

through retro-orbital injection at two different dosages, 0.75gI/kg and 2.75gI/kg. PBS was given to the mice in control group. Mice kidneys were harvested at day 3 and day 7 following Iodixanol administration. RNA and protein were then extracted. qRT-PCR and Western blot were used to quantify A1AR, A2AAR, A2BAR, and A3AR RNA and protein expression respectively, with GAPDH as endogenous control.

Results: qRT-PCR showed that Iodixanol induced AR transcription, specifically in the group treated with 2.75gI/kg at day 3 after injection. The RNA levels in all the four subtypes of ARs were increased 2-3 folds at day 3, but returned to normal at day 7 in Iodixanol groups compared to PBS controls. The Western blot results showed that A1AR, A2AAR, A3AR expressions were increased 1.5-2 folds in Iodixanol group at day 3 compare to PBS control. A2BAR expression was very low in normal physiological condition and no significant changes were detected by western blot.

Conclusions: Our results indicate that Iodixanol induces adenosine receptor gene expression in mice. Adenosine receptors may play a role in the development of CIN. Further investigation on the correlation between adenosine receptor gene expression and severity of CIN will be preformed.

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C6k: Poster Session - Research (2)

PS182.

Inhibition of Angiogenesis and Endothelial Cell Function by WISP-1/CCN4

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Objectives: Wnt-induced secreted protein-1 (WISP-1/CCN4) is involved in regulating cell proliferation, survival/apoptosis, migration and tumor growth. We previously observed that tumor stroma-produced WISP-1/CCN4 plays a critical role in melanoma growth and tumor angiogenesis. We sought to determine the biologic effect of WISP-1/CCN4 on endothelial cell biology and angiogenesis.

Methods: The effects of WISP-1/CCN4 on cell growth and migration of human endothelial cells (EC) were examined by MTT and transwell-migration assay. Two- and three-dimensional (2D and 3D) angiogenesis models were used to study the role of WISP-1/CCN4 in vascular network formation of EC. Melanoma xenograft murine model was employed to examine the biological effect of WISP-1/CCN4 on tumor angiogenesis and growth. Human melanoma cells transduced with WISP-1/lentivirus or GFP/lentivirus were xenografted on skin of SCID mice (n = 6/group). Expression of exogenous WISP-1/CCN4 in transduced melanoma cells was validated by Western blot. Tumor angiogenesis was assessed by mouse whole-body Dil-perfusion and tumor tissue confocal microscopy. Tumor size was measured to determine tumor growth.

Results: WISP-1/CCN4 inhibited cell growth and migration of EC in vitro. Vascular network formation of EC in 2D- and 3D angiogenesis models was suppressed considerably ($P < .01$) by supplementation of γ WISP-1 (100 ng/mL) in both Matrigel (2D) and type I collagen gel (3D). Elevated expression of WISP-1 in tumor tissue significantly antagonized tumor angiogenesis and retarded tumor growth in vivo.

Conclusions: Our results revealed a novel role of WISP-1/CCN4 in negatively regulating endothelial cell biology and angiogenesis, implicating that WISP-1/CCN4 may serve as a novel target for cancer therapeutic intervention.

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PS184.

Differential Proliferative and Migratory Activity of Human Myoblasts Isolated from CLI and Control Patients

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Objectives: Patients with critical limb ischemia (CLI) have attributable myopathy that impairs their functional ability as well as dampening the positive effect of successful revascularization outcomes. Adult skeletal muscle retains the potential to repair following different injury patterns. We aim to investigate the potential differences in proliferation and migration capability of human myoblasts with CLI and asymptomatic control patients.

Methods: Gastrocnemius muscle biopsies were obtained from patients with critical limb ischemia and control samples were obtained from non-ischemic patients. Human myoblasts were isolated and cultured from each sample and stained with the myoblast marker desmin. Confirmed myoblast cultures (<5% alternative cell type contamination) were then used to investigate the proliferative capability (MTT assay) as well as migratory potential (scratch-wound assay) under both ischemic and normoxic conditions of each group.

Results: Myoblasts isolated from CLI patients demonstrated greater proliferative ability in comparison to control samples. However, control samples revealed greater capacity to heal scratch wounds made in a monolayer of myoblasts in both normoxia and hypoxic conditions.

Conclusions: Isolation of human myoblasts, especially from disease groups provides a useful platform to perform in vitro analysis to better understand and characterise pathology. CLI myoblasts exhibited enhanced proliferative but diminished migratory potential in comparison to control myoblasts.

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PS186.**Efficacy of Antibiotic-Impregnated Bone Cement Beads Against Organisms Found in Abdominal Vascular Graft Infections**

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Objectives: Graft infection often involves several different organisms. Antibiotic-impregnated polymethylmethacrylate (PMMA) beads may be effective in controlling reinfection after infected graft replacement. We sought to determine an effective antibiotic combination in PMMA beads for use in vascular graft infection.

Methods: PMMA beads were impregnated with different combinations of antibiotics including daptomycin (DA), tobramycin (TO), and meropenem (ME). Beads were plated into separate 0.8% Mueller-Hinton agar plates with vancomycin-resistant enterococcus (VRE), *Klebsiella*, *S. epidermidis*, and methicillin-resistant *Staphylococcus aureus* (MRSA). Antibiotic inhibition was recorded in millimeters using the disc agar-diffusion method and averaged from three separate platings.

Results: DA alone was not active against *Klebsiella* (average = 0 mm). TO alone was not active against VRE, *Klebsiella*, or MRSA. ME showed broad-spectrum activity against all organisms with >15 mm inhibition halo. The efficacy of ME was decreased by TO but not DA. Adding DA and TO to ME did not improve efficacy.

Conclusions: Meropenem in PMMA beads shows activity against difficult pathogens encountered in vascular graft infections. Daptomycin can be added for double-coverage against multi-drug resistant gram-positive pathogens such as VRE and MRSA. Tobramycin appears to reduce the efficacy of meropenem when used in combination.

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Table. Average inhibition halo in millimeters

	VRE	<i>Klebsiella</i>	<i>S. epidermidis</i>	MRSA
DA	9.8	0	10.2	9.6
TO	2.0	10.7	14.0	4.7
DA + TO	8.0	10.8	14.3	9.9
ME	17.0	15.3	21.0	17.3
ME + DA	17.2	16	21.7	17.7
ME + TO	13.8	13.5	16.7	13.4
ME + DA + TO	16.9	15.0	21.0	17.0
Control	3.2	6.9	8.7	4.2

PS188.**Is Oxidative Stress Important in AAA Pathogenesis?**

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Objectives: Active investigations continue to identify markers other than size that would predict a risk of AAA rupture. Circulating biomarkers could also indicate optimal intervals between the surveillance intervals. Finally, the identification of biomarkers also may identify potential pathogenic pathways, and thus may open possibilities for pharmacological inhibition of growth. In the search of novel biomarkers of AAA progression, serum and wall material proteins were analyzed by a differential proteomic approach.

Methods: Same layers of AAA wall from ruptured (rAAA) and non-ruptured AAA were incubated, and the proteins released were analyzed by 2-dimensional difference in-gel electrophoresis. Proteins from serum were analyzed and correlated with AAA annual expansion rate.

Results: Several differentially expressed proteins involved in main AAA pathological mechanisms (proteolysis, oxidative stress, and thrombosis) were identified by mass spectrometry. Among the proteins identified, peroxiredoxin-2 (PRX-2) was more permanent, which was further validated by Western blot and immunohistochemistry. We demonstrated increased PRX-2 serum levels in rAAA patient wall material compared with AAA subjects and also positive correlation in serum among PRX-2 and AAA diameter and annual expansion rate. Finally, a prospective study revealed a positive correlation between PRX-2 serum levels and AAA expansion rate.

Conclusions: Several proteins associated with AAA pathogenesis have been identified by a proteomic approach. Protein profiles identified in the serum would appear to be a convenient monitoring tool that has the ability to be both sensitive and specific for AAAs. PRX-2 serum levels are increased in rAAA patients and correlate with AAA size and growth rate, suggesting the potential use of PRX-2 as a biomarker for AAA evolution.

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PS190.**Urine mRNA as an Early and Novel Marker of Contrast-Induced Kidney Injury (CIKI)**

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Objectives: CIKI is a common etiology of renal dysfunction in hospitalized patients, and affects the daily practice of vascular surgeons. It increases mortality, costs and LOS; yet no direct or specific marker exists. Indirect