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HEART & DIABETES INSTITUTE

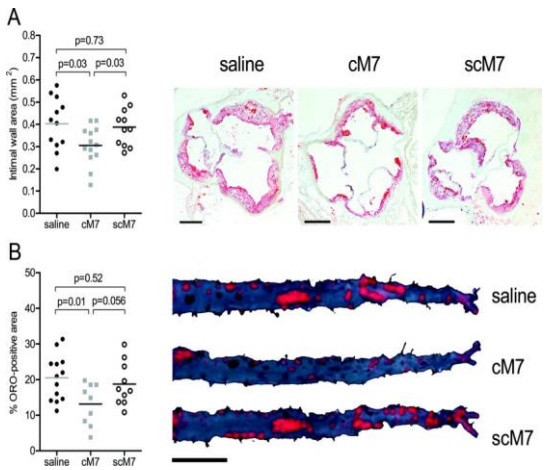
CD40L-Mac1 binding inhibitor for Inflammation

Specific inhibition of the CD40L/Mac-1 binding represents a novel anti-inflammatory treatment strategy for atherosclerosis.

SUMMARY OF INVESTMENT OPPORTUNITY:

Atherosclerosis is a chronic, inflammatory disease driven by the continuous emigration of monocytes into the vessel wall. CD40L is an established marker and mediator of chronic inflammatory diseases such as atherosclerosis. While anti-CD40L treatment generated promising results in clinical trials, elevated thrombembolic complications prohibited the pursuit of this strategy. We previously reported that CD40L mediates atherogenesis independently of CD40 in mice, proposing a novel interaction with the leukocyte integrin Mac-1. Targeting of CD40L/Mac-1 binding with an inhibitory peptide, cM7, proofed to be specific and ultimately effective in attenuating inflammation and atherosclerotic lesion formation in mice. Specific inhibition of the CD40L/Mac-1 dyad therefore represents an attractive novel anti-inflammatory treatment strategy for atherosclerosis and other chronic inflammatory diseases, promising minimal side effects.

While inflammation drives many chronic diseases, including atherosclerosis, few selective anti-inflammatory treatment options currently exist.
In the context of coronary syndrome, physicians are increasingly calling for drugs that stabilize coronary plaques and that control inflammatory processes that contribute to plaque instability and rupture. A therapy that specifically (and intentionally) treats these factors would be novel and would represent a new treatment paradigm in the field of CV disease prevention and management.
Drugs that have been designed to expressly control coronary plaque inflammation are still in their infancy, with very few in late-stage trials and none marketed.



Specific blockade of the CD40L-Mac-1 interaction with an inhibitory peptide (cM7) attenuates atherosclerosis and modulates leukocyte recruitment in mice.

INTELLECTUAL PROPERTY: WO2012052205 – National Phase AU, CA, EU, JP, US – Priority date 21/10/2010

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