

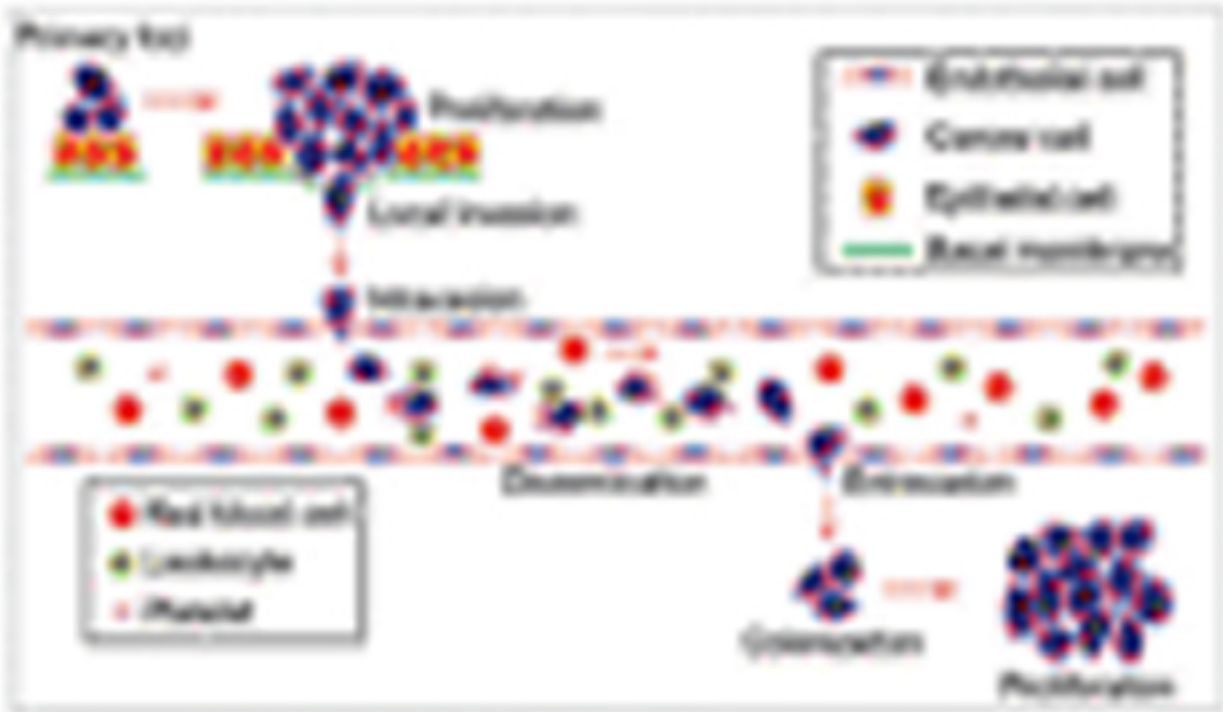
# Oncology Letters



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# Journal Articles

Open Access

Genetic alterations and epigenetic alterations of cancer-associated fibroblasts (Review)

Heng Du, Guowei Che

Pages: 3-12 Published online on: 30 November 2016

C-type lectins facilitate tumor metastasis (Review)

Dongbing Ding, Yao Yao, Songbai Zhang, Chunjie Su, Yonglian Zhang

Pages: 13-21 Published online on: 24 November 2016

Role of free testosterone levels in patients with metastatic castration-resistant prostate cancer receiving second-line therapy

Christoph A. von Klot, Markus A. Kuczyk, Alena Boeker, Christoph Reuter, Florian Imkamp, Thomas R.W. Herrmann, Hossein Tezval, Mario W. Kramer, Sven Perner, Axel S. Merseburger

Pages: 22-28 Published online on: 17 November 2016

Efficacy of computed tomography features in predicting stage III thymic tumors

Yan Shen, Jianding Ye, Wentao Fang, Yu Zhang, Xiaodan Ye, Yonghong Ma, Libo Chen, Minghua Li

Pages: 29-36 Published online on: 23 November 2016

Role of gambogic acid and Na131 in A549/DDP cells

Jing Huang, Xiaoli Zhu, Huan Wang, Shuhua Han, Lu Liu, Yan Xie, Daozhen Chen, Qiang Zhang, Li Zhang, Yue Hu

Pages: 37-44 Published online on: 25 November 2016

Enhanced expression of hydroxylated ceramide in well-differentiated endometrial adenocarcinoma

Toshiki Tajima, Masaki Miyazawa, Masaru Hayashi, Satoshi Asai, Masae Ikeda, Masako Shida, Takeshi Hirasawa, Masao Iwamori, Mikio Mikami

Pages: 45-50 Published online on: 22 November 2016

CRKL overexpression promotes cell proliferation and inhibits apoptosis in endometrial carcinoma

Le Cai, He Wang, Qing Yang

Pages: 51-56 Published online on: 17 November 2016

Effects of 17 $\beta$ -estradiol and tamoxifen on gastric cancer cell proliferation and apoptosis and ER- $\alpha$ 36 expression

Xuming Wang, Qiuyue Chen, Xuan Huang, Feng Zou, Zhengqi Fu, Ying Chen, Yan Li, Zhaoyi Wang, Lijiang Liu

Pages: 57-62 Published online on: 23 November 2016

Different but synergistic effects of bone marrow-derived VEGFR2+ and VEGFR2-CD45+ cells during hepatocellular carcinoma progression

Xiaolin Zhu, Hongyuan Zhou, Jingtao Luo, Yunlong Cui, Huikai Li, Wei Zhang, Feng Fang, Qiang Li, Ti Zhang

Pages: 63-68 Published online on: 22 November 2016

Therapeutic response to a novel enzyme-targeting radiosensitization treatment (KORTUC II) for residual lesions in patients with stage IV primary breast cancer, following induction chemotherapy with epirubicin and cyclophosphamide or taxane

Nobutaka Aoyama, Yasuhiro Ogawa, Miki Yasuoka, Hitomi Iwasa, Kana Miyatake, Rika Yoshimatsu, Tomoaki Yamanishi, Norihiko Hamada, Taiji Tamura, Kana Kobayashi, Yoriko Murata, Takuji Yamagami, Mitsuhiro Miyamura

Pages: 69-76 Published online on: 01 December 2016

Correlation between liver cancer pain and the HIF-1 and VEGF expression levels

Geng Zhang, Gui-Yin Feng, Yan-Ru Guo, Dong-Qi Liang, Yuan Yuan, Hai-Lun Wang

Pages: 77-80 Published online on: 21 November 2016

miR-433 reduces cell viability and promotes cell apoptosis by regulating MACC1 in colorectal cancer

Jiaxin Li, Xuping Mao, Xing Wang, Ganggang Miao

Pages: 81-88 Published online on: 30 November 2016

Effect of miR-23a on anoxia-induced phenotypic transformation of smooth muscle cells of rat pulmonary arteries and regulatory mechanism

Li Yan, Haixiang Gao, Chunzhi Li, Xiaowen Han, Xiaoyong Qi

Pages: 89-98 Published online on: 28 November 2016

Effects of anti-CD44 monoclonal antibody IM7 carried with chitosan polylactic acid-coated nano-particles on the treatment of ovarian cancer

Yizhuo Yang, Xinghui Zhao, Xiuli Li, Zhifeng Yan, Zhongyu Liu, Yali Li

Pages: 99-104 Published online on: 22 November 2016

Profiling of microRNAs in AML cells following overexpression or silencing of the VEGF gene

Li Li, Lixia Zhu, Yungui Wang, De Zhou, Jingjing Zhu, Wanzhuo Xie, Xiujin Ye

Pages: 105-110 Published online on: 22 November 2016

Effects of the overexpression of IFITM5 and IFITM5 c.-14C>T mutation on human osteosarcoma cells

Bao-Yan Liu, Yan-Qin Lu, Feng Han, Yong Wang, Xin-Kai Mo, Jin-Xiang Han

Pages: 111-118 Published online on: 23 November 2016

Goniothalamin induces mitochondria-mediated apoptosis associated with endoplasmic reticulum stress-induced activation of JNK in HeLa cells

Thanet Sophonnithiprasert, Wilawan Mahabusarakam, Yukio Nakamura, Ramida Watanapakasin

Pages: 119-128 Published online on: 11 November 2016

Combined detection of the expression of Nm23-H1 and p53 is correlated with survival rates of patients with stage II and III colorectal cancer

Yinying Wu, Yi Li, Xiaoi Zhao, Danfeng Dong, Chunhui Tang, Enxiao Li, Qianqian Geng

Pages: 129-136 Published online on: 23 November 2016

Clinical application of transanal ileal tube placement using X-ray monitoring

Dechun Li, Hongtao Du, Guoqing Shao, Yuanshun Xu, Ruihong Li, Qingzhong Tian

Pages: 137-140 Published online on: 29 November 2016

Expression and clinical significance of PcG-associated protein RYBP in hepatocellular carcinoma

Xiaonian Zhu, Meng Yan, Wei Luo, Wei Liu, Yuan Ren, Chunhua Bei, Guifang Tang, Ruiling Chen, Shengkui Tan

Pages: 141-150 Published online on: 11 November 2016

The effect of miR-146a on STAT1 expression and apoptosis in acute lymphoblastic leukemia Jurkat cells

Weihong Yan, Hua Guo, Feng Suo, Chunling Han, Hua Zheng, Tong Chen

Pages: 151-154 Published online on: 18 November 2016

Evaluation of minimally invasive laser ablation in children with osteoid osteoma

Hao Wu, Cheng Lu, Ming Chen

Pages: 155-158 Published online on: 22 November 2016

Differential distribution of tumor-associated macrophages and Treg/Th17 cells in the progression of malignant and benign epithelial ovarian tumors

Qinyi Zhu, Xiaoli Wu, Xipeng Wang

Pages: 159-166 Published online on: 23 November 2016

Bioinformatics analysis of key genes and latent pathway interactions based on the anaplastic thyroid carcinoma gene expression profile

Yun Huang, Yiming Tao, Xinying Li, Shi Chang, Bo Jiang, Feng Li, Zhi-Ming Wang

Pages: 167-176 Published online on: 30 November 2016

'Obligate' anaerobic Salmonella strain YB1 suppresses liver tumor growth and metastasis in nude mice

Chang-Xian Li, Bin Yu, Lei Shi, Wei Geng, Qiu-Bin Lin, Chang-Chun Ling, Mei Yang, Kevin T. P. Ng, Jian-Dong Huang, Kwan Man

Pages: 177-183 Published online on: 30 November 2016

Bilateral ovarian carcinomas differ in the expression of metastasis-related genes

Marianne Lislørud Smebye, Lisbeth Haugom, Ben Davidson, Claes Göran Tropé, Sverre Heim, Rolf Inge Skotheim, Francesca Micci

Pages: 184-190 Published online on: 14 November 2016

Sometimes it is better to wait: First Italian case of a newborn with transient abnormal myelopoiesis and a favorable prognosis

Guglielmo Salvatori, Silvia Foligno, Pietro Sirleto, Silvia Genovese, Serena Russo, Valentina Coletti, Andrea Dotta, Matteo Luciani

Pages: 191-195 Published online on: 21 November 2016

Differential effects of recombinant human endostatin treatment on differentiated and undifferentiated blood vessels in Lewis lung cancer

Weijiang Fu, Jing Zhuo, Likuan Hu

Pages: 196-200 Published online on: 30 November 2016

Influence of VEGFR single nucleotide polymorphisms on the efficacy of sunitinib therapy against renal cell carcinoma  
Rui Liu, Xiaojie Wang, Wei Li, Tao Shou, Likun Zhou, Yunfen Li, Ming Bai, Qiang Pei  
Pages: 201-205 Published online on: 18 November 2016

Increased chemosensitivity and radiosensitivity of human breast cancer cell lines treated with novel functionalized single-walled carbon nanotubes  
Yijun Jia, Ziyi Weng, Chuanying Wang, Mingjie Zhu, Yunshu Lu, Longlong Ding, Yongkun Wang, Xianhua Cheng, Qing Lin, Kejin Wu  
Pages: 206-214 Published online on: 21 November 2016

Molecular heterogeneity in the novel fusion gene AP1F-FGFR2: Diversity of genomic breakpoints in gastric cancer with high-level amplifications at 11p13 and 10q26  
Takashi Okuda, Tomohiko Taki, Kazuhiro Nishida, Yoshiaki Chinen, Hisao Nagoshi, Chouhei Sakakura, Masafumi Taniwaki  
Pages: 215-221 Published online on: 15 November 2016

Increased expression of S100A6 promotes cell proliferation in gastric cancer cells  
Xiao-Hong Wang, Hong Du, Lin Li, Duan-Fang Shao, Xi-Yao Zhong, Ying Hu, Yi-Qiang Liu, Xiao-Fang Xing, Xiao-Jing Cheng, Ting Guo, Shen Li, Zi-Yu Li, Zhao-De Bu, Xian-Zi Wen, Lian-Hai Zhang, Jia-Fu Ji  
Pages: 222-230 Published online on: 22 November 2016

Comparison of the breast and areola approaches for endoscopic thyroidectomy in patients with microcarcinoma  
Gaolei Jia, Zhilong Tian, Hailin Xi, Su Feng, Xiaokai Wang, Xinbao Gao  
Pages: 231-235 Published online on: 28 November 2016

CEP55 overexpression predicts poor prognosis in patients with locally advanced esophageal squamous cell carcinoma  
Wenpeng Jiang, Zhou Wang, Yang Jia  
Pages: 236-242 Published online on: 22 November 2016

DHA blocks TPA-induced cell invasion by inhibiting MMP-9 expression via suppression of the PPAR- $\gamma$ /NF- $\kappa$ B pathway in MCF-7 cells  
Jin-Ki Hwang, Hong-Nu Yu, Eun-Mi Noh, Jeong-Mi Kim, On-Yu Hong, Hyun Jo Youn, Sung Hoo Jung, Kang-Beom Kwon, Jong-Suk Kim, Young-Rae Lee  
Pages: 243-249 Published online on: 11 November 2016

Factors related to endocrine changes and hormone substitution treatment during pre- and post-operation stages in craniopharyngioma  
Fenglei Sun, Xintang Sun, Xiaolong Du, Hongshun Xing, Bin Yang  
Pages: 250-252 Published online on: 22 November 2016

Significance of clearing differentiated thyroid carcinoma lymph node by high-frequency color Doppler ultrasonography  
Bing Liu, Huadong Qin, Bin Zhang, Tiefeng Shi, Chuanle Li, Yao Liu, Meiyue Song  
Pages: 253-257 Published online on: 30 November 2016

Cell death in a co-culture of hepatocellular carcinoma cells and human umbilical vascular endothelial cells in a medium lacking glucose and arginine  
Minoru Tomizawa, Fuminobu Shinozaki, Yasufumi Motoyoshi, Takao Sugiyama, Shigenori Yamamoto, Naoki Ishige  
Pages: 258-262 Published online on: 30 November 2016

The diagnostic utility of PAX8 immunostaining of malignant peritoneal mesothelioma presenting as serous ovarian carcinoma: A single-center report of two cases  
Kohei Nakamura, Kentaro Nakayama, Risa Nagaoka, Kiyoka Nishisako, Masako Ishikawa, Hiroshi Katagiri, Tomoka Ishibashi, Emi Sato, Chika Amano, Satoru Kyo  
Pages: 263-266 Published online on: 30 November 2016

Multiple pulmonary emboli as a result of renal cell carcinoma: A case report  
Bing Li, Hong Zeng, Mei Ding, Ping Yang, Yuquan He  
Pages: 267-270 Published online on: 21 November 2016

Recognition of tumor antigens in 4T1 cells by natural IgM from three strains of mice with different susceptibilities to spontaneous breast cancer  
Mariana Diaz-Zaragoza, Ricardo Hernández-Ávila, Pedro Ostoa-Saloma  
Pages: 271-274 Published online on: 23 November 2016

Pathological significance and prognostic implications of heme oxygenase 1 expression in non-muscle-invasive bladder cancer: Correlation with cell proliferation, angiogenesis, lymphangiogenesis and expression of VEGFs and COX-2  
Tomohiro Matsuo, Yasuyoshi Miyata, Kensuke Mitsunari, Takuji Yasuda, Kojiro Ohba, Hideki Sakai  
Pages: 275-280 Published online on: 22 November 2016

Circulating tumor cells expressing cancer stem cell marker CD44 as a diagnostic biomarker in patients with gastric cancer  
Toru Watanabe, Tomoyuki Okumura, Katsuhisa Hirano, Tetsuji Yamaguchi, Shinichi Sekine, Takuya Nagata, Kazuhiro Tsukada  
Pages: 281-288 Published online on: 24 November 2016

Comparative proteomic analysis of paclitaxel resistance-related proteins in human breast cancer cell lines  
Hiroya Fujioka, Akiko Sakai, Satoru Tanaka, Kosei Kimura, Akiko Miyamoto, Mitsuhiko Iwamoto, Kazuhisa Uchiyama  
Pages: 289-295 Published online on: 30 November 2016

Neuroprotective effect of matrine on MPTP-induced Parkinson's disease and on Nrf2 expression  
Fanhua Meng, Jianhui Wang, Fuxiang Ding, Yunliang Xie, Yingjie Zhang, Jie Zhu  
Pages: 296-300 Published online on: 14 November 2016

Cervical intraepithelial neoplasia in pregnancy: Interference of pregnancy status with p16 and Ki-67 protein expression  
Andrea Ciavattini, Francesco Sopracordevole, Jacopo Di Giuseppe, Lorenzo Moriconi, Guendalina Lucarini, Francesca Manciola, Antonio Zizzi, Gaia Goteri  
Pages: 301-306 Published online on: 29 November 2016

Adenoviral diploxil-induced Fanconi syndrome and its predictive factors: A study of 28 cases  
Yong Lin, Fan Pan, Yingchao Wang, Ziqian Chen, Chun Lin, Lvfang Yao, Xin Zhang, Rui Zhou, Chen Pan  
Pages: 307-314 Published online on: 17 November 2016

A methodological procedure for evaluating the impact of hemolysis on circulating microRNAs  
Sara Pizzamiglio, Susanna Zanutto, Chiara M. Ciniselli, Antonino Belfiore, Stefano Bottelli, Manuela Gariboldi, Paolo Verderio  
Pages: 315-320 Published online on: 30 November 2016

ALDH1 and podoplanin expression patterns predict the risk of malignant transformation in oral leukoplakia  
Umma Habiba, Kyoko Hida, Tetsuya Kitamura, Aya Yanagawa Matsuda, Fumihiro Higashino, Yoichi M. Ito, Yoichi Ohno, Yasunori Totsuka, Masanobu Shindoh  
Pages: 321-328 Published online on: 10 November 2016

MicroRNA-101 suppresses progression of lung cancer through the PTEN/AKT signaling pathway by targeting DNA methyltransferase 3A  
Lumin Wang, Jiayi Yao, Hongfei Sun, Kang He, Dongdong Tong, Tusheng Song, Chen Huang  
Pages: 329-338 Published online on: 23 November 2016

Lymphangiomatosis of the sigmoid colon - a rare cause of lower gastrointestinal bleeding: A case report and review of the literature  
Guifang Lu, Hongxia Li, Yuan Yuan Li  
Pages: 339-341 Published online on: 21 November 2016

Tumor necrosis factor receptor 2 promotes growth of colorectal cancer via the PI3K/AKT signaling pathway  
Tao Zhao, Huihui Li, Zifeng Liu  
Pages: 342-346 Published online on: 21 November 2016

Resveratrol mediates cell cycle arrest and cell death in human esophageal squamous cell carcinoma by directly targeting the EGFR signaling pathway  
Zixuan Jin, Wei Feng, Ying Ji, Longyu Jin  
Pages: 347-355 Published online on: 17 November 2016

Inhibitory effect of dexamethasone on residual Lewis lung cancer cells in mice following palliative surgery  
Ningbo Sun, Huaijun Ji, Wei Wang, Qiang Zhu, Ming Cao, Qi Zang  
Pages: 356-362 Published online on: 23 November 2016

H19 promotes endometrial cancer progression by modulating epithelial-mesenchymal transition  
Le Zhao, Zhen Li, Wei Chen, Wen Zhai, Jingjing Pan, Huan Pang, Xu Li  
Pages: 363-369 Published online on: 16 November 2016

Anticancer activity of sesquiterpenoids extracted from Solanum lyratum via the induction of mitochondria-mediated apoptosis  
Min Chen, Jian Wu, Xing-Xing Zhang, Qiong Wang, Shi-Hai Yan, Hai-Dan Wang, Sheng-Lin Liu, Xi Zou  
Pages: 370-376 Published online on: 21 November 2016

Role of Annexin A2 in the EGF-induced epithelial-mesenchymal transition in human CaSki cells  
Lei Cui, Jian Song, Litong Wu, Luhui Cheng, Aijun Chen, Yanlin Wang, Yingdi Huang, Liming Huang  
Pages: 377-383 Published online on: 21 November 2016

Gambogic acid inhibits the growth of ovarian cancer tumors by regulating p65 activity

Qiusha Tang, Mudan Lu, Huan Zhou, Daozhen Chen, Lu Liu

Pages: 384-388 Published online on: 24 November 2016

Solid lipid nanoparticles with TPGS and Brij 78: A co-delivery vehicle of curcumin and piperine for reversing P-glycoprotein-mediated multidrug resistance in vitro

Jingling Tang, Hongyu Ji, Jinmei Ren, Mengting Li, Nannan Zheng, Linhua Wu

Pages: 389-395 Published online on: 23 November 2016

Transcription factor Oct4 promotes osteosarcoma by regulating lncRNA Ak055347

Hongwu Fan, Guangyao Liu, Changfu Zhao, Xuefeng Li, Xiaoyu Yang

Pages: 396-402 Published online on: 21 November 2016

HDAC2 regulates cell proliferation, cell cycle progression and cell apoptosis in esophageal squamous cell carcinoma EC9706 cells

Shenglei Li, Feng Wang, Yunhui Qu, Xiaoqi Chen, Ming Gao, Jianping Yang, Dandan Zhang, Na Zhang, Wencai Li, Hongtao Liu

Pages: 403-409 Published online on: 25 November 2016

Anti-tumor and anti-invasion effects of a combination of 4-methylumbelliferone and ionizing radiation in human fibrosarcoma cells

Ryo Saga, Satoru Monzen, Mitsuru Chiba, Hironori Yoshino, Toshiya Nakamura, Yoichiro Hosokawa

Pages: 410-416 Published online on: 15 November 2016

miR-506 suppresses neuroblastoma metastasis by targeting ROCK1

Dianguo Li, Yanhua Cao, Jinliang Li, Jialong Xu, Qian Liu, Xiaogang Sun

Pages: 417-422 Published online on: 29 November 2016

Per2 participates in AKT-mediated drug resistance in A549/DDP lung adenocarcinoma cells

Bo Chen, Yaoxi Tan, Yan Liang, Yan Li, Lei Chen, Shuangshuang Wu, Wei Xu, Yan Wang, Weihong Zhao, Jianqing Wu

Pages: 423-428 Published online on: 24 November 2016

Percutaneous microwave ablation for benign focal liver lesions: Initial clinical results

Zhigang Cheng, Ping Liang, Xiaoling Yu, Zhiyu Han, Fangyi Liu, Jie Yu, Xin Li

Pages: 429-434 Published online on: 22 November 2016

miR-143 inhibits bladder cancer cell proliferation and enhances their sensitivity to gemcitabine by repressing IGF-1R signaling

Hengbing Wang, Qi Li, Xiaobing Niu, Gongcheng Wang, Sinian Zheng, Guangbo Fu, Zengjun Wang

Pages: 435-440 Published online on: 16 November 2016

Effects of physical activity on systemic oxidative/DNA status in breast cancer survivors

Barbara Tomasello, Giuseppe Antonio Malfa, Angela Strazzanti, Santi Gangi, Claudia Di Giacomo, Francesco Basile, Marcella Renis

Pages: 441-448 Published online on: 30 November 2016

Protective effects of sodium selenite supplementation against irradiation-induced damage in non-cancerous human esophageal cells

Irma M. Puspitasari, Chiho Yamazaki, Rizky Abdulah, Mirasari Putri, Satomi Kameo, Takashi Nakano, Hiroshi Koyama

Pages: 449-454 Published online on: 24 November 2016

ETS-related gene is a novel prognostic factor in childhood acute lymphoblastic leukemia

Hai-Zhao Zhao, Ming Jia, Ze-Bin Luo, Xiao-Jun Xu, Si-Si Li, Jing-Ying Zhang, Xiao-Ping Guo, Yong-Min Tang

Pages: 455-462 Published online on: 18 November 2016

Association of leptin, visfatin, apelin, resistin and adiponectin with clear cell renal cell carcinoma

Hai-Ping Zhang, Jian Zou, Zhuo-Qun Xu, Jun Ruan, Shu-Dong Yang, Ying Yin, Hui-Jun Mu

Pages: 463-468 Published online on: 22 November 2016

Cigarette smoke extract-induced proliferation of normal human urothelial cells via the MAPK/AP-1 pathway

Hao Geng, Li Zhao, Zhaofeng Liang, Zhiqiang Zhang, Dongdong Xie, Liangkuan Bi, Yi Wang, Tao Zhang, Lei Cheng, Dexin Yu, Caiyun Zhong

Pages: 469-475 Published online on: 22 November 2016

Host knockout of E-prostanoid 2 receptors reduces tumor growth and causes major alterations of gene expression in prostaglandin E2-producing tumors

Annika Gustafsson Asting, Britt-Marie Iresjö, Camilla Nilsberth, Ulrika Smedh, Kent Lundholm

Pages: 476-482 Published online on: 30 November 2016

Downregulation of caveolin-1 increases the sensitivity of drug-resistant colorectal cancer HCT116 cells to 5-fluorouracil

Zhaoyang Li, Ning Wang, Changxin Huang, Yanhong Bao, Yiqian Jiang, Guiting Zhu

Pages: 483-487 Published online on: 16 November 2016

Analysis of molecular markers as predictive factors of lymph node involvement in breast carcinoma

Luciana Marques Paula, Luis Henrique Ferreira de Moraes, Abaeté Leite do Canto, Laurita dos Santos, Ailton Abrahão Martin, Silvia Regina Rogatto, Renata de Azevedo Canevari

Pages: 488-496 Published online on: 28 November 2016

Y-box-binding protein 1 contributes to IL-7-mediated survival signaling in B-cell precursor acute lymphoblastic leukemia

Amina Kariminia, Sabine M. Ivison, Vivian M. Leung, Susanna Sung, Nicole Couto, Jacob Rozmus, Nina Rolf, Aru Narendran, Sandra E. Dunn, Gregor S.D. Reid, Kirk R. Schultz

Prognostic significance of Notch ligands in patients with non-small cell lung cancer

Joanna Pancewicz-Wojtkiewicz, Andrzej Eljaszewicz, Oksana Kowalczyk, Wiesława Niklinska, Radosław Charkiewicz, Mirosław Kozłowski, Agnieszka Miasko, Marcin Moniuszko

Pages: 506-510 Published online on: 23 November 2016

Bovine lactoferricin P13 triggers ROS-mediated caspase-dependent apoptosis in SMMC7721 cells

Lixiang Meng, Geliang Xu, Jiansheng Li, Wenbin Liu, Weidong Jia, Jinliang Ma, Decheng Wei

Pages: 511-517 Published online on: 22 November 2016

Isoimperatorin induces apoptosis of the SGC-7901 human gastric cancer cell line via the mitochondria-mediated pathway

Kehui Tong, Chang Xin, Wenzhong Chen

Pages: 518-524 Published online on: 15 November 2016



# Protective effects of sodium selenite supplementation against irradiation-induced damage in non-cancerous human esophageal cells

IRMA M. PUSPITASARI<sup>1,2</sup>, CHIHO YAMAZAKI<sup>1</sup>, RIZKY ABDULAH<sup>2</sup>, MIRASARI PUTRI<sup>1</sup>, SATOMI KAMEO<sup>1</sup>, TAKASHI NAKANO<sup>3</sup> and HIROSHI KOYAMA<sup>1</sup>

<sup>1</sup>Department of Public Health, Gunma University Graduate School of Medicine, Maebashi, Gunma 371-8511, Japan; <sup>2</sup>Department of Pharmacology and Clinical Pharmacy, Faculty of Pharmacy, Universitas Padjadjaran, Bandung, West Java 45363, Indonesia; <sup>3</sup>Department of Radiation Oncology, Gunma University Graduate School of Medicine, Maebashi, Gunma 371-8511, Japan

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**Abstract.** The administration of radioprotective compounds is one approach to preventing radiation damage in non-cancerous tissues. Therefore, radioprotective compounds are crucial in clinical radiotherapy. Selenium is a radioprotective compound that has been used in previous clinical studies of radiotherapy. However, evidence regarding the effectiveness of selenium in radiotherapy and the mechanisms underlying the selenium-induced reduction of the side effects of radiotherapy remains insufficient. To further investigate the effectiveness of selenium in radiotherapy, the present study examined the protective effects of sodium selenite supplementation administered prior to X-ray radiation treatment in CHEK-1 non-cancerous human esophageal cells. Sodium selenite supplementation increased glutathione peroxidase 1 (GPx-1) activity in a dose- and time-dependent manner. The sodium selenite dose that induced the highest GPx-1 activity was determined to be 50 nM for 72 h prior to radiotherapy. The half-maximal inhibitory concentration of sodium selenite in CHEK-1 cells was 3.6  $\mu$ M. Sodium selenite supplementation increased the survival rate of the cells in a dose-dependent manner and enhanced the degree of cell viability at 72 h post-irradiation ( $P<0.05$ ). Combined treatment with 50 nM sodium selenite and 2 gray (Gy) X-ray irradiation decreased the number of sub-G<sub>1</sub> cells from 5.9 to 4.2% ( $P<0.05$ ) and increased the proportion of G<sub>1</sub> cells from 58.8 to 62.1%, compared with 2 Gy X-ray irradiation alone; however, this difference was not statistically significant

( $P=1.00$ ). Western blot analysis revealed that treatment with 2 Gy X-ray irradiation significantly increased the expression levels of cleaved poly (ADP-ribose) polymerase (PARP;  $P<0.05$ ). In addition, combined treatment with 50 nM sodium selenite and 2 Gy X-ray irradiation reduced the expression levels of cleaved PARP protein, compared with 2 Gy X-ray irradiation alone; however, this reduction was not statistically significant ( $P=0.423$ ). These results suggest that 50 nM sodium selenite supplementation administered for 72 h prior to irradiation may protect CHEK-1 cells from irradiation-induced damage by inhibiting irradiation-induced apoptosis. Therefore, sodium selenite is a potential radioprotective compound for non-cancerous cells in clinical radiotherapy.

## Introduction

Radiotherapy is one of the most common and effective treatments for cancer (1). Over 40% of patients with cancer require radiotherapy during the management of the disease (2). Although clinical radiotherapy treatment planning and delivery technologies have improved, the toxicity of radiotherapy to non-cancerous tissues and organs remains a problem (2,3). Thus, radioprotective compounds are crucial in clinical radiotherapy (3), and the administration of radioprotective compounds has been suggested as an approach for preventing radiation-damage in normal tissues (4,5).

Selenium is a trace element with a fundamental role in human biology (6). It detoxifies reactive oxygen species (ROS) produced by radiation treatment (4,7). In human antioxidant systems, selenium acts in the form of selenocysteine, which is incorporated into various selenoproteins (8,9). At least 25 selenoproteins have been identified in humans, including glutathione peroxidase (GPx), thioredoxin reductases, iodothyronine deiodinase and the selenoproteins P, W and R (10). Selenium exists in numerous chemical forms, of which the most studied are selenomethionine, sodium selenite, methylselenocysteine, 1,4-phenylenebis (methylene) selenocyanate and methylseleninic acid (9). Sodium selenite is the chemical form

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*Correspondence to:* Dr Hiroshi Koyama, Department of Public Health, Gunma University Graduate School of Medicine, 3-39-22 Showa Machi, Maebashi, Gunma 371-8511, Japan  
E-mail: hkoyama@gunma-u.ac.jp

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