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Invade and conquer: Nicotine's role in promoting heart and blood vessel disease

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Cigarette smoke has long been considered the main risk factor for heart disease. But new research from Brown University in Providence, R.I., shows that nicotine itself, a component of cigarette smoke, can contribute to the disease process by changing cell structure in a way that promotes migration and invasion of the smooth muscle cells that line blood vessels. In particular, invading cells can remodel structures called podosomes, and this leads to further degradation of vessel integrity.

Ultimately, this cellular migration and invasion process gives rise to the formation of vessel-clogging fatty deposits known as plaque – the hallmark of heart and blood vessel disease. The results on the nicotine-podosome link will be presented at the 56th Annual Meeting of the Biophysical Society (BPS), held Feb. 25-29 in San Diego, Calif.

If confirmed in further studies, the finding that nicotine itself promotes vessel damage by changing podosomes appears to question the health benefits of helping people quit smoking through smokeless nicotine delivery agents such as gum or patches.

"The finding that nicotine is as effective as cigarette smoke in enhancing cellular structural changes, and breakdown of scaffold proteins by vascular smooth muscle cells, suggests that replacing cigarette smoking by nicotine treatment may have limited beneficial effects on atherosclerosis," notes lead researcher Chi-Ming Hai, professor of medical science in the department of molecular pharmacology, physiology, and biotechnology at Brown University.

Hai's research illuminates the multistep process of plaque formation, and suggests that a new powerful player, nicotine, may be involved. The plaque formation process begins as a response to cellular injury, and progresses to destructive and chronic inflammation of the vessel walls that attracts mobs of white blood cells, further inflaming the vessels. This damage-causing inflammation can be triggered by chemical insults from high blood sugar, modified low-density lipoproteins (LDL, the "bad cholesterol"), physical stress from high blood pressure, or chemical insult from tobacco smoke. Now nicotine itself appears to remodel key structures in a way that primes and enhances the invasion of smooth muscle lining the vessel wall.

Identifying a possible nicotine-posodome link in the invasion step of plaque formation process suggests a new means of intervening in the process: targeting the cell structures that are changed by nicotine and that promote invasion of the smooth muscle lining the vessel wall. If a therapy could prevent, slow, or reverse that step, it would likely interrupt the plaque-production cycle.

Fatty deposits accumulate in blood vessels beginning as young as age 10 and progress over a person's lifetime. Heart disease results if the deposits continue to build and harden into vessel-clogging plaque. When plaque ruptures, it can block blood flow, starving the heart or brain of oxygen and leading to a heart attack or stroke.

More information: The presentation, "Cigarette smoke and nicotine-induced remodeling of actin cytoskeleton and extracellular matrix by vascular smooth muscle cells," is at 1:45 p.m. on Sunday, Feb. 26, 2012, in the San Diego Convention Center, Hall FGH. ABSTRACT: http://tinyurl.com/73e836j

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