GLP-1 receptor expression in epicardial adipose tissue is associated with genes involved in fatty acid oxidation and white-to-brown fat differentiation

Elena Dozio[1], Elena Vianello[2], Alexis E. Malavazos[3], Lorenza Tacchini[2], Gerd Schmitz[4], Gianluca Iacobellis[5], Massimiliano M. Corsi Romanelli[6]
[1]Department of Biomedical Sciences for Health, Università degli Studi di Milano, Milan, Italy; Laboratory of Molecular Pathology, IRCCS Policlinico San Donato, San Donato Milanese, Milan, Italy
[2]Department of Biomedical Sciences for Health, Università degli Studi di Milano, Milan, Italy
[3]High Speciality Center for Dietetics, Nutritional Education and Cardiometabolic Prevention, IRCCS Policlinico San Donato, San Donato Milanese, Milan, Italy
[4]Institute for Clinical Chemistry and Laboratory Medicine, University of Regensburg, Regensburg, Germany
[5]Division of Endocrinology, Diabetes and Metabolism, Department of Medicine, University of Miami, Miller School of Medicine, Miami, FL, USA
[6]Department of Biomedical Sciences for Health, Università degli Studi di Milano, Milan, Italy; Service of Laboratory Medicine1-Clinical Pathology, IRCCS Policlinico San Donato, San Donato Milanese, Milan, Italy

INTRODUCTION: Epicardial adipose tissue (EAT) is a risk factor for cardiovascular diseases. Glucagon-like peptide 1 analogs (GLP-1A) were reported to induce beneficial cardiovascular effects and reduce EAT, possibly throughout targeting GLP-1 receptor (GLP-1R). Nevertheless, the role of EAT GLP-1R, GLP-2R and their interplay with EAT genes involved in adipogenesis and fatty acid (FA) metabolism are unknown. We aimed to analyze whether EAT transcriptome is related to GLP-1R and GLP-2R gene expression, and GLP-1 and GLP-2 plasma levels in coronary artery disease patients (CAD).
METHODS: EAT was collected from 17 CAD patients undergoing coronary artery bypass grafting for microarray analysis of GLP-1R, GLP-2R and genes involved in FA metabolism and adipogenesis. EAT thickness was measured by echocardiography. GLP-1 and GLP-2 levels were quantified by enzyme-linked immunosorbent assay in CAD and healthy subjects (CTR).
RESULTS: EAT GLP-1R was directly correlated with genes promoting beta-oxidation and white-to-brown adipocyte differentiation, and inversely with pro-adipogenic genes. GLP-2R was positively correlated with genes involved in adipogenesis and lipid synthesis, and inversely with genes promoting beta-oxidation. GLP-1 and GLP-2 levels were higher in CAD than CTR and in patients with greater EAT thickness.
CONCLUSION: GLP-1 analogs may target EAT GLP-1R and therefore reduce local adipogenesis, improve fat utilization and induce brown fat differentiation. As EAT lies in direct contiguity to myocardium and coronary arteries, the beneficial effects of GLP-1 activation may extent to the heart. The increased levels of circulating GLP-1 and GLP-2 and EAT GLP-2R may be compensatory mechanisms related to CAD and also EAT expansion, but the meaning of these observations needs to be further investigated.
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