New Understanding Of Atherosclerosis Could Change The Way We Treat Heart Disease

Atherosclerosis -- the build-up of plaque in arteries, raising the risk for heart disease -- may not form the way we previously thought, according to a new study.

The discovery could change the way we treat cardiovascular disease, the leading cause of death in the U.S. and around the world, researchers noted.

Research from the University of Toronto and Massachusetts General Hospital, found that macrophages -- white blood cells in tissue associated with inflammation -- replicate within the plaques to cause atherosclerosis. Scientists originally thought the inflammatory macrophages collected mainly with the support of monocytes -- white blood cells that respond to pathogens -- from the bloodstream.

"Our study shows that the accumulation of macrophages also depends on their proliferation locally within the developing plaque," study researcher Clint Robbins, assistant professor in the laboratory medicine and pathobiology departments at the University of Toronto, said in a statement.

The new understanding of how macrophages reproduce within the plaque calls for the re-evaluation of current atherosclerosis therapy. Research has been concentrated on therapies that would block white cells from getting into the plaques, but not the growth of cells *within* the plaque.

Statin therapy is now used to lower blood lipids that contribute to plaque. With more research, scientists may use statins -- which also have anti-inflammatory properties -- to limit the spread of macrophages within the plaques.

Results of the study, published in the journal *Nature Medicine*, are based on mice subjects. More research is needed to know if findings are relevant to humans, though researchers did find evidence of macrophage growth in plaque on human carotid arteries. Scientists are interested in further finding if all macrophages or only subsets replicate within the plaque.