狼瘡病嗜中性白血球障礙
Neutrophil defect in SLE

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Polymorphonuclear neutrophils (PMNs) act by phagocytosis, and are regarded as the first line of defense against pathogen invasion. In addition, they have many previously unknown functions including mitogen-induced cell-mediated cytotoxicity, production of cytokines/chemokines/growth factors as well as release of neutrophil extracellular traps (NETs) and ectosomes/exosomes. There is also a trogocytosis (cell bite) following direct cell-cell contact with other immune cells for modulating innate and adaptive immunity. In other words, neutrophils play an important role in the immune network. Systemic lupus erythematosus (SLE) is characterized by multiple immune dysfunctions including excessive interferon alpha (IFN-α) expression, aberrant cytokine/chemokine/growth factor production, skewing of T cell immune responses toward Th2 and Th17 pathways, polyclonal B cell activation, increased apoptosis and NET formation, defective clearance of cell debris and NET-related molecules as well as abnormal ectosome/exosome release in plasma. SLE-PMNs *per se* exhibit aberrant cytokine/chemokine expression, defective glucose metabolism, and increased mitochondrial DNA D310 heteroplasmy with reduced redox capacity. The autoantibodies purified from SLE sera disrupt PMN functions. In the present review, we will show these abnormalities as well as a potential pathogenetic role of disrupted PMN functions in the development of SLE.