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## Letter to the Editor



Hamid Namazi

## The role of garlic in the prevention of ischemia-reperfusion injury: A new mechanism

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I read with great interest the article by Sener *et al.* [1]. They discuss that garlic inhibits migration of neutrophils. I would like to complete the discussion of Sener *et al.* [1] by introducing a major route through which garlic could suppress the activity of neutrophils.

The recent focus on ischemia-reperfusion injury has been on interaction between neutrophils and endothelial cells. The injury attributed to plugging of the microvasculature by neutrophils may initiate the cascade of injury by releasing free radicals, enzymes, and cytokines and physically injuring the endothelium and obstructing the capillaries, thus impairing oxygen supply to the tissue. Also transendothelial migration of neutrophils, with release of reactive oxygen species and cytokines, causes further damage to the injured tissue [2, 3]. However, a key component in the pathogenesis of reperfusion syndrome is the upregulation of surface adhesion molecules on the vascular endothelium and their subsequent interaction with the activated neutrophils [4]. The most important adhesion protein identified

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on neutrophils is the integrin lymphocyte function-associated antigen-1 (LFA-1; CD11a/CD18), which is the ligand for intercellular adhesion molecule-1 (ICAM-1) expressed on the endothelium. The LFA-1/ICAM-1 interaction is crucial for the ingress of neutrophils into the inflammatory sites [5, 6]. Garlic downregulates the expression of ICAM-1 and LFA-1, and through binding to LFA-1, they interfere with ICAM-1–LFA-1 interaction [7, 8]. This important mechanism should be borne in mind as the major mechanism for garlic-induced inhibition of neutrophil activity.

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