

IL23R and COL1A1 genes as key determinants of inflammatory processes and collagen strength in intervertebral structures

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Abstract:

The IL23R and COL1A1 genes control immune-inflammatory processes and the synthesis of type I collagen—key links in the pathogenesis of spinal degeneration [1, 2, 3]. Dysfunction of these genes contributes to increased inflammation and decreased collagen fiber strength, which increases the risk of recurrence of intervertebral disc herniations [4, 5].

Objective : To study the influence of IL23R (rs11209026) and COL1A1 (C/A) polymorphisms on the clinical course and relapses of MPGD.

Materials and methods.

The study included 92 patients with confirmed intervertebral disc herniation (IVDH) and 94 apparently healthy control volunteers. Genotyping was performed using polymerase chain reaction (PCR) followed by analysis of IL23R (A/G) and COL1A1 (C/A) polymorphisms. DNA was isolated using standard phenol-chloroform methods or commercial whole blood extraction kits. Allele and genotype frequencies were compared between groups using the χ^2 test, and associations with the risk of IVDH recurrence were assessed using the odds function. odds ratio (OR) with 95% confidence interval (CI).

Results.

The analysis showed that carriage of the A allele of the IL23R gene in patients with MPHD is associated with an increased likelihood of recurrence after surgery ($p < 0.05$). Furthermore, the AA genotype of the COL1A1 polymorphism significantly affects the structure of collagen fibers in the intervertebral disc, which is reflected in a nearly fourfold increase in the risk of recurrent disc herniation compared to other genotypes (OR ≈ 3.8 ; $p < 0.05$). These data confirm the

significant role of these genes in the regulation of inflammatory processes and the strength of spinal connective tissue.

Conclusion.

Polymorphisms of the IL23R and COL1A1 genes significantly influence inflammatory mechanisms and the structural strength of collagen fibers in intervertebral discs. The A allele of the IL23R gene promotes the activation of immune-inflammatory processes, increasing the risk of recurrence after surgery, while the AA genotype of COL1A1 disrupts the architecture of the collagen matrix, significantly increasing the likelihood of disc recurrence. These data confirm that these polymorphisms are key genetic markers determining the individual risk of degenerative and inflammatory changes in the spine and can be used to predict treatment outcomes and personalized rehabilitation for patients.

Literature.

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