

Publication

"The NET Outcome": Are Neutrophil Extracellular Traps of Any Relevance to the Pathophysiology of Autoimmune Disorders in Childhood?

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Author(s) Giaglis, S.; Hahn, S.; Hasler, P. **Author(s)** at **UniBasel** Hahn, Sinuhe;

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Neutrophil extracellular trap (NET) formation represents a form of cell death distinct from apoptosis or necrosis, by which invading pathogens are simultaneously entangled and potentially eliminated. Increased NET formation is observed in systemic lupus erythematosus (SLE), rheumatoid arthritis, antineutrophil cytoplasmic antibody-associated small vessel vasculitis, antiphospholipid antibody syndrome (APS), and psoriasis. NETs contribute to the pathogenesis of autoimmunity by exposing cryptic autoepitopes, which may facilitate the generation of autoantibodies, induce the production of interferons, and activate the complement cascade. In SLE, augmented disease activity and renal disease are associated with increased NET formation, so that NETs could serve as a marker for the monitoring of disease activity. NETs can additionally cause endothelial cell damage and death and stimulate inflammation in atheromatous plaques, adding to the accelerated atherosclerosis witnessed in autoimmune disease. Since NETs induce production of interferons, assessing the extent of NET formation might facilitate the prediction of IFN-alpha levels and identification of SLE patients with presumably better responses to anti-IFN-alpha therapies or other novel therapeutic concepts, such as N-acetyl-cysteine and inhibitors of DNase 1 and peptidylarginine deiminase 4 (PAD4), which also target NETs. In summary, the study of NETs provides a novel approach to the understanding of autoimmune disease pathogenesis in childhood and opens new vistas in the development of sensitive disease markers and targeted therapies.

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