Atherosclerosis is the main cause of coronary heart disease, the leading cause of death in western societies, and costs the United States more than $200 billion in medical expenses and lost wages each year.

Also known as hardening of the arteries, atherosclerosis is an inflammatory condition in which plaque builds up inside arteries and restricts blood flow, which can lead to reduced flow of blood through coronary arteries, heart attack and stroke.

A University of Toledo researcher has received a $378,750 grant from the National Heart, Lung and Blood Institute to study a new way to treat this devastating condition.

“You hear the commercials all the time for drugs that help control cholesterol and blood pressure,” said Dr. Guillermo Vazquez, associate professor in the Department of Physiology and Pharmacology and associate director of UT’s Center for Hypertension and Personalized Medicine. “These medications help manage two of the major risk factors for atherosclerosis progression and can reduce the risk of heart attack and stroke, but it is our goal to find new, complementary strategies that could also help reduce the plaque burden in coronary heart disease.”

He said the body has natural ways of clearing arteries of this buildup, but cells called macrophages that take part in this process can become overwhelmed as the plaque grows thicker.

“Specialized cells called macrophages should carry lipids out of the plaque, but they can get stuck, which then contributes to the buildup and further reduces blood supply to the heart,” Vazquez said.

Vazquez and his team have discovered that a protein named TRPC3, which is present in these macrophages, could be controlled in order to help reduce the size of the plaque inside the arteries. They say that removing or turning off the TRPC3 protein may allow the macrophages to leave the arteries, reducing plaque buildup.

“We have developed mouse models of atherosclerosis in which we can test our hypothesis that interrupting TRPC3 functions may lead to increased mobility of the macrophage cells,” Vazquez said. “This concept shows promise for the development of complementary pharmaceuticals that could eventually be used in conjunction with traditional cholesterol-lowering drugs to accelerate the reduction in plaque burden.”

Vazquez also was the recipient of the Department of Physiology and Pharmacology’s Service Award in recognition for his excellent support and promotion of the department and the Center for Hypertension and Personalized Medicine. He was recognized at the department’s annual retreat last month.