Epicardial adipose tissue (EAT) and pericardial adipose tissue (PAT) as cell models to assess patient responsiveness to therapeutics

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Background. The abundance and inflammatory stigmata of epicardial(EAT) and pericardial adipose tissue(PAT) is associated with higher carotid intima-media thickness, suggesting a role of EAT and PAT in the development of atherosclerosis. Curbing EAT and PAT dysmetabolism and inflammation may represent a novel therapeutic target to prevent coronary atherosclerosis.

Purpose. Development of a feasible and efficient method for isolation of adipocytes from human PAT and evaluation of adipocyte response to therapeutics and micronutrients

Methods. PAT was collected from coronary patients undergoing surgery for coronary stenosis and from patients undergoing aortic or mitral valve surgery and immediately enzymatically processed. Isolated adipocytes were morphologically and molecularly characterized. Cell responsivity was evaluated by exposure to inflammatory stimuli or to docosahexaenoic acid(DHA), a well-known cardio-protective fatty acid.

Results We obtained pure cultures of adipocytes that can be sub-cultured for several days without losing viability and retaining the ability to respond to stimuli. Basal expression of inflammatory genes in adipose cells was higher in coronary patients than in aortic and mitral surgery patients. Exposure of adipocytes to TNFα significantly induced the expression of MCP-1 and IL-6, ($p < 0.05$), while downregulated the expression of UCP-2 and PPARγ ($p < 0.05$). On the other hand, the exposure of adipocytes to DHA resulted in a downregulation of MCP-1 and IL-6 expression ($p < 0.05$) and in the upregulation of UCP-1,-2 and PPARγ ($p < 0.05$).

Conclusion(s). Our data propose a new efficient method for isolating adipocytes from PAT and for using them as a bio-reactor to test differences in different cardiovascular conditions.