SOUTHWESTERN NEWS

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VITAMIN E PREVENTS EARLY PLAQUE FORMATION

DALLAS – November 24, 1998 – In laboratory tests vitamin E prevented the early stages of plaque formation by preventing white blood cells from sticking to cells that line the artery wall – another weapon in the antioxidant's attack on heart disease, according to research at UT Southwestern Medical Center at Dallas.

The study, the first to examine how vitamin E-enrichment of these white plaque-producing cells – called monocytes – affects adhesion to the cells that line arteries, was published in the November 24 edition of *Circulation: Journal of the American Heart Association.*

"This beneficial effect of vitamin E further strengthens its role as an adjunctive therapy in the management of atherosclerosis," said lead author Dr. Ishwarlal Jialal, a professor of pathology and internal medicine at UT Southwestern.

Scientists, including the UT Southwestern researchers, had already established that vitamin E can reduce susceptibility to atherosclerosis, or hardening of the arteries, because it inhibits the oxidation of low-density lipoprotein (LDL), or "bad" cholesterol. Two years ago, work by Jialal and Dr. Sridevi Devaraj, an instructor of pathology at UT Southwestern, showed the first intracellular effect of vitamin E – that it suppressed the function of monocytes.

The monocyte is the critical cell in early plaque development. An early stage of artery-clogging plaque involves the attachment of the monocyte to human endothelial cells – the artery wall. Preventing this step could be another important target in the treatment of atherosclerosis, said Jialal, who is also a senior investigator in the Center for Human Nutrition at UT Southwestern.

The laboratory study examined the effect that vitamin E had on the monocyte's ability to bind itself to endothelial cells.

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"These and other studies support the concept that the possible beneficial effects of vitamin E supplementation in reducing coronary-artery disease can be attributed to its combined effects on inhibition of the oxidative modification of lipoproteins and its intracellular effects on cells critical in atherogenesis, such as monocytes," Jialal said.

While enrichment of monocytes with vitamin E decreased adhesion to endothelium, enrichment of both monocytes and endothelial cells resulted in greater inhibition of adhesion, the work showed.

"This is most likely what is occurring when one ingests vitamin E since it gets in all cell membranes," Jialal said.

This also represents the first demonstration that vitamin E has effects at the nuclear level on an important transcription factor. It inhibited the transcription factor NF-kappaB that is important for adhesion and inflammation. Jialal said they have elucidated the molecular events that cause vitamin E to decrease clogged arteries: It inhibits this transcription factor and decreases adhesion molecules, resulting in less adhesion of monocytes to the endothelium. These actions reduce plaque formation.

Humans can obtain this response by taking vitamin E supplements.

Kazi Islam, a fellow in the Center of Human Nutrition, also participated in the study.

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