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Inhibitory actions of glucosamine, a therapeutic agent for osteoarthritis, on the functions of neutrophils.

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Abstract

Glucosamine, an amino monosaccharide naturally occurring in the connective and cartilage tissues, contributes to maintaining the strength, flexibility, and elasticity of these tissues. In recent years, glucosamine has been used widely to treat osteoarthritis in humans and animal models. Neutrophils, which usually function as the primary defenders in bacterial infections, are also implicated in the destructive, inflammatory responses in arthritis. In this study, we have evaluated the effects of glucosamine on neutrophil functions using human peripheral blood neutrophils. Glucosamine (0.01-1 mM) dose-dependently suppressed the superoxide anion generation induced by formyl-Met-Leu-Phe (fMLP) or complement-opsonized zymosan and inhibited the phagocytosis of complement-opsonized zymosan or IgG-opsonized latex particles. Furthermore, glucosamine inhibited the release of granule enzyme lysozyme from phagocytosing neutrophils and suppressed neutrophil chemotaxis toward zymosan-activated serum. In addition, glucosamine inhibited fMLP-induced up-regulation of CD11b significantly, polymerization of actin, and phosphorylation of p38 mitogen-activated protein kinase (MAPK). In contrast, N-acetyl-glucosamine, an analogue of glucosamine, did not affect these neutrophil functions (superoxide generation, phagocytosis, granule enzyme release, chemotaxis, CD11b expression, actin polymerization, and p38 MAPK phosphorylation) at the concentrations examined (1-10 mM). Together these observations likely suggest that glucosamine suppresses the neutrophil functions, thereby possibly exhibiting anti-inflammatory actions in arthritis.

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