scoliosis (AIS), type I fibers are predominant on the convex side, while there are more type II fibers on the concave side. According to the proponents of the cause theory, the presence of more type 1 fibers on the convex side may provide a sustained pull on the spine, resulting in scoliosis [1,14,15]. The proponents of the effect theory considered that lower proportion of type I fibers on the concave side may be a result of the disuse of the paraspinal muscles associated with spinal deformity [1,2]. Myoelectrical studies like surface electromyography (S-EMG) showed conflicting results. S-EMG activity was found to be higher on the convex side of the scoliotic curve; these findings suggest an overactivation of the paraspinal muscles as a cause of AIS [5–8,10]. Conversely, one study reported that there was no significant difference in electromyographic amplitude of erector spinae on the convex and concave sides [8]. These conflicting results might be due to methodological differences, based on patient selection, curve type or lack of controlling mechanisms to exclude inadequate posture [5,9,10,22,23].

Molecular genetic studies showed that he AIS susceptibility gene *Tent5a* was differentially expressed in AIS paravertebral muscles. *Tent5a* plays an important role in the proliferation and migration of myoblasts, and it regulates muscle fiber maturation by maintaining the stability of myogenin. *Tent5a* knockdown inhibited the proliferation and migration of C2C12 cells and inhibited the maturation of type I muscle fibers *in vitro* and *in vivo*. Mechanistically, the expression of myogenin was decreased along with the suppression of *Tent5a* [19]. Jiang *et al*., (2018) studied gene expression levels of *H19* (a long noncoding RNA) and *ADIPOQ* (encoding adiponectin) in paravertebral muscle samples obtained from concave and convex sides of patients undergoing surgery for severe AIS. The convex side showed higher expression of *H19* and *ADIPOQ*. This asymmetry correlated with greater adipogenic differentiation and muscle imbalance. These molecular differences might contribute to the asymmetric muscle development seen in AIS. The study suggests that the H19–ADIPOQ pathway contributes to asymmetric adipogenesis and muscle imbalance in AIS, providing molecular evidence that paravertebral muscle asymmetry is not merely a result of spinal curvature but may actively participate in scoliosis pathogenesis [20].

Zhang *et al.* (2025) performed morphological and molecular analysis of neuromuscular junctions (NMJs) in paraspinal muscles from AIS patients and also in patients with congenital scoliosis (CS) and non- scoliosis controls (NSC). Morphological analysis of NMJs showed that the nerve terminal-related variables in the convex side were significantly decreased. The expression of denervation markers was increased in the synapse-rich regions as well as in the convex side paraspinal muscles. Compared with CS and NSC, paraspinal muscles of AIS showed the phenomenon of fiber-type grouping,

confirming the presence of neurogenic abnormalities. This study proved that neurogenic abnormalities existed in the paraspinal muscle of the convex side, which could lead to the conversion and grouping of fiber types. This resulted in an imbalance of paraspinal muscles and might be a potential driver of scoliosis. [21]

It is too early to conclude that paraspinal muscles are the key player in causation of IS. Molecular studies have revealed a complex landscape of genetic variability in individuals with AIS, with inconsistent findings and numerous candidate genes proposed to be associated with the condition [24]. In the future, a more integrated approach combining orthopedic and molecular research may help elucidate the full pathophysiology of AIS and determine whether paraspinal muscle abnormalities play a primary role in its development, represent secondary changes to the deformity, or involve elements of both mechanisms.

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