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Transcriptional Activity of Vitamin D Receptor Gene (VDR) and Transforming Growth Factor Beta (TGF- B) Signaling Differentiate Juvenile and Adolescent Idiopathic Scoliosis

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Short Communication

Idiopathic scoliosis (IS) is the most common spinal deformity in humans. The exact causes of this condition are unknown but the genetic background seems undisputable. Genetic background doesn't preclude that IS could be a systemic molecular disorder reflected by local processes affecting musculoskeletal structures of the spine during periods of intensive growth. The age of scoliosis onset is one of the most important factors determining the natural history and the risk of progression. Most of the idiopathic curves are diagnosed in juvenile (Juvenile Idiopathic Scoliosis -JIS) or adolescent period (Adolescent Idiopathic Scoliosis - AIS). Transcriptional profile of candidate genes potentially related to IS susceptibility and natural history may be changed in the structural tissues of the scoliotic spine and potentially influence responsive signaling pathways. In our two recent studies we investigated with the use of qRT-PCR and HG-U133A microarrays the transcriptional activity of several candidate genes that could potentially be involved in the pathogenesis of IS. The analyzed transcriptomes were grouped according to the side of the curve and the age of scoliosis onset (JIS and AIS). The results of the first study indicated that in IS transcriptional activity and alternative splicing of vitamin D receptor gene (VDR) are tissue specific and equal on both sides of the scoliotic curve. The mRNA abundance of long VDR isoform in concave paravertebral muscles appeared to be related with the age of scoliosis onset. Tob2 and Med13 genes were selected among vitamin D responsive genes as genes differentiating paravertebral muscle transcriptomes of JIS and AIS [1]. The results of the second study pointed at the possibility of different involvement of transforming growth factor beta (TGF-beta) signaling in the pathogenesis of curves with different age of onset. Analysis of the TGF-beta responsive genes differentiating both sides of the paravertebral muscle transcriptomes of AIS patients highlighted the upregulation of genes localized in the extracellular region of the curve concavity (latent TGF-beta binding proteins: LTBP3, LTBP4 and integrins: ITGB4, ITGB5). This could suggest extracellular matrix of paravertebral muscles as an interesting target for future molecular research [2]. At present treatment of IS is symptomatic and involves bracing and severe surgery. The identification of the molecular factors and signaling pathways involved in etiology and pathogenesis could suggest new biomarkers of progression risk and may help to develop new drugs to prevent or combat this crippling deformity.

References

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