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**HDL preserves mitochondrial complex I activity during myocardial reperfusion injury**

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On behalf: Atherolab

Topic(s):
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HDL reduces the final infarct size in different myocardial ischemic-reperfusion injury models. This effect has been attributed to the activation of STAT3, a transcriptional factor with protective effect over mitochondrial complex I of the electron transport chain. We hereby tested whether HDL treatment during reperfusion preserves complex I integrity. Methods. Male wistar rats with 12-14 weeks were exposed to 35min of ischemia, followed by 90min of reperfusion in a Langendorff ex vivo model. HDL (200ug/mL) or PBS were administered during the first seven minutes of reperfusion. The heart was then excised and the area at risk was permeabilized with saponin and carried to the Oroboros oxygraph. Oxygen consumption rate was modulated by addition of ADP, rotenone, succinate and FCCP. Mitochondrial viability was tested by citrate synthase assay. Statistical analysis: student t-test was used to compare the means. The software used was the GraphPad Prism 6.0. Results. A total of 20 and 6 heart samples were analyzed after treatment with HDL and PBS, respectively. ADP-stimulated oxygen consumption was significantly higher in the group treated with HDL compared to Ctrl. (33.8±6.6 vs. 7±2.2 pmol/s*mg for HDL and Ctrl. respectively). The mitochondrial viability of each group was comparable (1.1±0.75 vs. 1.3±0.8 for Ctrl. and HDL, respectively). Conclusion. Myocardial post-conditioning with HDL preserves mitochondrial complex I activity.
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Conclusion. Myocardial post-conditioning with HDL preserves mitochondrial complex I activity.