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日本臨床免疫学会

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以上
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DNA methylome signature in rheumatoid arthritis

○中野和久1,2, Gary S Firestein2, 田中良哉1
(1産業医科大学 第一内科学講座, 2カリフォルニア大学サンディエゴ校 リウマチ・アレルギー・免疫科)

Epigenetics such as DNA methylation can potentially influence disease susceptibility and severity. While methylation of individual genes has been explored in autoimmunity, no systematic analyses have been reported. We performed an unbiased genome-wide evaluation of DNA methylation in fibroblast-like synoviocytes (FLS) isolated from rheumatoid arthritis (RA) synovium. Using the Infinium HumanMethylation450 BeadChip, cluster analysis of the methylation state was performed on 476,331 CpG loci. RA and control FLS unexpectedly segregated into separate groups based on DNA methylation. Hypomethylated loci were identified in key genes relevant to RA, such as CHIBL1, CASP3, and WISP3. Hypomethylated genes was associated with increased gene expression. Hypomethylation was increased in multiple pathways related to cell migration, including focal adhesion, cell adhesion, and extracellular matrix interactions. These pathways could contribute to migration of synoviocytes from the synovium to the surface of cartilage and other joints. DNA methylation of critical genes suggests that RA FLS are imprinted and implicate non-DNA encoding contributions to synovitis and joint damage.