MMP-2 activity by −8 ± 1% (n = 2), Est-stimulated migration by −34 ± 2% ($p < 0.01$; n = 4-8), and Est-stimulated proliferation by −9 ± 3% ($p < 0.05$; n = 3), indicating they occur in an ERK1/2 dependent manner.

Conclusions: Estrogen stimulates the cellular processes of IH development via ERK1/2 dependent signaling. Inhibition of ERK1/2 results in downregulation of Est-mediated MMP activity and Est-induced VS MC migration and proliferation. Future studies will include targeted ERK1/2 silencing to assay a possible mechanism for effective IH inhibition. Understanding the signaling mechanisms involved in hormone-mediated IH development could provide a basis for therapeutic strategies of inhibition.

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PS216.

Short-term Diet Induced Obesity Drives Negative Vein Graft Remodeling

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Objectives: Inflammatory pathways are associated with vein graft failure. Short-term high-fat feeding induces a proinflammatory state in perivascular adipocytes, but the longer-term impact of such dietary induced dysfunction on clinically relevant vascular events is unclear. We tested the hypothesis that the inflammatory phenotype resulting from diet induced obesity (DIO) drives accelerated vein graft failure (increased intimal hyperplasia, enhanced negative wall remodeling).

Methods: Male 9-week-old DIO mice (n = 5; 3 wks of high caloric diet) and controls (n = 5) underwent isograft (IVC from same diet donor) unilateral carotid interposition vein graft with a focal mid-graft stenosis. Perfusion fixed vein graft was harvested 4 wks later. DIO/control mice also underwent blood, spleen, and adipose cell harvest for immune profiling (flow cytometry).

Results: Despite a 40% larger body size, DIO mice had 34% smaller residual vein graft lumens ($p = 0.02$). Lumen loss was not due to accelerated intimal hyperplasia, or other differentials in wall thickness (all layer thicknesses and intima/media ratio were equivalent), but rather acceleration of overall negative wall remodeling (cross sectional wall area 47% smaller, $p = 0.03$; outer vein graft perimeter 19% shorter, $p = 0.01$). Resting blood and spleen immune cell profiles were similar; DIO fat held significantly more NK cells, macrophages, and dendritic cells.

Conclusions: These findings highlight negative wall remodeling as a factor leading to vein graft failure, and provide direct evidence that short-term dietary alterations in the mammalian metabolic milieu can have lasting implications relating to acute vascular interventions.

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PS218.

Effect of Remote Ischemic Preconditioning in Liver Ischemia—Reperfusion Injury Produced by Supraceliac Aortic Cross-clamping in a Swine Model of Open Repair of Thoracoabdominal Aortic Aneurysm

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Objectives: Visceral ischemia is inevitable during the open repair of a thoracoabdominal aortic aneurysm (TAAA). Remote ischemic preconditioning (RIPC) has been described as a potential protective measure from ischemia - reperfusion injury (IRI) in various distal tissues. The aim of this experimental study has been to identify any protective effect of RIPC in liver IRI caused during aortic temporal occlusion as performed during a TAAA open repair.

Methods: Three groups of 6 swines each underwent a 30 minutes of aortic supraceliac cross-clamping after a left sided visceral rotation. Liver functional and pathological status was assessed 24 hours after ischemia. The first group was the sham group, the second group was the ischemia - reperfusion (IR) group and the third one was the RIPC group, where remote ischemic preconditioning with a temporary occlusion of the infrarenal aorta was performed before supraceliac aortic cross-clamping. Statistical analysis was done with parametric and non-parametric techniques.

Results: Attenuation of liver damage was noted in the RIPC group as compared to the IR group. Statistically significant reduced values of liver functional markers were found at 24 hours post ischemia between the two groups as follows: alanine aminotransferase (ALT), 36.6 ± 13.5 IU/L in RIPC group vs 58.6 ± 12.5 IU/L in IR group ($p = 0.01$), and aspartate transaminase (AST), 43.4 ± 14.7 IU/L in RIPC group vs 119.5 ± 25.7 IU/L in IR group ($p < 0.001$). With regard to the pathological liver status, the median values of the parameters examined in RIPC and IR groups were respectively: congestion 1 vs 2 ($p = 0.05$), inflammation 1 vs 2 ($p = 0.02$), necrosis 0 vs 2 ($p = 0.02$). No difference was found in liver degeneration (1 vs 1, p = NS).

Conclusions: There is considerable evidence that RIPC with a temporary infrarenal aortic occlusion can reduce the occasionally hazardous liver IRI that is caused during a TAA open repair.