

Henry Ford Health System Publication List June 2007

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Ananthasubramaniam K and Jaffery Z. (2007). "Postoperative right atrial compression by extracardiac hematoma: transesophageal echocardiographic diagnosis in the critically ill patient." *Echocardiography* 24(6): 661-3. [PDF Full-Text](#)

Heart and Vascular Institute and the Department of Internal Medicine, Henry Ford Hospital, Detroit, Michigan, USA.

Armada T, Berman H, Hopkins J, Kreykes B, Wegmiller D and McPherson B. (2007). "What does it take to build a strong nonprofit health care board?" *Inquiry* 44(1): 8-14. **Full-Text Not Available** / [Click for Article Request Form](#)

Henry Ford Hospital and Health Network, Detroit, Mich, USA.

Bey MJ, Brock SK, Beierwaltes WN, Zauel R, Kolowich PA and Lock TR. (2007). "In vivo measurement of subacromial space width during shoulder elevation: Technique and preliminary results in patients following unilateral rotator cuff repair." *Clin Biomech (Bristol, Avon)*. EPub Ahead of Print. [PDF Full-Text](#)

Henry Ford Hospital, Department of Orthopaedic Surgery, Bone and Joint Center, 2799 W. Grand Blvd., E&R 2015 Detroit, MI 48202, United States.

BACKGROUND: The shoulder's subacromial space is of significant clinical interest due to its association with rotator cuff disease. Previous studies have estimated the subacromial space width to be 2-17mm, but no study has measured in vivo subacromial space width during shoulder motion. The purpose of this study was to measure the in vivo subacromial space width during shoulder elevation in patients following rotator cuff repair. **METHODS:** Biplane X-ray images were collected during shoulder elevation of 11 patients who had undergone rotator cuff repair. Glenohumeral joint motion was measured from the biplane X-ray images for each subject's repaired and asymptomatic, contralateral shoulders. The joint motion data were combined with subject-specific CT models to measure the subacromial space width during shoulder motion. **FINDINGS:** Subacromial space width decreased with shoulder elevation, ranging from 2.3 to 7.4mm in the repaired shoulder and 1.2-7.1mm in the contralateral shoulder. Subacromial space width in the repaired shoulder was only 0.5mm less than the contralateral shoulder when averaged over 10-60 degrees of

glenohumeral elevation. INTERPRETATION: The results indicate that the humerus in the repaired shoulder is positioned more cranially on the glenoid than in the contralateral shoulder. It is unclear if these subtle differences in subacromial space width are due to the surgical procedure or post-operative stiffness, or if subacromial impingement contributed to the development of the rotator cuff tear. Future research will ascertain if these results represent a transient response to the surgery or a more fundamental difference in rotator cuff function between repaired and contralateral shoulders.

Brar I, Shuter J, Thomas A, Daniels E and Absalon J. (2007). "A comparison of factors associated with prevalent diabetes mellitus among HIV-Infected antiretroviral-naive individuals versus individuals in the National Health and Nutritional Examination Survey cohort." J Acquir Immune Defic Syndr **45**(1): 66-71. [PDF Full-Text](#)

Department of Medicine, Division of Infectious Diseases, Henry Ford Hospital, Detroit, MI 48202, USA. ibrar1@hfhs.org.

BACKGROUND: In the general population, diabetes mellitus (DM) is associated with age, minority race/ethnicity, and obesity. Among HIV-infected persons, antiretroviral therapy (ART) use and hepatitis C virus (HCV) infection have been associated with DM. This study examined DM prevalence and its predictors in ART-naive HIV-infected patients. METHODS: A cross-sectional analysis of ART-naive HIV-infected adults enrolled in 3 Terry Bein Community Programs for Clinical Research on AIDS (CPCRA) clinical trials versus adults enrolled in the National Health and Nutritional Examination Survey (NHANES). RESULTS: The prevalence of DM in the CPCRA clinical trials versus the NHANES was 3.3% versus 4.8%. The mean body mass index (BMI) was lower in the CPCRA trials versus the NHANES (25 kg/m vs. 28 kg/m). HCV was associated with DM only in univariate analyses in the CPCRA trials. In univariate and multivariate analyses, race/ethnicity, age, and BMI were associated with DM in both cohorts. Among women, age and BMI were associated with DM in both cohorts; race/ethnicity was associated with DM only in the NHANES. HCV was predictive of DM in blacks in the CPCRA trials (P = 0.004 before adjustment for multiple comparisons) but not in the full cohort. CONCLUSIONS: Our findings did not suggest an increased prevalence of DM in ART-naive HIV-infected patients. Although there was a trend toward increased prevalence of DM in HIV-HCV-coinfected patients, dominant risk factors associated with DM among ART-naive HIV-infected adults mirrored those of the general population.

Burd EM, Juzych LA, Rudrik JT and Habib F. (2007). "Pustular dermatitis caused by *Dermatophilus congolensis*." J Clin Microbiol **45**(5): 1655-8. **Full-Text Not Available / [Click for Article Request Form](#) / [Print Copy Available in Sladen Library K-17](#)**

Department of Pathology, Henry Ford Hospital, 2799 W. Grand Blvd., Detroit, MI 48202, USA. eburd1@hfhs.org.

We describe a case of pustular dermatitis in a 15-year-old girl who had just returned from horseback riding camp. Based on gram staining, colony characteristics, biochemical reactions, and whole-cell fatty acid analysis, the causative agent was identified as *Dermatophilus congolensis*. The literature contains few reports of human infection with this organism.

Cankovic M, Mikkelsen T, Rosenblum ML and Zarbo RJ. (2007). "A simplified laboratory validated assay for MGMT promoter hypermethylation analysis of glioma specimens from formalin-fixed paraffin-embedded tissue." Lab Invest **87**(4): 392-7. [PDF Full-Text](#)

Department of Pathology, Henry Ford Hospital, Detroit, MI 48202, USA. mcankov1@hfhs.org.

Glioma, and in particular high-grade astrocytoma termed glioblastoma multiforme (GBM), is the most common primary tumor of the brain. Epigenetic silencing of the MGMT (O(6)-methylguanine-DNA Methyl transferase) DNA repair gene by promoter methylation compromises DNA repair and has been associated with longer survival in patients with GBM who receive alkylating agents. The methylation status of the MGMT promoter is determined by methylation-specific polymerase chain reaction analysis (MSP). This protocol is often challenging with GBM specimens, because of extensive necrosis and scarcity of malignant cells. The objective of this study was to develop a reliable, clinically validated assay for detection of epigenetic silencing of the MGMT gene using formalin-fixed, paraffin-embedded brain tumor resections and methylation-specific PCR.

Chen J, Cui X, Zacharek A, Jiang H, Roberts C, Zhang C, Lu M, Kapke A, Feldkamp CS and Chopp M. (2007). "Niaspan increases angiogenesis and improves functional recovery after stroke." Ann Neurol. Epub Ahead of Print. [PDF Full-Text](#)

Department of Neurology, Henry Ford Hospital, Detroit, MI.

OBJECTIVE: High-density lipoprotein (HDL) is implicated in the modulation of angiogenesis. In this study, we investigated whether the Niacin-mediated increase of HDL regulates angiogenesis and thereby improves functional outcome after stroke. **METHODS:** Adult male rats were subjected to middle cerebral artery occlusion and were treated with or without different doses (40 and 80mg/kg) of Niaspan, starting 24 hours after middle cerebral artery occlusion and daily for 14 days. Neurological functional tests were performed, and serum HDL level was measured. Angiogenesis and angiogenic factor expression were measured by immunohistochemistry, corneal neovascularization and capillary tube formation assay, and Western blot, respectively. **RESULTS:** Niaspan significantly increased HDL level, promoted angiogenesis in the ischemic brain, and improved functional outcome after stroke. Niaspan also significantly increased corneal neovascularization compared with nontreatment control. Mechanisms underlying the Niaspan-induced vascular remodeling were investigated. Niaspan increased the expression of vascular endothelial growth factor and angiopoietin-1 (Ang1), and phosphorylation of Akt, endothelial nitric oxide synthase (NOS), and Tie2 in the ischemic brain. Niacin upregulated Ang1 expression in cultured brain endothelial cells and increased vascular endothelial growth factor, Ang1, and endothelial NOS expression in cultured astrocytes, and dose-dependently increased capillary tube formation compared with nontreatment control. Inhibition of NOS partially decreased Niacin-induced capillary tube formation. Inhibition of phosphoinositide 3-kinase or knockdown of Tie2 substantially and significantly decreased Niacin-induced capillary tube formation. **INTERPRETATION:** Niacin increases HDL and promotes angiogenesis, which may contribute to improvement of functional outcome after stroke. The Ang1/Tie2, phosphoinositide 3-kinase/Akt, and endothelial NOS pathways appear to mediate Niacin-induced angiogenesis.

Chong B, Nydorf E and Lim H. (2007). "Concomitant morphea and pigmented purpuric dermatosis in a 62-year-old woman." J Eur Acad Dermatol Venereol **21**(6): 826-8. [PDF Full-Text](#)

Department of Dermatology, Henry Ford Medical Center - New Center One, 3031 West Grand Blvd., Suite 800, Detroit, MI 48202, USA.

Decarvalho AC, Zhang X, Roberts C, Jiang F, Kalkanis SN, Hong X, Lu M and Chopp M. (2007). "Subclinical photodynamic therapy treatment modifies the brain microenvironment and promotes glioma growth." Glia **55**(10): 1053-60. [PDF Full-Text](#)

Departments of Neurology, Henry Ford Health Sciences Center, Detroit, Michigan.

Photodynamic therapy (PDT) has been clinically investigated as an adjuvant local therapy for brain tumors. Therapeutic interventions intended to promote tumor cell death can also promote changes in the tumor

microenvironment that could favor tumor growth. We have previously shown that PDT can activate pro-angiogenic factors in the normal rodent brain. This study seeks to further elucidate the effects of subtherapeutic doses of Photofrin-PDT on normal brain and to establish a mouse model for studying glioma progression in an environment modified by oxidative stress. Photofrin was administered to nude mice, and a defined intracranial area was illuminated with laser to deliver an optical dose equivalent to 80 J/cm². Three and 7 days after PDT, mice were sacrificed and brains were fixed and analyzed by immunohistochemistry. PDT treatment resulted in transient increase in cell proliferation, associated with a robust activation of astrocytes and microglia in the treated region, without causing substantial cell death. To test how this modified environment would affect glioma growth, human glioblastoma U87 cells were implanted in the PDT-treated hemisphere or in the control brain subjected to sham surgery. Significantly larger tumors were observed after 3 weeks in the PDT treated brains relative to control treatment. Our results indicate that subclinical Photofrin-PDT locally alters the brain homeostasis without inflicting significant disruption to the tissue architecture, providing a model to study the effects of the microenvironment on glioma growth, with implications for the optimization of the clinical use of PDT for brain tumors.

Deeb D, Jiang H, Gao X, Al-Holou S, Danyluk AL, Dulchavsky SA and Gautam SC. (2007). "Curcumin [1,7-bis(4-hydroxy-3-methoxyphenyl)-1-6-heptadine-3,5-dione; C₂₁H₂₀O₆] sensitizes human prostate cancer cells to tumor necrosis factor-related apoptosis-inducing ligand/Apo2L-induced apoptosis by suppressing nuclear factor-kappaB via inhibition of the prosurvival Akt signaling pathway." *J Pharmacol Exp Ther* **321**(2): 616-25. [PDF Full-Text](#)

Department of Surgery, Henry Ford Health System, Detroit, MI 48202, USA.

Our previous studies have shown that dietary pigment curcumin [1,7-bis(4-hydroxy-3-methoxyphenyl)-1-6-heptadine-3,5-dione; C₂₁H₂₀O₆] sensitizes human prostate cancer cells to tumor necrosis factor-related apoptosis-inducing ligand (TRAIL/Apo2L)-induced apoptosis by inhibiting nuclear factor (NF)-kappaB. In the present study, we demonstrate that activated (phosphorylated) Akt kinase plays a pivotal role in regulation of NF-kappaB and sensitization of LNCaP and PC3 prostate cancer cells to TRAIL by curcumin. Curcumin inhibited the expression of phospho-Akt (p-Akt), which was not due to activation of phosphatase and tensin homolog deleted on chromosome 10 phosphatase activity by curcumin. Because NF-kappaB is a downstream target of Akt, we investigated whether inhibition of NF-kappaB by curcumin is mediated through suppression of p-Akt. Data demonstrate that treatment of PC3 cells with SH-6 (JAm Chem Soc 125:1144-1145, 2003), a specific inhibitor of Akt, or transfection with small inhibitory RNA (siRNA)-Akt not only inhibited p-Akt but also abrogated the expression and transcriptional activity of NF-kappaB. Furthermore, overexpression of constitutively active Akt1 in cancer cells prevented the inhibition of NF-kappaB by curcumin. In addition, treatment with SH-6 or transfection with siRNA-Akt sensitized PC3 cells to TRAIL-induced cytotoxicity. On the other hand, SH-6 does not inhibit NF-kappaB or sensitize DU145 cancer cells to TRAIL because these cells do not express p-Akt. Because expression of antiapoptotic Bcl-2, Bcl-xL, and X-chromosome-linked inhibitor of apoptosis protein (XIAP) is regulated by NF-kappaB, both curcumin and SH-6 decreased the levels of these proteins in PC3 cells through inhibition of NF-kappaB. Furthermore, gene silencing of Bcl-2 with siRNA-Bcl-2 sensitized PC3 cells to TRAIL. Collectively, these data define a pathway whereby curcumin sensitizes prostate cancer cells to TRAIL by inhibiting Akt-regulated NF-kappaB and NF-kappaB-dependent antiapoptotic Bcl-2, Bcl-xL, and XIAP.

Dereczyk D and Kunkel P. (2007). "Cervical spine clearance in blunt trauma: evaluation of a computed tomography-based protocol." *J Trauma Nurs* **14**(2): 114-5. [PDF Full-Text](#)

Darlene Dereczyk, BSN, RN, is the Trauma Clinical Coordinator and Patti Kunkel, BSN, RN, is the Trauma Injury Prevention Outreach Coordinator at Henry Ford Health System, Detroit, Mich.

Drake CL. (2007). "Managing chronic insomnia in the psychiatric patient." Psychiatric Times 1-4. **Full-Text Not Available** / [Click for Article Request Form](#)

Sleep Disorders and Research Center, Henry Ford Hospital.

Elder JS. (2007). "Circumcision." BJU Int **99**(6): 1553-64. [PDF Full-Text](#)

Department of Urology, Henry Ford Health System and Vattikuti Urology Institute, Detroit, Michigan, USA. jack.elder@case.edu

El-Essawi D, Musial JL, Hammad A and Lim HW. (2007). "A survey of skin disease and skin-related issues in Arab Americans." J Am Acad Dermatol **56**(6): 933-8. [PDF Full-Text](#)

Multicultural Dermatology Center, Department of Dermatology, Henry Ford Hospital, Detroit, USA.

BACKGROUND: There is a paucity of knowledge relating to dermatologic conditions in Arab Americans. **OBJECTIVE:** To assess common skin diseases and concerns and to evaluate access to dermatologic care and perception of skin in Arab Americans. **METHODS:** Arab Americans from 3 Southeast Michigan locations (community health center [n = 207], mosque [n = 95], and church [n = 99]) completed a survey questionnaire. **RESULTS:** The most common self-reported skin conditions were acne, eczema/dermatitis, warts, fungal skin infections, and melasma. The most pressing skin concerns were uneven skin tone, skin discoloration, dry skin, acne, and facial hair. Significant associations exist between socioeconomic status and having seen a dermatologist. Attitudes surrounding skin perception were related to the number of years of residence in the United States. **LIMITATIONS:** The skin condition data were gathered from a self-reported survey. **CONCLUSIONS:** Skin conditions and other related issues that affect Arab Americans are similar to those which affect other skin-of-color populations.

Freytag SO, Barton KN, Brown SL, Narra V, Zhang Y, Tyson D, Nall C, Lu M, Ajlouni M, Movsas B and Kim JH. (2007). "Replication-competent Adenovirus-mediated Suicide Gene Therapy with Radiation in a Preclinical Model of Pancreatic Cancer." Mol Ther. Epub Ahead of Print. **Full-Text Not Available** / [Click for Article Request Form](#)

Department of Radiation Oncology, Henry Ford Health System, Detroit, Michigan, USA.

In preparation for a Phase I trial, we evaluated the efficacy and toxicity of replication-competent adenovirus-mediated suicide gene therapy in combination with radiation in a preclinical model of pancreatic cancer. Human MiaPaCa-2 and PANC-1 pancreatic adenocarcinoma cells were found to be sensitive to the oncolytic effects of the Ad5-yCD/mutTK(SR39)rep-ADP adenovirus and also to the cytotoxic effects of the yeast cytosine deaminase (yCD) and herpes simplex virus thymidine kinase (HSV-1 TK(SR39)) genes in vitro. Combining Ad5-yCD/mutTK(SR39)rep-ADP-mediated suicide gene therapy with radiation significantly increased tumor control beyond that of either modality alone. Injection of Ad5-yCD/mutTK(SR39)rep-ADP in the dog pancreas at doses (10¹² virus particle (vp)) to be used in humans resulted in mild pancreatitis but not peritonitis or hepatotoxicity. Following administration of 9-(4-[(18)F]-fluoro-3-hydroxymethylbutyl)guanine ([18F]-FHBG), a positron-emitting substrate of HSV-1 TK, Ad5-yCD/mutTK(SR39)rep-ADP activity could be monitored non-invasively by positron emission tomography (PET). [18F]-FHBG uptake was readily detected in the pancreas but not in other major abdominal organs, indicating that little of the injected adenovirus disseminates to collateral tissues. These results demonstrate that Ad5-yCD/mutTK(SR39)rep-ADP-mediated suicide gene therapy has the potential to augment the effectiveness of pancreatic radiotherapy without resulting in excessive toxicity. Hence they provide the scientific basis for an ongoing Phase I trial in pancreatic cancer.

Gomel R, Xiang C, Finnis S, Lee HK, Lu W, Okhrimenko H and Brodie C. (2007). "The localization of protein kinase Cdelta in different subcellular sites affects its proapoptotic and antiapoptotic functions and the activation of distinct downstream signaling pathways." Mol Cancer Res **5**(6): 627-39. **Full-Text Not Available** / [Click for Article Request Form](#)

Hermelin Brain Tumor Center, Department of Neurosurgery, Henry Ford Hospital, Detroit, MI 48202, USA.

Protein kinase Cdelta (PKCdelta) regulates cell apoptosis and survival in diverse cellular systems. PKCdelta translocates to different subcellular sites in response to apoptotic stimuli; however, the role of its subcellular localization in its proapoptotic and antiapoptotic functions is just beginning to be understood. Here, we used a PKCdelta constitutively active mutant targeted to the cytosol, nucleus, mitochondria, and endoplasmic reticulum (ER) and examined whether the subcellular localization of PKCdelta affects its apoptotic and survival functions. PKCdelta-Cyto, PKCdelta-Mito, and PKCdelta-Nuc induced cell apoptosis, whereas no apoptosis was observed with the PKCdelta-ER. PKCdelta-Cyto and PKCdelta-Mito underwent cleavage, whereas no cleavage was observed in the PKCdelta-Nuc and PKCdelta-ER. Similarly, caspase-3 activity was increased in cells overexpressing PKCdelta-Cyto and PKCdelta-Mito. In contrast to the apoptotic effects of the PKCdelta-Cyto, PKCdelta-Mito, and PKCdelta-Nuc, the PKCdelta-ER protected the cells from tumor necrosis factor-related apoptosis-inducing ligand-induced and etoposide-induced apoptosis. Moreover, overexpression of a PKCdelta kinase-dead mutant targeted to the ER abrogated the protective effect of the endogenous PKCdelta and increased tumor necrosis factor-related apoptosis-inducing ligand-induced apoptosis. The localization of PKCdelta differentially affected the activation of downstream signaling pathways. PKCdelta-Cyto increased the phosphorylation of p38 and decreased the phosphorylation of AKT and the expression of X-linked inhibitor of apoptosis protein, whereas PKCdelta-Nuc increased c-Jun NH(2)-terminal kinase phosphorylation. Moreover, p38 phosphorylation and the decrease in X-linked inhibitor of apoptosis protein expression played a role in the apoptotic effect of PKCdelta-Cyto, whereas c-Jun NH(2)-terminal kinase activation mediated the apoptotic effect of PKCdelta-Nuc. Our results indicate that the subcellular localization of PKCdelta plays important roles in its proapoptotic and antiapoptotic functions and in the activation of downstream signaling pathways.

Herrera M and Garvin JL. (2007). "Novel role of AQP-1 in NO-dependent vasorelaxation." Am J Physiol Renal Physiol **292**(5): F1443-51. [PDF Full-Text](#)

Henry Ford Hospital, Division of Hypertension and Vascular Research, Wayne State University, Detroit, Michigan 48202, USA. mherrer1@hfhs.org.

Nitric oxide (NO) produced by endothelial cells diffuses to vascular smooth muscle cells to cause dilatation of the renal vasculature and other vessels. Although it is generally assumed that NO moves from cell to cell by free diffusion, we recently showed that aquaporin-1 (AQP-1) transports NO across cell membranes. AQP-1 is expressed in endothelial and vascular smooth muscle cells. We hypothesized that diffusion of NO into vascular smooth muscle cells and out of endothelial cells is facilitated by AQP-1, and transport of NO by AQP-1 is involved in endothelium-dependent relaxation. In intact aortic rings from AQP-1 $-/-$ mice, vasorelaxation induced by acetylcholine (which increases endogenous NO) was reduced ($P < 0.0001$ vs. control). No differences were found in the relaxation caused by intracellular delivery of NO or intracellular cGMP between strains. In endothelium-denuded aortic rings from AQP-1 $-/-$ mice, the vasorelaxant capability of NO released in the extracellular space was reduced ($P < 0.0001$ vs. control). Influx of NO (5 microM) into vascular smooth muscle cells was 0.17 ± 0.02 f.u./s for control and 0.07 ± 0.01 f.u./s for AQP-1 $-/-$ mice, 62% lower ($P < 0.002$). NO released by endothelial cells in response to 1 microM acetylcholine was 96.2 ± 17.7 pmol NO/mg for control and 41.9 ± 13.4 pmol NO/mg for AQP-1 $-/-$ mice, 56% reduction ($P < 0.04$). NOS3 expression was 1.33 ± 0.29 O.D. units for control and 3.84 ± 0.76 O.D. units for AQP-1 $-/-$ mice, 188% increase ($P < 0.01$). We conclude that 1) AQP-1 facilitates NO

influx into vascular smooth muscle cells, 2) AQP-1 facilitates NO diffusion out of endothelial cells, and 3) transport of NO by AQP-1 is required for full expression of endothelium-dependent relaxation.

Hong X, Jiang F, Kalkanis SN, Zhang ZG, Zhang X, Zheng X, Mikkelsen T, Jiang H and Chopp M. (2007). "Decrease of endogenous vascular endothelial growth factor may not affect glioma cell proliferation and invasion." J Exp Ther Oncol 6(3): 219-29. **Full-Text Not Available / [Click for Article Request Form](#)**

Department of Neurosurgery, Henry Ford Health Science Center, Detroit, MI 48202, USA.

Vascular endothelial growth factor (VEGF) is abundantly produced by glioma cells especially glioblastoma, the most malignant form of astrocytoma. VEGF, a well known angiogenic factor, acts in a paracrine fashion on endothelial cells to develop tumor vasculature. However, recent studies have found that several tumor cells express VEGF receptors, and an autocrine action of VEGF on tumor cells has been suggested. To test this hypothesis, three human glioma cell lines (U251n, U87 and A172) were checked for VEGF and VEGFR expression. These cells express 0.1-0.6 ng/ml VEGF₁₆₅ in cell culture medium within 24 hours. Western blot analysis showed that these cells express all of the VEGF receptors, VEGFR-1/Flt-1, VEGFR-2/KDR, Neuropilin-1 (NRP-1) and Neuropilin-2(NRP-2), even though tyrosine kinase receptor VEGFR-2/KDR exhibited baseline levels of expression. VEGF expression was significantly down regulated by phosphorothioate oligodeoxynucleotide (PS-ODN) and VEGF RNAi transfection. However, VEGF RNAi transfection as well as VEGF and VEGFR2 neutralization antibody treatment did not decrease cell proliferation detected by MTT and CyQuant NF proliferation assay except that PS-ODN transfection caused a non-specific decrease on cell proliferation. VEGF RNAi transfection did not alter cell invasion, as demonstrated in a matrigel invasion assay. Matrix metalloproteinase-2 (MMP-2) and MMP-9, facilitating cell invasion and over expressed in glioma cells, were not altered by VEGF RNAi transfection, as shown by zymographic assays. Our data indicate that the decrease of endogenous VEGF expression may not affect glioma cell proliferation and invasion.

Hurbanek JG, Anderson K, Kaatz S, Shepard A, Workings M and Rand K. (2007). "Ulnar Deep Venous Thrombosis in a Professional Baseball Pitcher: A Case Report." Am J Sports Med. Epub Ahead of Print. [PDF Full-Text](#)

Henry Ford Hospital, Detroit, Michigan.

Joseph CL, Peterson E, Havstad S, Johnson CC, Hoerauf S, Stringer S, Gibson-Scipio W, Ownby DR, Elston-Lafata J, Pallonen U and Strecher V. (2007). "A web-based, tailored asthma management program for urban African-American high school students." Am J Respir Crit Care Med 175(9): 888-95. [PDF Full-Text](#)

Senior Staff Epidemiologist, Henry Ford Health System, Department of Biostatistics & Research Epidemiology, Detroit, MI 48202, USA. cjoseph1@hfhs.org.

RATIONALE: Urban African-American youth, aged 15-19 years, have asthma fatality rates that are higher than in whites and younger children, yet few programs target this population. Traditionally, urban youth are believed to be difficult to engage in health-related programs, both in terms of connecting and convincing. **OBJECTIVES:** Develop and evaluate a multimedia, web-based asthma management program to specifically target urban high school students. The program uses "tailoring," in conjunction with theory-based models, to alter behavior through individualized health messages based on the user's beliefs, attitudes, and personal barriers to change. **METHODS:** High school students reporting asthma symptoms were randomized to receive the tailored program (treatment) or to access generic asthma websites (control). The program was made available on school computers. **MEASUREMENTS AND MAIN RESULTS:**

Functional status and medical care use were measured at study initiation and 12 months postbaseline, as were selected management behaviors. The intervention period was 180 days (calculated from baseline). A total of 314 students were randomized (98% African American, 49% Medicaid enrollees; mean age, 15.2 yr). At 12 months, treatment students reported fewer symptom-days, symptom-nights, school days missed, restricted-activity days, and hospitalizations for asthma when compared with control students; adjusted relative risk and 95% confidence intervals were as follows: 0.5 (0.4-0.8), $p = 0.003$; 0.4 (0.2-0.8), $p = 0.009$; 0.3 (0.1-0.7), $p = 0.006$; 0.5 (0.3-0.8), $p = 0.02$; and 0.2 (0.2-0.9), $p = 0.01$, respectively. Positive behaviors were more frequently noted among treatment students compared with control students. Cost estimates for program delivery were \$6.66 per participating treatment group student. **CONCLUSIONS:** A web-based, tailored approach to changing negative asthma management behaviors is economical, feasible, and effective in improving asthma outcomes in a traditionally hard-to-reach population.

Kaul SA and Menon M. (2007). "Da Vinci assisted cystoprostatectomy and urinary diversion: a paradigm shift in surgical management of bladder cancer." *Minerva Urol Nefrol* **59**(2): 149-57. **Full-Text Not Available / [Click for Article Request Form](#)**

Vattikuti Urology Institute, Henry Ford Health System, Detroit, MI, USA skaul1@hfhs.org.

Radical cystoprostatectomy remains the gold standard treatment for muscle invasive bladder cancer. Use of minimally invasive approaches have gained prominence aided by surgical adjuncts such as harmonic scalpel and laparoscopic bowel staplers, however laparoscopic radical cystoprostatectomy remains extremely technically challenging even for experienced laparoscopic surgeons. Following the successful application of the da Vinci robotic surgical system for radical prostatectomy, attention has now turned to the use of robot assistance for laparoscopic cystoprostatectomy. Several centers have explored the feasibility of robotic cystoprostatectomy although long-term data is lacking. Controversy exists on the oncologic efficacy and safety, need for intracorporeal diversion and standardization of technique. This article details the history, technique, results and current status of robotic cystoprostatectomy and urinary diversion.

Lash TL, Fox MP, Buist DS, Wei F, Field TS, Frost FJ, Geiger AM, Quinn VP, Yood MU and Silliman RA. (2007). "Mammography Surveillance and Mortality in Older Breast Cancer Survivors." *J Clin Oncol.* Epub Ahead of Print. [PDF Full-Text](#)

Departments of Epidemiology and International Health, Boston University School of Public Health; Geriatrics Section, Department of Medicine, Boston University School of Medicine, Boston; University of Massachusetts Medical School, Worcester; Fallon Community Health Plan, Worcester, MA; Group Health Center for Health Studies, Seattle, WA; HealthPartners Research Foundation, Minneapolis, MN; Lovelace Health Systems, Albuquerque, NM; Wake Forest University School of Medicine, Winston-Salem, NC; Kaiser Permanente Southern California, Pasadena, CA; Henry Ford Health System, Detroit, MI; and the Yale University School of Medicine, New Haven, CT.

PURPOSE: There are more than 2,000,000 breast cancer survivors in the United States today. While surveillance for asymptomatic recurrence and second primary is included in consensus recommendations, the effectiveness of this surveillance has not been well characterized. Our purpose is to estimate the effectiveness of surveillance mammography in a cohort of breast cancer survivors with complete ascertainment of surveillance mammograms and negligible losses to follow-up. **PATIENTS AND METHODS:** We enrolled 1,846 stage I and II breast cancer patients who were at least 65 years old at six integrated health care delivery systems. We used medical record review and existing databases to ascertain patient, tumor, and therapy characteristics, as well as receipt of surveillance mammograms. We linked personal identifiers to the National Death Index to ascertain date and cause of death. We matched four controls to each breast cancer decedent to estimate the association between receipt of surveillance

mammogram and breast cancer mortality. RESULTS: One hundred seventy-eight women died of breast cancer during 5 years of follow-up. Each additional surveillance mammogram was associated with a 0.69-fold decrease in the odds of breast cancer mortality (95% CI, 0.52 to 0.92). The protective association was strongest among women with stage I disease, those who received mastectomy, and those in the oldest age group. CONCLUSION: Given existing recommendations for post-therapy surveillance, trials to compare surveillance with no surveillance are unlikely. This large observational study provides support for the recommendations, suggesting that receipt of surveillance mammograms reduces the rate of breast cancer mortality in older patients diagnosed with early-stage disease.

Li XC, Navar LG, Shao Y and Zhuo JL. (2007). "Genetic deletion of AT1a receptors attenuates intracellular accumulation of angiotensin II in the kidney of AT1a receptor-deficient mice." *Am J Physiol Renal Physiol.* Epub Ahead of Print. [PDF Full-Text](#)

Hypertension and Vascular Division, Henry Ford Hospital, Detroit, Michigan, United States.

We and others have shown that angiotensin II (Ang II) is accumulated in the rat kidney via an AT1 receptor-mediated mechanism, but it is not known which AT1 receptor mediates this response. We tested the hypothesis that AT1a receptor-deficient mice (*Agtr1a^{-/-}*) are unable to take up Ang II intracellularly in the kidney due to the absence of AT1a receptor-mediated endocytosis. Wild-type (*Agtr1a^{+/+}*), heterozygous (*Agtr1a^{+/-}*) and *Agtr1a^{-/-}* were treated with vehicle, Ang II (40 ng/min, i.p.), or Ang II plus losartan (10 mg/kg/day, p.o.) for 2 weeks. In wild-type mice, Ang II induced hypertension, increased kidney weight, caused pressure natriuresis, and elevated plasma and whole kidney Ang II levels ($p < 0.01$). These responses to Ang II were attenuated by losartan. *Agtr1a^{-/-}* mice had lower basal systolic pressure, smaller kidneys with fewer AT1b receptors, higher basal 24 hour urinary sodium excretion, as well as basal plasma and whole kidney Ang II levels ($p < 0.01$). However, intracellular Ang II levels in the kidney were lower in *Agtr1a^{-/-}* mice. In *Agtr1a^{-/-}* mice, Ang II slightly increased systolic pressure, but had no effect on the kidney weight, urinary sodium excretion, and whole kidney Ang II levels. Losartan restored systolic pressure to basal levels and decreased whole kidney Ang II levels by ~ 20% ($p < 0.05$). These results demonstrate a predominant role of AT1a receptors in overall blood pressure and renal regulation in response to Ang II, but also suggest that AT1b receptors may play a limited role when AT1a receptors are deleted.

Lu M, Zhang RL, Zhang ZG, Yang JJ and Chopp M. (2007). "Linkage of cell cycle kinetics between embryonic and adult stroke models: an analytical approach." *J Neurosci Methods* **161**(2): 323-30. **Full-Text Not Available** / [Click for Article Request Form](#)

Department of Biostatistics and Research Epidemiology, Henry Ford Health Sciences Center, One Ford Place, Ste. 3E, Detroit, MI 48202, USA. mlu1@hfhs.org.

In the adult brain, neurogenesis occurs in the subventricular zone (SVZ) of the lateral ventricle. The proliferating population and the cell cycle kinetics in the ventricular zone regulate cortical neurogenesis during the development. Using the embryonic model, we investigated kinetics of SVZ cells in adult rats after stroke, incorporating migration of SVZ cells to the ischemic boundary. Concurrent linear regressions were considered through iteration to improve precision of parameter estimation. We found no model-fit difference in stroke with and without the migration ($p = 0.31$), suggesting no migration effect on assessment of the cell kinetics. Stroke increased SVZ cell proliferation (20% in non-stroke and 31% in stroke $p < 0.01$). Cell cycle durations in stroke were reduced for the total cycle length (19h for non-stroke and 15.3h for stroke, $p < 0.05$), in G1 phase (12.6 h for non-stroke and 9.6 h for stroke, $p < 0.01$), but were the same in S, M2, and in G2 phases compared to non-stroke, indicating that stroke reduces the total cell cycle length, specially in G1 phase. We conclude that cell cycle kinetic models for cortical development can be adapted to the kinetics of adult SVZ cells after stroke. The analytical approach may be useful for studying neural progenitor cell proliferation under different treatments.

Maltsev VA, Silverman N, Sabbah HN and Undrovinas AI. (2007). "Chronic heart failure slows late sodium current in human and canine ventricular myocytes: implications for repolarization variability." Eur J Heart Fail **9**(3): 219-27. [PDF Full-Text](#)

Heart and Vascular Institute, Henry Ford Health System, Detroit, Michigan 48202-2689, United States.

BACKGROUND: Late Na⁽⁺⁾ current (I_{NaL}) in human and dog hearts has been implicated in abnormal repolarization associated with heart failure (HF). HF slows inactivation gating of late Na⁽⁺⁾ channels, which could contribute to these abnormalities. **AIMS:** To test how altered gating affects I_{NaL} time course, Na⁽⁺⁾ influx, and action potential (AP) repolarization. **METHODS:** I_{NaL} and AP were measured by patch clamp in left ventricular cardiomyocytes from normal and failing hearts of humans and dogs. Canine HF was induced by coronary microembolization. **RESULTS:** I_{NaL} decay was slower and I_{NaL} density was greater in failing hearts than in normal hearts at 24 degrees C (human hearts: tau=659±16 vs. 529±21 ms; n=16 and 4 hearts, respectively; mean±SEM; p<0.002; dog hearts: 561±13 vs. 420±17 ms; and 0.307±0.014 vs. 0.235±0.019 pA/pF; n=25 and 14 hearts, respectively; p<0.005) and at 37 degrees C this difference tended to increase. These I_{NaL} changes resulted in much greater (53.6%) total Na⁽⁺⁾ influx in failing cardiomyocytes. I_{NaL} was sensitive to cadmium but not to cyanide and exhibited low sensitivity to saxitoxin (IC₅₀=62 nM) or tetrodotoxin (IC₅₀=1.2 μM), tested in dogs. A 50% I_{NaL} inhibition by toxins or passing current opposite to I_{NaL}, decreased beat-to-beat AP variability and eliminated early afterdepolarizations in failing cardiomyocytes. **CONCLUSIONS:** Chronic HF leads to larger and slower I_{NaL} generated mainly by the cardiac-type Na⁽⁺⁾ channel isoform, contributing to larger Na⁽⁺⁾ influx and AP duration variability. Interventions designed to reduce/normalize I_{NaL} represent a potential cardioprotective mechanism in HF via reduction of related Na⁽⁺⁾ and Ca⁽²⁺⁾ overload and improvement of repolarization.

Moser HW and Mahmood A. (2007). "New insights about hematopoietic stem cell transplantation in adrenoleukodystrophy." Arch Neurol **64**:631-2. [PDF Full-Text](#)

Department of Neurosurgery, Henry Ford Hospital.

Rempel SA, Hawley RC, Gutierrez JA, Mouzon E, Bobbitt KR, Lemke N, Schultz CR, Schultz LR, Golembieski W, Koblinski J, VanOsdol S and Miller CG. (2007). "Splenic and immune alterations of the Sparc-null mouse accompany a lack of immune response." Genes Immun **8**(3): 262-74. **Full-Text Not Available** / [Click for Article Request Form](#)

Department of Neurosurgery, Barbara Jane Levy Laboratory of Molecular Neuro-Oncology, Hermelin Brain Tumor Center, Henry Ford Hospital, Detroit, MI, USA. nssan@neuro.hfh.edu.

Sparc-null mice have been used as models to assess tumor-host immune cell interactions. However, it is not known if they have a competent immune system. In this study, the immune systems of Sparc wild-type and null mice were compared. Mice were assessed for differences in total body weight, spleen weight and spleen-to-body weight ratios. Spleens were compared with respect to morphology, and Sparc, Ki-67, MOMA-1 and IgM expression. Immune cells in blood, bone marrow and spleen were assessed by blood smears, automated blood panel, and flow cytometry. Additionally, the ability of Sparc-null mice to respond to immune challenge was evaluated using a footpad model. The morphological and immunohistochemical results indicated that Sparc-null spleens had more white pulp, hyperproliferative B cells in the germinal centers, and decreased marginal zones. Sparc-null spleens lacked normal Sparc expression in red and white pulp, marginal zones, endothelial and sinusoidal cells. By flow analysis, B cells were decreased and T cells were increased in the bone marrow. Finally, Sparc-null mice were unable to mount an immune response following footpad lipopolysaccharide challenge. These data confirm that Sparc-null mice have an impaired immune system.

Ren Y, Garvin JL, Liu R and Carretero OA. (2007). "Possible mechanism of efferent arteriole (Ef-Art) tubuloglomerular feedback." *Kidney Int* **71**(9): 861-6. [PDF Full-Text](#)

Hypertension and Vascular Research Division, Henry Ford Hospital, Detroit, Michigan 48202, USA. yren1@hfhs.org.

Adenosine triphosphate (ATP) is liberated from macula densa cells in response to increased tubular NaCl delivery. However, it is not known whether ATP from the macula densa is broken down to adenosine, or whether this adenosine mediates efferent arteriole (Ef-Art) tubuloglomerular feedback (TGF). We hypothesized that increased macula densa Ca(2+), release of ATP and degradation of ATP to adenosine are necessary for Ef-Art TGF. Rabbit Ef-Arts and adherent tubular segments (with the macula densa) were simultaneously microperfused in vitro while changing the NaCl concentration at the macula densa. The Ef-Art was perfused orthograde through the end of the afferent arteriole (Af-Art). In Ef-Arts precontracted with norepinephrine (NE), increasing NaCl concentration from 10 to 80 mM at the macula densa dilated Ef-Arts from 7.5+/-0.7 to 11.1+/-0.3 microm. Buffering increases in macula densa Ca(2+) with the cell-permeant Ca(2+) chelator BAPTA-AM diminished Ef-Art TGF from 3.1+/-0.3 to 0.1+/-0.2 microm. Blocking adenosine formation by adding alpha-beta-methyleneadenosine 5'-diphosphate (MADP) blocked Ef-Art TGF from 2.9+/-0.5 to 0.1+/-0.2 microm. Increasing luminal NaCl at the macula densa from 10 to 45 mM caused a moderate Ef-Art TGF response, 1.3+/-0.1 microm. It was potentiated to 4.0+/-0.3 microm by adding hexokinase, which enhances conversion of ATP into adenosine. Our data show that in vitro changes in macula densa Ca(2+) and ATP release are necessary for Ef-Art TGF. ATP is broken down to form adenosine, which mediates signal transmission of Ef-Art TGF.

Robinson SJ, Tenney K, Yee DF, Martinez L, Media JE, Valeriote FA, Soest RW and Crews P. (2007). "Probing the Bioactive Constituents from Chemotypes of the Sponge *Psammocinia aff. bulbosa*." *J Nat Prod* **70**(6): 1002-09. **Full-Text Not Available** / [Click for Article Request Form](#)

Department of Chemistry and Biochemistry and Institute for Marine Sciences, University of California, Santa Cruz, Santa Cruz, California 95064, Josephine Ford Cancer Center, Henry Ford Health System, Detroit, Michigan 48202, and Zoologisch Museum, University of Amsterdam, P.O. Box 94766, 1090 GT, Amsterdam, The Netherlands.

Since the report of (+)-psymberin (2) in 2004, many synthetic groups have pursued the synthesis of this compound, and our group has further collected *Psammocinia aff. bulbosa* to successfully isolate more 2. With more (+)-psymberin (2) in hand, additional clonogenic studies, a therapeutic efficacy assessment, and the hollow fiber assay have been completed. The inconsistent production of (+)-psymberin (2) and the classification of six *Psammocinia* species are further discussed herein. The most recent of these six collections resulted in the isolation of a new brominated cyclic peptide, (-)-psymbamide A (4), which is the first report of a *Psammocinia*-derived compound in this peptide class. The planar structure was solved via dereplication with Marinlit, HRESIMS, and 1D and 2D NMR techniques, and the absolute configuration determined using Marfey's method.

Shafi ST and Gupta M. (2007). "Risk of vascular access thrombosis in patients with systemic lupus erythematosus on hemodialysis." *J Vasc Access* **8**(2): 103-8. **Full-Text Not Available** / [Click for Article Request Form](#)

Division of Nephrology, Department of Internal Medicine, University of Iowa Hospitals and Clinics, Iowa City IA - USA.

Introduction: Anticardiolipin antibody is associated with increased risk of vascular access thrombosis (VAT) in hemodialysis (HD) patients. Systemic lupus erythematosus (SLE) patients have a high prevalence of anticardiolipin antibodies, but it is not clear whether these patients are at high risk of developing vascular access thrombosis. Methods: Thirty six SLE patients on HD, who had either an arterio-venous (AV) graft or AV fistula as vascular access, and were not on anticoagulation treatment, were retrospectively identified at Henry Ford Hospital. A similar number of patients without SLE, matched for age, sex, race and type of vascular access were selected as a control population. Vascular access thrombosis rate at one year was compared between two groups. Results: Mean age at dialysis was 36+/-10 years in 36 SLE patients (28 females, 8 males) and was 38+/-6.4 years in 36 non-SLE patients (29 females, 7 males). Of all patients, 29/36 (80.5%) SLE and 27/36 (75%) non-SLE patients had AV grafts, whereas the rest had AV fistulas as vascular access (19.5% SLE and 25% non-SLE patients). Out of 36 SLE patients, 24 (66.6%) patients developed VAT at one year as compared to 14 (38.9%) patients in non-SLE group ($p < 0.05$). The odds ratio of VAT in SLE patients was 3.1 (95% CI 1.2-8.2). Conclusion: SLE patients on hemodialysis are more likely to develop vascular access thrombosis as compared to non-SLE patients.

Siddiqui F, Barton KN, Stricker HJ, Steyn PF, Larue SM, Karvelis KC, Sparks RB, Kim JH, Brown SL and Freytag SO. (2007). "Design considerations for incorporating sodium iodide symporter reporter gene imaging into prostate cancer gene therapy trials." Hum Gene Ther **18**(4): 312-22. **Full-Text Not Available** / [Click for Article Request Form](#)

Department of Radiation Oncology, Henry Ford Health System, Detroit, MI 48202.

This study was done to aid in the design of a phase I gene therapy trial in patients with prostate cancer. We determined the dosimetric characteristics of our reporter gene system when coupled with intravenous administration of radioactive sodium pertechnetate ($^{99m}\text{TcO}(4)$) and determined the feasibility of using human sodium iodide symporter (hNIS) as a reporter gene to study the dynamics of adenoviral transgene expression in a large animal tumor. A replication-competent Ad5-yCD/mutTK(SR39) rep-hNIS adenovirus was injected into the prostate gland of dogs for dosimetry purposes, and into a canine soft tissue sarcoma (STS) for imaging purposes. After resection of the prostate, the amount of ($^{99m}\text{TcO}(4)$) sequestered in the prostate was determined, the radiation dose absorbed by the prostate and nontarget critical organs was calculated, and hNIS reporter gene expression was imaged in the STS by single-photon emission computed tomography (SPECT). On the basis of the findings from 25 dogs, the amount of ($^{99m}\text{TcO}(4)$) sequestered in the prostate ranged from 13 to 276 μCi . Using the highest value observed, absorbed radiation dose to critical organs was calculated and found to be below U.S. Food and Drug Administration limits for diagnostic imaging. Also, ($^{99m}\text{TcO}(4)$) uptake was readily detected by SPECT and found to persist *in vivo* for at least 4 days. On the basis of our dosimetry calculations, up to five imaging procedures can be safely performed in humans after intraprostatic injection of the Ad5-yCD/mutTK(SR39)rep-hNIS adenovirus and the hNIS reporter gene system can be used to study the dynamics of adenoviral gene therapy vectors in large animal tumors.

Silver B, Greenbaum A and McCarthy S. (2007). "Improvement in sleep apnea associated with closure of a patent foramen ovale." J Clin Sleep Med **3**(3): 295-6. [PDF Full-Text](#)

Department of Neurology, Henry Ford Hospital, Detroit, MI 48202, USA. silver@neuro.hfh.edu.

Recent reports have documented an association between patent foramen ovale and obstructive sleep apnea. We report on a 51-year-old man with obstructive sleep apnea and recent stroke who was enrolled in a clinic trial evaluating the efficacy of closure of patent foramen ovale following ischemic stroke. He was randomly assigned to device closure. There was subjective dramatic improvement in sleep-apnea symptoms and objective improvement in polysomnographic testing after device implantation. Aside from a drop in apneas and hypopneas from 181 and 8 on the first polysomnogram to 19 and 0 on the second, there was no significant weight loss nor were there other significant changes in sleep parameters or medications. He stopped using continuous positive airway pressure 2 months after implantation and has had no recurrent

sleep complaints during 18 months of follow-up. Further studies evaluating the relationship among patent foramen ovale, sleep apnea, and device implantation are warranted.

Silverman NA. (2007). "A Model of Heterotopic Aortic Valve Replacement: A Critique." J Surg Res. EPub Ahead of Print. [PDF Full-Text](#)

Department of Surgery, Henry Ford Hospital Cardiac and Thoracic Surgery Detroit, Michigan
NSILVER1@hfhs.org.

Szpunar SM, Williams PD, Dagroso D, Enberg RN and Chesney JD. (2007). "An assessment of user acceptance and satisfaction with the tobacco use cessation automated clinical practice guideline." Am J Manag Care **13**(6 Part 1): 313-5. [PDF Full-Text](#)

Henry Ford Health System, Detroit, MI, USA. sszpunal@stjohn.org.

OBJECTIVE: To describe user acceptance of and satisfaction with the Tobacco Use Cessation (TUC) Automated Clinical Practice Guideline (ACPG) at the Henry Ford Health System. STUDY DESIGN: A previous investigation assessed compliance with the 5 As (ask, advise, assess, assist, and arrange) of the TUC ACPG across 3 study arms. This article describes user satisfaction with the TUC ACPG after implementation. METHODS: In all study arms, providers completed a survey before participating in a focus group. RESULTS: All providers in the TUC arm indicated that they "almost always" asked their patients about tobacco use. Providers in the TUC arm were generally satisfied with the features of the TUC ACPG, particularly the ease of electronically referring a patient to the Smoking Intervention Program. Barriers to use included time constraints, lack of staff, and the desire to "opt out" of the program for patients in specific situations (eg, patients with terminal illnesses). CONCLUSION: Because ACPGs are incorporated into electronic medical records, it is important to obtain provider input before implementation, to supply technology that is user friendly and fits into the work flow of the clinic, and to afford physicians the autonomy to opt out of the guideline in specific clinical circumstances.

Tunceli K, Bradley CJ, Lafata JE, Pladevall M, Divine GW, Goodman AC and Vijan S. (2007). "Glycemic control and absenteeism among individuals with diabetes." Diabetes Care **30**(5): 1283-5. [PDF Full-Text](#)

Center for Health Services Research, Henry Ford Health System, Detroit, Michigan, USA.
ktuncel1@hfhs.org.

Van Dyke DL, Ebrahim SA, Al Saadi AA, Powell SA, Zenger-Hain JL, Micale MA, Wiktor AE and Zou YS. (2007). "The impact of maternal serum screening programs for Down syndrome in southeast Michigan, 1988-2003." Prenat Diagn **27**(6): 583-4. [PDF Full-Text](#)

Henry Ford Health System, Detroit, MI, USA.

van Holsbeeck MT. (2007). "A role for US screening in juvenile idiopathic arthritis." Pediatr Radiol **37**(7): 623-4. [PDF Full-Text](#)

Department of Radiology, Musculoskeletal Radiology, Henry Ford Hospital, 2799 W. Grand Blvd, Detroit, MI, 48202, USA, vanholsbeeck@comcast.net.

Williams TR, Longoria OJ, Asselmeier S and Menon M. (2007). "Incidence and imaging appearance of urethrovesical anastomotic urinary leaks following da Vinci robotic prostatectomy." *Abdom Imaging*. EPub Ahead of Print. [PDF Full-Text](#)

Department of Radiology, Abdominal Imaging Division, Henry Ford Hospital, 2799 West Grand Blvd, Detroit, MI, 48202, USA, toddw@rad.hfh.edu.

BACKGROUND: The advent of the da Vinci robotic prostatectomy has several advantages over open and laparoscopic prostatectomy, including fewer complications, better continence and potency. We evaluate the incidence and imaging features of urinary leaks after robotic prostatectomy. **METHODS:** A retrospective study examining the anastomotic leak rates from 490 consecutive robotic prostatectomy patients. Routine postoperative cystography on day 7 was reviewed for presence and severity of urinary anastomotic leaks. **RESULTS:** A total of 490 patients were reviewed, of which 442 had cystographic imaging postoperatively (n = 442). A total of 67 urinary leaks were identified; 40 were small, limited extraperitoneal leaks confined to the surgical bed, 21 were moderate sized leaks limited to the extraperitoneal pelvic space, and six extended in to the peritoneal cavity. Two of these six patients required CT-guided drainage for peritoneal urinoma. Other cystography findings included two cases of vesicoureteral reflux and one case of colovesical fistula. **CONCLUSION:** The incidence of postoperative anastomotic urinary leaks following robotic prostatectomy (13.6%) is the same or better than laparoscopic prostatectomy and traditional radical retropubic prostatectomy. The vast majority of urethrovesical leaks are transient, requiring no follow-up intervention. The incidence of large anastomotic leaks requiring CT guided intervention is exceedingly low 2/490 (<0.5%).

Yang H, Chopp M, Zhang X, Jiang F, Zhang Z, Kalkanis S and Schallert T. (2007). "Using behavioral measurement to assess tumor progression and functional outcome after antiangiogenic treatment in mouse glioma models." *Behav Brain Res*. EPub Ahead of Print. **Full-Text Not Available / [Click for Article Request Form](#)**

Institute for Neuroscience, University of Texas at Austin, Austin, TX 78712, United States;
Department of Psychology, University of Texas at Austin, Austin, TX 78712, United States;
Department of Neurology, Henry Ford Health Sciences Center, Detroit, MI 48202, United States.

The objective of the current study was to investigate the behavioral changes of glioma-bearing nude mice and functional outcome from treatment with a novel antiangiogenesis regimen, which is a combination of monoclonal antibodies against both vascular endothelial growth factor receptor (VEGFR)-1 (MF1) and VEGFR-2 (DC101). The reliability and responsiveness of behavioral measurement with the rearing test were first examined in nude mice bearing two kinds of gliomas-9L gliosarcoma and U87 human glioma, which have different growth rates. Using immunohistochemical staining and fluorescent imaging techniques, upregulation of the angiogenesis marker VEGF, coincident with the abnormal neovascular architecture, was confirmed in the human U87 glioma model. The behavioral measurement was then applied to assess functional outcome with the combination antibody treatment in the orthotopic mouse model of human U87 glioma. The combination antibody therapy retarded tumor progression and delayed the onset of significant behavioral deficits. Histologically, tumor necrosis and apoptosis were increased and tumor cell proliferation was decreased after treatment. In clinical trials for novel interventions, functional end points typically are included in the assessment of potential efficacy. Because certain interventions that successfully treat tumor progression in animal models might interfere with compensatory neuroplasticity, functional measurement may be valuable for improving the clinical relevance of translational brain tumor research.

Zheng X, Jiang F, Katakowski M, Kalkanis SN, Hong X, Zhang X, Zhang ZG, Yang H and Chopp M. (2007). "Inhibition of ADAM17 reduces hypoxia-induced brain tumor cell invasiveness." *Cancer Sci* **98**(5): 674-84. [PDF Full-Text](#)

Department of Neurology, Henry Ford Health Sciences Center, Detroit, Michigan 48202, USA.

The membrane-anchored metalloproteinase tumor necrosis factor-alpha-converting enzyme (TACE/a disintegrin and metalloproteinase [ADAM] 17) is key in proteolytic ectodomain shedding of several membrane-bound growth factors, cytokines and receptors. The expression and activity of ADAM17 increases under some pathological conditions including stroke, and promotes neural progenitor cell migration and contributes to stroke-induced neurogenesis. Hypoxia initiates cellular invasive processes that occur under both physiological and pathological conditions such as invasion and metastasis of some tumors. In the present study, we sought to elucidate whether ADAM17 contributes to brain tumor invasion. To this end, we examined the role of ADAM17 in the invasiveness of two different brain tumor cell lines, 9L rat gliosarcoma and U87 human glioma, under normoxic and hypoxic conditions. Additionally, we tested the effects of ADAM17 suppression on in vitro tumor cell invasion by means of ADAM17 proteolytic inhibitors and specific small interfering RNA. We found that tumor cells upregulated ADAM17 expression under hypoxia, and that ADAM17 activity correlated with increased tumor cell invasion. Conversely, suppression of ADAM17 proteolysis decreased invasiveness induced by hypoxia in 9L and U87 cells. Furthermore, the contribution of ADAM17 to tumor invasion was independent of matrix metalloproteinase (MMP)-2 and MMP-9 activity. ADAM17 was also found to activate the epidermal growth factor/phosphoinositide-3 kinase/serine/threonine kinase signal transduction pathway. Our data suggest that hypoxia-induced ADAM17 contributes to glioma cell invasiveness through activation of the EGFR signal pathway.

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