ABSTRACT FORM DEADLINE: DECEMBER 1, 2018

Association of University Cardiologists

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Fortilin is a New Molecular Target of Atherosclerosis.

Atherosclerosis—the thickening of the wall of arteries that limits transport of oxygen-containing blood to vital organs—remains one of the most serious health problems in the U.S. today and costs nearly $286 billion every year. After reviewing 20 randomized clinical trials of PCSK9 inhibitors (the most advanced treatment for atherosclerosis) involving 67,237 participants, the Cochrane Organization concluded that they were only modestly effective at decreasing cardiovascular disease (CVD) events (absolute risk reduction of 1%) despite a drastic 54% reduction of low density lipoprotein (LDL), suggesting that lowering LDL does not necessarily eliminate CVD and that we need to identify alternative molecular targets to cure atherosclerosis.

Fortilin is a 172-amino acid multi-functional protein abundantly expressed in atherosclerotic tissue. We previously showed that constitutional knockdown of fortilin protected hypercholesterolemic $Ldlr^{-/-}Apobec1^{-/-}$ (HC) mice against atherosclerosis. In these mice, both intracellular and extracellular fortilins were downregulated.

More recently, we found that the lack of fortilin in macrophages (MΦ) is sufficient to protect HC mice against atherosclerosis.

However, we still did not know how the lack of fortilin in MΦ ameliorated atherosclerosis.

I will present both published and unpublished set of data to explore the mechanism by which fortilin facilitates atherosclerosis. In addition, I will present our efforts to identify small molecular weight inhibitors of fortilin to ameliorate atherosclerosis.

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