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Intracellular generation of superoxide by human polymorphonuclear leukocytes stimulated by formyl-methionyl-leucyl-phenylalanine (FMLP). We have examined the ability of isolated human polymorphonuclear leukocytes (PMN) to generate superoxide following stimulation by the chemotactic peptide FMLP. PMN and monocytes are equally sensitive to the generation of O₂⁻ by FMLP, while macrophages are less sensitive. Despite an initial elevation in intracellular calcium, FMLP induces no further increase in cytosolic calcium concentration, nor does it cause an additional elevation in the formation of NADPH-cytochrome c reductase inactivated cytochrome b558.

FMLP-induced O₂⁻ production is dependent on the presence of metabolic energy (ATP).

Agents which affect metabolic energy production, such as N-ethylmaleimide, 2,4-dinitrophenol, and oligomycin, inhibit O₂⁻ production by FMLP, while agents which block the Na⁺, K⁺-ATPase, such as ouabain and monensin, stimulate O₂⁻ production. The characteristics of FMLP-induced O₂⁻ generation closely resemble the action of PMA. FMLP is incapable of stimulating the generation of O₂⁻ in the presence of a NADPH oxidase inhibitor (DPI), indicating that O₂⁻ is generated via a second mechanism in addition to that induced by PMA. FMLP-induced O₂⁻ generation is inhibited by the addition of 10 micromM (dihydro)furoic acid, suggesting that FMLP stimulates the formation of O₂⁻ via a peroxidase activity associated with the enzyme cytochrome b558. These data indicate that PMN possess a FMLP receptor which is coupled to the production of superoxide and that O₂⁻ generation by PMN is dependent on the same metabolic energy and cyclic AMP-related mechanisms which regulate PMA-induced O₂⁻ generation.

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