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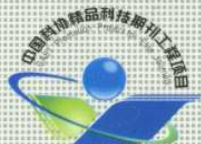
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目次

寄语青年肿瘤医师 裘法祖 561

专题综述

PET-CT 影像引导非小细胞肺癌的放射治疗
..... 李建彬 张英杰 于金明 563

基础研究

纳米载体介导的人端粒酶逆转录酶反义寡核苷酸
对食管癌 EC9706 细胞的增殖抑制作用及端粒酶
表达的影响 王瑾 张振中 周天洋等 566

凋亡抑制蛋白基因在多西紫杉醇耐药胃癌
细胞中的差异表达及其意义 王婷婷 魏嘉 钱晓萍等 573

血管内皮生长因子及其受体双靶向阻断对
膀胱癌细胞生长及血管生成的抑制作用
..... 陈秀玲 刘禄成 许宗革等 578

p21 对肝癌细胞中 survivin 转录的影响及
调控机制的探讨 熊娟 李一荣 汤兆明等 583

白桦脂酸对 Jurkat 白血病细胞增殖和凋亡的
影响 陈子 吴秋玲 陈燕等 588

临床研究

肝细胞癌中 PGC-1 α 基因的表达下调
及其意义 巴一 张春妮 张燕等 593

组织因子途径抑制物 2 基因在胰腺癌中的
表达及对胰腺癌患者预后的影响 汤志刚 孙振阳 胡何节等 598

直肠癌中血管内皮生长因子和细胞增生
核抗原的表达及其与放疗敏感性关系
..... 姜书梅 王仁本 于金明等 602

血管生成素 2 在口腔颌面部鳞癌中的表达
及意义 陈海红 汪审清 吴求亮 606

受体三阴性乳腺癌中基底细胞角蛋白的
表达及其与预后的关系 刘哲斌 吴灵 平波等 610

恶性肿瘤患者血清骨钙素和甲状旁腺素水平与骨转移关系的研究	金成禹 李雨林 张彤等	614
临床应用		
相关蛋白标记物辅助液基薄层细胞检测纤维支气管镜刷片细胞学肺癌 分型诊断的探讨	杨艳 潘秦镜 腾茂芳等	616
选择性出入肝血流阻断在肝脏巨大肿瘤切除术中的应用	胡智明 吴伟顶 张成武等	620
重组人血小板生成素治疗化疗相关血小板减少的临床价值	戴晓芳 喻杰 刘莉等	623
294 例睾丸精原细胞瘤的远期疗效分析	宋岩 杨林 马建辉等	626
150 例早期霍奇金淋巴瘤的综合治疗	牛奕 石远凯 何小慧等	630
癌症论坛		
胃癌内科治疗现状的认识	徐建明	635
病例报告		
索拉非尼治疗晚期双侧肾细胞瘤一例	沈周俊 邵远 芮文斌等	640
新书介绍		
美国国家癌症综合网非霍奇金淋巴瘤治疗指南 2008 年第二版介绍	石远凯	638
读者·作者·编者		
本刊对来稿中统计学处理的有关要求		637
简 讯		
第十五届国际乳腺癌大会暨第三届上海国际乳腺癌论坛通知		577
《中国医学文摘·外科学》杂志更名启事		597
CAMS-MRL 分子肿瘤学研究会即将召开		601
本刊稿约见本卷第 1 期第 79 页和第 7 期第 559 页		
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CONTENTS IN BRIEF

REVIEWS

- PET-CT-guided radiotherapy for non-small cell lung cancer** 563
LI Jian-bin, ZHANG Ying-jie, YU Jin-ming

BASIC RESEARCH

- Effect of nanosize delivery system for ASODN against hTERT on the expression of telomerase in the esophageal cancer EC9706 cells** 566
WANG Jin, ZHANG Zhen-zhong, ZHOU Tian-yang, et al
- Differential gene expression of the inhibitor of apoptosis proteins in docetaxel-resistant gastric cancer cells** 573
WANG Ting-ting, WEI Jia, QIAN Xiao-ping, et al
- Inhibition of bladder cancer cell growth and angiogenesis by co-blockage of vascular endothelial growth factor and its receptor kdr** 578
CHEN Xiu-ling, LIU Lu-cheng, XU Zong-ge, et al
- The effect of p21 on transcription of survivin in hepatocellular carcinoma HepG2 cells and its regulation mechanism** 583
XIONG Juan, LI Yi-rong, TANG Zhao-ming, et al
- Effect of betulinic acid on proliferation and apoptosis in Jurkat cells and its mechanism** 588
CHEN Zi, WU Qiu-ling, CHEN Yan, et al

CLINICAL INVESTIGATIONS

- Down-regulation of PGC-1 α expression in human hepatocellular carcinoma** 593
BA Yi, ZHANG Chun-ni, ZHANG Yan, et al
- Expression of TFPI-2 gene in pancreatic carcinoma and its prognostic significance** 598
TANG Zhi-gang, SUN Zhen-yang, HU He-jie, et al
- Correlation of VEGF and Ki67 expression with sensitivity to neoadjuvant chemoradiation in rectal adenocarcinoma** 602
JIANG Shu-mei, WANG Ren-ben, YU Jin-ming, et al
- Clinical significance of angiopoietin-2 expression in oral squamous cell carcinoma** 606
CHEN Hai-hong, WANG Shen-qing, WU Qiu-liang
- Expression of CK5/6 and CK17 and its correlation with prognosis of triple-negative breast cancer patients** 610
LIU Zhe-bin, WU Jiong, PING Bo, et al
- The correlation between serum osteocalcin and parathyroid hormone levels in cancer patients and bone metastasis** 614
JIN Cheng-yu, LI Yu-lin, ZHANG Tong, et al

CLINICAL APPLICATIONS

- Application of protein markers in combination with ThinPrep bronchial brush cytology in classification of lung cancer subtypes** 616
YANG Yan, PAN Qin-jing, TENG Mao-fang, et al
- Selective exclusion of hepatic outflow and inflow in hepatectomy for huge hepatic tumor** 620
HU Zhi-ming, WU Wei-ding, ZHANG Cheng-wu, et al

Value of recombinant human thrombopoietin in the treatment of chemotherapy-induced thrombocytopenia in patients with solid tumor	623
<i>DAI Xiao-fang, YU Jie, LIU Li, et al</i>	
Long-term outcome of testicular seminoma in 294 patients	626
<i>SONG Yan, YANG Lin, MA Jian-hui, et al</i>	
Combined-modality therapy for 150 cases of early-stage Hodgkin's lymphoma	630
<i>NIU Yi, SHI Yuan-kai, HE Xiao-hui, et al</i>	
CANCER FORUM	
The present understanding of medicinal treatment of stomach cancer	635
<i>XU Jian-ming</i>	

Research News

Stromal contribution to cancer malignancy

Recently, there has been increasing interest in studying the impact of tumor stroma on the initiation and progression of cancer. For instance, Finak et al. (Nature Med 14, 518-527, 2008) demonstrated a cluster of 26 genes stratify the risk of breast cancer progression using molecular markers that are independent of, but add power to, standard clinical prognosis factors. Postovit et al. (PNAS 105, 4329-4334, 2008) took a special approach to study the stromal contribution to cancer malignancy. They used an in vitro three-dimensional model that exposes cancer cells to the microenvironment to which human embryonic stem cells are commonly exposed, trying to identify conditions in the stroma that suppress malignant characteristics of cancer cells. Stromal cells surrounding embryonic stem cells secrete a protein called Lefty, which inhibits the Nodal protein. Nodal, which during embryonic development prevents stem-cell differentiation, is abnormally expressed in human tumor cells, causing malignancy. Postovit et al. found that metastatic tumor cells do not express Lefty. These results strongly support stromal regulation of malignancy and indicate that Lefty has a suppressive effect on cancer cells. More interesting is the work by Hu et al. (Cancer Cell 13, 394-406, 2008) that reveals how carcinogenesis is initiated. They used a cell line, when injected into mice, developed cancer that mimics human breast ductal carcinoma in situ (DCIS). With this human tumor model, they studied the role of myoepithelial cells (the cells that separate the basement membrane of the duct from the epithelial cells) in these lesions in suppressing the transition of DCIS to invasive neoplasia. By functional analysis of cell-specific gene expression, they identified several pathways that could be essential for interactions between stromal fibroblasts and myoepithelial cells in controlling integrity of basement membrane. These pathways are mediated by essential signaling molecules such as TGF- β , Hedgehog, cell adhesion molecules and gene transcription factor p63. Myoepithelial cells secrete maspin, a tumor suppressor protein, by inhibiting degradation of the extracellular matrix. Malfunction of these signaling pathways leads to loss of myoepithelial cells and subsequent invasion of basement by their adjoining epithelial cells. Since only a small fraction of patients with DCIS will develop invasive disease, identification of the factors associated with subsequent invasive events could help avoid over-or under-treatment.

(Whispering sweet somethings by Tlsty T, abridged, Nature, 2008, 453:604-605)