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PLEIOTROPIC EFFECTS OF INCRETIN-BASED THERAPIES IN CARDIOPROTECTION

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Incidence of cardiovascular disease (CVD) events can be reduced by therapeutic interventions targeting multiple cardiovascular and metabolic derangements. Evidences suggest that modulation of incretin signalling by glucagon-like peptide-1 (GLP-1) agonists or dipeptidyl-peptidase 4 (DPP4) inhibitions are cardioprotective. In this regard, dipeptidylpeptidase-4 inhibitors (DPP4-i) specifically developed as incretin-based oral anti-hyperglycemia therapies, including sitagliptin, saxagliptin, linagliptin, alogliptin and vildagliptin are now emerging as new novel agents with high potential in reducing the progression of CVD. DPP4 inhibitors exhibit multiple pleiotropic protective effects in the vasculature and the heart, in case of hypertension, atherosclerosis, stroke and myocardial infarction. Earlier preclinical and clinical investigations displayed the activation of several cardioprotective signalling pathways by GLP-1 upregulation, leading to decrease in cardiomyocyte apoptosis and infarct size after ischemia-reperfusion (I/R) injury through activation of phosphoinositol-3 kinase (PI3K) and RISK (AKT and ERK1/2) pathways, improvement in coronary blood flow through increased coronary artery vasorelaxation in a cAMP and endothelial nitric oxide synthase (e-NOS)-dependent manner and also preventing heart failure by enhancing oxygen uptake, augmenting left ventricular ejection fraction (LVEF) and increase in glucose uptake through GLP-1-dependent AKT activation and glucose transporter type 4 (GLUT4) translocation. Although GLP-1 receptor agonists including exenatide and liraglutide have been extensively used, DPP4 inhibitors still have the potential to exert either a broader range of beneficial effects or a different subset of benefits on overall CVD outcomes. In addition to its anti-hyperglycemic effect, the cardioprotective mechanisms of action of these drugs are poorly understood and finding the best drug is still a challenge.

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