2 IMMUNE COMPLEX FORMATION AFTER EXPOSURE OF AUTOANTIGENS ON THE SURFACE OF SECONDARY NECROTIC CELLS (SNEC) PROMOTES INFLAMMATION IN SLE

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Background and objectives Systemic lupus erythematosus (SLE) is a chronic inflammatory disease characterised by the production of autoantibodies, formation of immune complexes (IC), and activation of complement. Accumulation in blood and tissues of postapoptotic remnants is considered of etiological and pathological importance for patients with SLE. The goal of this work is to analyse whether the remnants of apoptotic cells are involved in the induction of inflammatory responses in SLE.

Materials and methods SNEC were generated by UVB irradiation of human lymphocytes followed by incubation for 48 h. The authors employed fluorescence microscopy, FACS, and an ex vivo phagocytosis assays to demonstrate the availability of autoantigens on the surface of SNEC to form immune complexes that provoke inflammation.

Results The authors describe the exposure of dsDNA on the surface of SNEC and the binding of serum opsonins to SNEC. The authors show that anti-dsDNA and other autoantibodies bind and sensitise SNEC. Autoantibody-sensitised SNEC were cleared by macrophages in vitro and induced a pro-inflammatory cytokine response.

Conclusions Lupus autoantigens are exposed on the surface of SNEC and available to assemble intricate immune complexes with various autoantibodies. The clearance of cellular remnants that are not properly removed from the organism is targeted by lupus autoantibodies causing systemic inflammation in some patients with SLE.