



Beth Israel Deaconess
Medical Center



Harvard
Medical School
Teaching Affiliate

BIDMC 301: Gene Therapy Treatment for Atherosclerosis and Prevention of Vascular Graft Failure

➤ **New use of A20 as an anti-proliferative, pro-apoptotic agent**

Atherosclerosis-related diseases are a leading cause of death and impairment in the United States, affecting >60 million people. Statistics by the American Heart Association indicate these diseases result in 39% of total deaths and cost >\$352 billion in medical costs and disabilities annually.

Transplant-associated vasculopathy (TAV), closely related to atherosclerosis, is a major barrier to successful organ transplantation. Although the initiating factors of atherosclerosis and TAV are different, their pathological features are similar and relate to the dysregulation of 2 major cell types of the vessel wall: smooth muscle cells (SMC) and endothelial cells (EC).

The inventors demonstrate that the selective transfection of SMC in the vessel wall to induce high levels of A20 expression results in both prevention of and regression of the neointimal hyperplasia associated with atherosclerosis.

Stage of Development:

Proof of concept demonstrated in human aorta SMC in culture and in a balloon angioplasty rat model.

Inventors:

Christiane ferran and Marie Arvelo

*Contact: Catherine Lenich, PhD
Senior Licensing Associate, TVO
Tel: 617.667.0568
Fax: 617.667.0646
clenich@bidmc.harvard.edu*

Commercial Opportunity:

- ✓ Potentially all vascular conditions such as restenosis, ischemia-reperfusion injury and angioplasty-related injury.
- ✓ Potentially all autogenous bypass operations including:
 - Coronary artery bypass grafts
 - Peripheral vascular bypass grafts
 - Hemodialysis fistulae
 - Organ transplantation

Patent Status:

US Patent No. 7,297,685

Publications (available upon request):

FASEB J. (2006) 20: 1418.
Blood (2004) 104: 2376.
Circulation (2003) 108: 1113.

Competitive Advantage:

Targeting A20 allows inhibition of both EC activation and SMC proliferation, the two major culprits of atherosclerotic lesion progression.