

Factor H Binds to Extracellular DNA Traps Released from Human Blood Monocytes in Response to *Candida albicans*.

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Abstract

Upon systemic infection with human pathogenic yeast *Candida albicans* (*C. albicans*), human monocytes and polymorph nuclear neutrophilic granulocytes are the first immune cells to respond and come into contact with *C. albicans*. Monocytes exert immediate candidacidal activity and inhibit germination, mediate phagocytosis, and kill fungal cells. Here, we show that human monocytes spontaneously respond to *C. albicans* cells via phagocytosis, decondensation of nuclear DNA, and release of this decondensed DNA in the form of extracellular traps (called monocytic extracellular traps: MoETs). Both subtypes of monocytes (CD14(++)CD16(-)/CD14(+)CD16(++)) formed MoETs within the first hours upon contact with *C. albicans*. MoETs were characterized by the presence of citrullinated histone, myeloperoxidase, lactoferrin, and elastase. MoETs were also formed in response to *Staphylococcus aureus* and *Escherichia coli*, indicating a general reaction of monocytes to infectious microbes. MoET induction differs from extracellular trap formation in macrophages as MoETs are not triggered by

simvastatin, an inhibitor of cholesterol synthesis and inducer of extracellular traps in macrophages. Extracellular traps from both monocytes and neutrophils activate complement and C3b is deposited. However, factor H (FH) binds via C3b to the extracellular DNA, mediates cofactor activity, and inhibits the induction of the inflammatory cytokine interleukin-1 beta in monocytes. Altogether, the results show that human monocytes release extracellular DNA traps in response to *C. albicans* and that these traps finally bind FH via C3b to presumably support clearance without further inflammation.

Involved units

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