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[Medicine] Pterostilbene Found Effective to Chemosensitivity; NCKU Researchers Prove Nicotine Hinders Bladder Cancer Chemotherapy

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NCKU Research Express (2012/02/24) Cigarette smoke is regarded as one major risk factor inducing bladder cancer and enhancing its resistance to chemotherapy. Professor Ying-jen WANG at the Department of Environment and Occupational Health, Medical College of National Cheng Kung University (NCKU) discovered that pterostilbene induces cancer cells' autophagocytosis and then can be used to treat chemotherapy-related allergic reaction and nicotine-induced chemoresistant bladder cancer during his investigation of the mechanism of nicotine's promotion of cancer development. The findings were already published in an article, "Long-term Nicotine Exposure–Induced Chemoresistance Is Mediated by Activation of Stat3 and Downregulation of ERK1/2 via nAChR and Beta-Adrenoceptors in Human Bladder Cancer Cells" in Toxicological Sciences (2010 115:1).

According to Professor Ying-Jen WANG, bladder cancer is the eighth most common cancer among the male population, the fourteenth among the female, in Taiwan. On the basis of the proved conclusion that nicotine is a common carcinogen causing lung cancer, Professor WANG and his postdoctoral researcher Rong-Jane CHEN furthered the study of nicotine's mechanism of the promotion of the cancer cell development and its chemoresistance. By the contrast that the bladder cancer patients who continue to smoke while receiving chemotherapy have poorer outcomes than the nonsmoking counterparts, they tried to suggest that certain substance in cigarette smoke can help the cancer cells resist the cell toxicity caused by drug and increase chemoresistance.

Professor WANG pointed out, nicotine is the major substance in cigarette smoke, which can be found in the urine of smokers. Nicotine has been proved to be a common carcinogen, and the activation of nicotinic receptor, especially acetylcholine receptor and epidermal growth factor receptor, can accelerate proliferation of cancer cells, hinder apoptosis, induce angiogenesis and even promote cancer cell invasion and metastasis. Thus, the team thought that long-term nicotine exposure may be the cause of bladder cancer cells' chemoresistance. Via the study of the molecular mechanism of nicotine's inducing chemoresistance, the team tried to understand how cancer cells can bear the toxicity caused by drug and survive after chemotherapy. The study aims to provide information for clinic therapeutic strategy as well as to suggest ways to enhance patients' chemosensitivity.

According to WANG's research, which uses Con-T24 cells that is adopted from bladder cancer cell lines and Nic-T24 cells that have been exposed to nicotine for a long time, the T24 cells that have been interacting with nicotine for a long time, can reduce the drug-induced apoptosis than the counterparts, while more expression of cyclin D1 and PCNA proteins in Nic-T24 cells, that detain the cells in the phase of G0/G1 cell cycle, can be observed.

Besides, the suppression of ERK1/2 caused by the overactivation of Stat3, the indicator for chemoresistance, is observed in Nic-T24 cells, and by testing with the inhibitor of nicotinic receptor or  $\beta$ -receptors, it is proved that the signaling transduction pathway is regulated by these receptors.

WANG's research indicates that the reduction of nicotine exposure in clinic cancer therapy can increase chemosensitivity, while the findings about the function of nicotinic receptors, β-receptors or Stat3 could contribute valuable information for new drug development for chemoresistant bladder cancer patients.

Further Information:
NCKU Research Express 2012/02/24 (Chinese)
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National Science Council International Cooperation Sci-Tech Newsbrief

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