# The extracellular adherence protein (Eap) of *Staphylococcus aureus* exhibits DNase activity

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*Staphyloccocus aureus* is equipped with a number of virulence factors allowing it to modulate or circumvent the immune responses of the host. One of these factors is the extracellular adherence protein Eap. We and others have previously shown that Eap interferes with the host innate immune system by reducing NFκB activation in leukocytes, decreasing neutrophil extravasation, and blocking neutrophil serine protease activity. Here we report that Eap also provides exonuclease activity: Incubation of double-stranded DNA with Eap led to a rapid degradation of linearized DNA fragments. Atomic force microscopy confirmed that Eap binds to and degrades linearized DNA in a time-dependent manner, while circular DNA did not interact with Eap and remained undegraded. Eap binding preferentially occurred to the termini of the double-stranded polynucleotide chains of DNA and was not affected by the type of overhang. In a dose-dependent manner, Eap also inhibited/prevented formation of bacteria-killing “neutrophil extracellular traps” (NETs), which represent the entire chromatin content of neutrophils that becomes ejected by incubation of cells with various agonists. These data indicate that Eap, via its DNase-associated activity, appears to express another immune-evading function by degrading NETs and thereby destroying an effective anti-microbial mechanism of the host.