Hydrolysis of kininogens by degranulated human neutrophils and analysis of bradykinin as chemotactic factor for cells isolated from peripheral blood

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Abstract

Human neutrophils play a pivotal role in acute inflammation including the regulation of vascular permeability. We have examined the capacity of neutrophil enzymes to hydrolyse human kininogens in vitro and have also explored the potentiality of bradykinin to induce chemotactic migration on neutrophils isolated from peripheral blood. Isolated neutrophils were stimulated with either f-Met-Leu-Phe, thrombin or silica particles coated with human IgG. Neutrophil enzymes obtained by degranulation produced, after 45 min of incubation with high and low molecular weight kininogens, the complete transformation of both proteins in polypeptides ranging from 20 to less than 10 kDa in molecular mass. Supernatants obtained from nonstimulated neutrophils did not modify the molecular size of kininogens. The assay used to test the chemoattractant capacity of synthetic bradykinin on human neutrophils showed that this peptide has no chemotactic activity on cells isolated from healthy subjects. Our results show that stimulation of human neutrophils with opsonized silica, thrombin and the chemotactic peptide f-Met-Leu-Phe induces release of kininogen-hydrolyzing enzymes from these cells.