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综述

系统性红斑狼疮（SLE）的表遗传学发病机制研究进展

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摘 要 表遗传学是研究DNA序列没有发生改变的情况下基因表达的遗传变化。研究表明DNA甲基化异常可能与系统性红斑狼疮（systemic lupus erythematosus, SLE）发病有关。DNA调节序列的低甲基化与B和T淋巴细胞的激活与分化有关。SLE患者血浆中低甲基化基因组DNA片段，可能模仿了微生物DNA，诱导了抗dsDNA抗体的生物合成。HERV序列、外源性逆转录病毒和核抗原显示出极度的同源性。外源性逆转病毒能够识别HERV抗原，增加抗DNA抗体的产生。针对病毒抗原的免疫反应可能被扩展到其它抗原，如核抗原或DNA片段，从而对SLE患者抗DNA抗体的生物合成产生影响。

关键词 系统性红斑狼疮 表遗传 DNA甲基化

分类号

The Epigenetic Pathogenesis of Systemic Lupus Erythematosus

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Abstract Epigenetics is defined as the investigation of heritable changes in gene in expression that occur without a change in DNA sequence。Several lines of evidence have indicated that abnormalities of DNA methylation may contribute to the development of SLE。It has been observed that hypomethylation of DNA regulatory sequences is involved in activation and differentiation of B and T lymphyocytes。The hypomethylated genomic DNA fragments in the plasma of SLE patients may induce biosynthesis of anti-dsDNA antibodies，which plays a role in the pathogenesis of SLE。The HERV sequences， exogenous retoviruses， and nuclear antigens exhibit profound homology。exogenous retroviruses are also able to recognise HERV antigens and augment production of anti-DNA antibodies

Key words Systemic lupus erythematosus Epigenetics DNA methylation

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