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Curcumin inhibits agent-induced human neutrophil functions in vitro and lipopolysaccharide-induced neutrophilic infiltration in vivo



Francis Antoine, Jean-Christophe Simard, Denis Girard *

Laboratoire de recherche en inflammation et physiologie des granulocytes, Université du Québec, INRS-Institut-Armand-Frappier, Laval, Quebec Canada

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ABSTRACT

Curcumin, extracted from the rhizome of Curcuma longa, is known to possess anti-inflammatory activities. Despite the fact that neutrophils are key player cells in inflammation, the role of curcumin on neutrophil cell biology is not well documented and, in particular, how curcumin can alter primed neutrophils is unknown. In addition, the effect of curcumin on agent-induced neutrophilic inflammation is not well documented. Here, we demonstrated that curcumin inhibited formyl-methionyl-leucyl-phenylalanine (fMLP)- or lipopolysaccharide (LPS)-induced suppression of human neutrophil apoptosis. In addition, we found that curcumin reversed the ability of phorbol myristate acetate (PMA) to induce reactive oxygen species as assessed by flow cytometry using the CM-H2DCF-DA probe. Using an antibody array approach, curcumin was found to inhibit LPS-induced cytokine production, including MIP-1α, MIP-1β, IL-6, IL-8 (CXCL-8) and GRO-α. The inhibitory effect of curcumin on IL-8 production was confirmed by ELISA. Using both an electrophoretic mobility shift assay and a TransFactor p50 NF-κB EUSA, we demonstrated that curcumin inhibited LPS-induced NF-κB activation. In vivo, using the murine air pouch model of acute inflammation, we demonstrated that intraperitoneal administration of curcumin inhibited LPS-induced neutrophilic infiltration in vivo. As assessed by a murine antibody array approach, curcumin was found to decrease the local production of several cytokines/chemokines induced by LPS, including, but not limit to, MIP-1α and MIP-1β. We conclude that curcumin possesses potent modulatory activities on primed or agent-induced human neutrophils in vitro and that it possesses important antiinflammatory activities in vivo by inhibiting LPS-induced neutrophilic inflammation.

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