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Results: Five microRNAs were differentially expressed between NC- and HyC- HDL (P-value <0·05). Specifically, HyC-HDLs had higher levels of miR-126-5p, miR-126-3p and miR-30b-5p (2·7×, 1·7×, 1·3× respectively) while the levels of miR-103a-3p and let- 7 g-5p were found to be reduced (−1·6×, −1·4×, respectively) vs NC-HDL. Only miR126 (both -3p and -5p) was found to be enhanced in endothelial cells upon HDL treatment. Interestingly, miR-126-3p and -5p levels were found to be 3-fold higher in those endothelial cells incubated with HyC-HDL as compared to NC-HDL (P < 0·05), an effect that persisted despite HDL removal and was independent of SRB1 expression. Eighteen top miRNA126-target genes were evaluated being PI3KR2 a potential target gene (P-value < 0·05).

Conclusions: Our results collectively suggest that hypercholesterolemia induces changes in HDL-miRNA signature and enhances HDL-miR126 delivery to endothelial cells likely modulating key processes related with vascular survival and proliferation.

P092-T | Dynamics of nitric oxide production in the rat heart during hypokinesia: effects of inhibitors of NO-synthase L-NAME and aminoguanidine

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The basis of regulation of the cardiac work are sympathoparasympathetic interaction. Currently, a considerable value to realization of regulatory effects contribute by the nitric oxide (NO). There are two main ways of NO production in the body: enzymatic and non-enzymatic. Prolonged hypokinesia causes significant changes in the contractile function of the cardiac muscle. All these phenomena are inevitably lead to a serious deterioration of tissue oxygen supply, i.e. hypoxia. Previously, we carried out the EPR spectroscopic studies of the dynamics of NO production in cardiac and hepatic tissues during hypokinesia of various duration, in which we found a significant increase in NO content on the 30 days of hypokinesia. Therefore, the aim of the study was to investigate the role of NO in the consequences resulting from the hypokinesia by analyzing the NO containing paramagnetic complexes in various tissues of rats which was growing under restricted physical activity.

By the method of EPR spectroscopy it was found an increase in the intensity of production of NO in the rats hearts after 90-days hypokinesia. The nonselective blockade of NO-synthase activity by L-NAME in hypokinesed rats resulted in a decrease of content of NO by 67–70% in atrias and ventricles of the heart. Selective blockade of inducible NO-synthase by aminoguanidine caused a decrease of the content of NO by 60–65% in the tissues of the atrias and ventricles. The obtained results suggested that increasing of NO production under conditions of hypokinesia occurred through the activation of NO-synthase activity.

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P093-T | A multi-biomarker panel of myocardial remodelling provides incremental prognostic value in heart failure patients

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Background: Cardiomyocyte injury (CMI), myocardial interstitial fibrosis (MIF) and coronary microvascular endothelial dysfunction and inflammation (EDI) are structural alterations of myocardial remodelling in heart failure (HF). We evaluated the prognostic value of a combination of biomarkers of these alterations in HF patients.

Material and methods: Circulating high-sensitivity troponin-T (hs-TnT), carboxy-terminal propeptide of procollagen type-I (PICP) and carboxy-terminal telopeptide of collagen type-I to matrix metalloproteinase-1 ratio (CITP: MMP-1), and vascular cell adhesion molecule-1 (VCAM-1) as biomarkers of CMI, MIF and EDI, respectively, were measured in HF patients from the Generation Scotland (n = 71) and Leizaran (n = 197) cohorts. The association of their combination with a composite outcome of hospitalization for HF (HHF) or cardiovascular death (CVD) was