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FILE: ▪ Ginger (*Zingiber officinale*)
▪ Alzheimer's Disease
▪Inflammation

HC040456-287

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RE: Ginger Extract May Offers a Safe Treatment to Protect Against Alzheimer's Disease Inflammation

Grzanna R, Phan P, Polotsky A, Lindmark L, Frondoza CG. Ginger extract inhibits beta-amyloid peptide-induced cytokine and chemokine expression in cultured THP-1 monocytes. *J Altern Complement Med*. December 2004;10(6):1009-1013.

Fibrillar β-amyloid peptide is prevalent in brains of people with Alzheimer's disease. β-amyloid peptide causes microglial cells (cells in the brain that provide support) to increase production of cytokines and chemokines. Cytokines and chemokines are substances in the body that promote inflammation. Some hypotheses state that inflammation plays a key role in the pathogenesis of Alzheimer's disease. Ginger (*Zingiber officinale*) has anti-inflammatory properties. The purpose of this study was to determine whether a ginger extract could dampen the induction of inflammation-related genes in human THP-1 cells exposed to lipopolysaccharide (LPS), proinflammatory, cytokines, and fibrillar amyloid peptide Aβ(1-42), a major component of neuritic plaques.

In vitro, human monocyte THP-1 cells (a model of human microglial cells) were treated with reincubated LPS, the proinflammatory cytokines tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β), the chemokines monocyte chemoattractant protein-1 (MCP-1), macrophage inflammatory protein 1α (MIP-1α), and interferon-gamma inducible protein 10 (IP-10), the proinflammatory mediator cyclooxygenase-2 (COX2), or ginger extract. The ginger preparation was a standardized extract of the dry rhizomes of *Z. officinale* (3000 mg) and greater galangal (a.k.a. Chinese/Siamese ginger) *Alpinia galangal*) (500 mg). After treatment, RNA was extracted from the THP-1 cells and the genes were evaluated.

As expected, the proinflammatory mediators caused upregulation of inflammatory markers. The ginger extract had a broad inhibitory effect on cytokine and β-amyloid peptide induced expression of inflammation-related genes in activated THP-1 cells. The extract inhibited the induction of genes involved in the inflammatory cascade (TNF-α ,

IL-1β, MCP-1, MIP-1α, IP-10, and COX2). The ginger extract has a mechanism of action that is different from that of nonsteroidal anti-inflammatory drugs.

The authors conclude that this ginger extract may help prevent the sustained elevation of the proinflammatory cytokines, as would be seen in people with Alzheimer's disease. The authors believe that the ginger extract offers a promising and safe treatment to protect against the harmful effects of chronic microglial cell-associated inflammation in Alzheimer's disease. The next logical step would be to test the extract in vivo.

—Heather S. Oliff, PhD

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