

New twist to how 'deadly' plaques grow in heart diseases

Washington, August 12 ([ANI](#)): Researchers have determined that the growth of deadly plaque inside the walls of arteries may not be happening as they had previously believed.

The research from the University of Toronto and Massachusetts General Hospital also suggested a new potential target in the treatment of atherosclerosis, a leading cause of cardiovascular disease and death globally.

The research team found that macrophages, white blood cells that drive atherosclerosis, replicate inside plaques. Moreover, this growth is not reliant on cells outside the plaques called monocytes, as scientists had assumed.

Clint Robbins, lead author on the study and an Assistant Professor in U of T's Departments of Laboratory Medicine and Pathobiology, and Immunology, said that until now, the thinking was that inflammatory macrophages arise mainly from the recruitment of their precursors - monocytes - from the bloodstream.

He said that their study had showed that the accumulation of macrophages also depends on their proliferation locally within the developing plaque.

Filip Swirski, the study's principal investigator who is a scientist in the Center for Systems Biology at Massachusetts General Hospital and an Assistant Professor at Harvard Medical School, said that people have been thinking of targeting monocyte influx to treat atherosclerosis, but they need to consider macrophage proliferation as an additional or alternative approach, especially in established disease.

Swirski said that approach might be better than targeting circulating monocytes, since interrupting disease-causing processes within plaques could spare other beneficial immune responses that monocytes control.

Robbins said that additional targeting of macrophage proliferation may further decrease inflammation in atherosclerosis and prove clinically advantageous.

The researchers conducted their study in mice.

The study has been published in journal Nature Medicine. ([ANI](#))