Shouldering the Risk Burden: Infection, Atherosclerosis, and the Vascular Endothelium

To the Editors:

The recent editorial by Drs Vita and Loscalzo was most informative and presents an overview of the relationships among infection, atherosclerosis, and consequent endothelial dysfunction. Their conclusion that “chronic infection may be added to the list of factors that induce endothelial dysfunction” is certainly warranted.

Similarly, the scientific papers presented at the recent symposium in Washington, DC, *Infection and Atherosclerosis*, solidify the case for an infectious etiology of atherosclerosis.

Multiple causative organisms were mentioned, but only Dr Paul Ewald listed the recently discovered *Nanobacterium sanguineum* (Ns) as a possible etiologic infectious agent because of its unique ability to cause infection, calcium deposition, and inflammatory responses. Ns is currently under intense investigation in many academic medical institutions and government agencies.

Ns is the smallest known (20 to 200 nanometers) self-replicating organism and has been isolated from multiple human tissues, including atherosclerotic plaque. Ns also produces a lipopolysaccharide biofilm that induces the inflammatory cascade. While doing so, Ns fixes calcium and phosphorus to form a self-protecting apatite coating.

In their recent Poster Presentation at the annual meeting of The American College of Cardiology, Rasmussen et al reported the immunological identification of nanobacteria in calcified human vascular tissue. This corroborates the previous findings of the Hungarian researcher, Laszlo Puskas, PhD (personal communication, 2001).

Ns replicates at the rate of 44% per year. Could this explain the 33% to 38% annual increases in coronary artery calcium scores as seen from EBCT and helical CT scanning by Budhoff and Yoon, respectively?

I am currently completing an Western Independent Review Board investigation to address this question. Patients with known coronary artery disease and positive coronary artery calcium scans are being treated with NanobacTX, a proprietary compounded Nanobiotic (NanobacLabs, Tampa, Fla).

If therapy “unroofs” the carbonate coating of Ns, kills the organism while decreasing CAC scores and plaque volume, one might conclude that plaque regression has occurred. Such a finding will warrant expanded investigation into the role of Nanobacterium sanguineum in atherogenesis.

Immunological and serologic tests have confirmed the presence of Ns in the study patients. Final study results are expected in the fourth quarter 2002.

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