

CURRICULUM VITAE

Qi Zhang, Ph.D

Personal Information

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Education

1995.9-1999.7 B.A, major in biochemistry, school of Life Sciences, Lanzhou University, China.
2000.9-2003.7 M.S, major in immunology chemistry, school of Life Sciences, Lanzhou University
2003.9-Present Ph.D, major in Cell Biology, school of Life Sciences, Lanzhou University

Research Interests

To obtain a full-time visiting researcher/postdoc position with the research background of Cell biology, pharmacology, molecular biology or related research background, which utilizes my experiences of cell biology, biochemistry, biophysics, molecular biology, DNA damage and repair, cell signal transduction and making animal model.

Current Status

I will defend my Ph. D thesis in June 2006. Now I'm working as a doctor candidate in Prof. Rongliang Zheng's Lab, at school of life sciences, Lanzhou University.

Participated Research Projects

DNA damage and repair in cells

Anticancer and antibacterial research of some natural or synthesized compounds

Differentiation of human hepatoma cells

Oxidative stress mechanisms in heroin-addicted mice and the therapeutic effects of different antioxidants

Professional Experiences

2000.9-2003.7 Graduate Student,

My thesis topic is *Study on antitumor activities and mechanisms of 6-(p-chlorophenyl)-3-[1-(p-chlorophenyl)-5-methyl-1H-1,2,3-triazol-4-yl]-1,2,4-triazolo[3,4-b]-1,3,4-thiadiazole.*

Cancer is a disease with deregulated cell proliferation, abnormal differentiation and apoptosis. Most tumor cells appear with relative undifferentiation or dedifferentiation. Conventional cancer therapies such as surgery, chemotherapy and radiotherapy have a number of limitations such as relatively high cost, development of resistance and are often accompanied with many adverse side effects. Therefore, there is a pressing need to search for new drugs and to establish new therapeutic strategies that are safe and effective for cancer treatments. Differentiation therapy is a novel approach for the treatment of human hepatoma carcinoma in which immature human hepatoma cells are induced to attain a mature phenotype when exposed to differentiation inducers, We studied the effects of synthesized s-triazolo[3,4-b]-1,3,4-thiadiazoles derivatives (TDZ) on

proliferation, differentiation and apoptosis of human hepatoma carcinoma cell line. Our results showed that tumor cells could be induced to differentiate not only phenotypically (by scanning electronic microscopy) but also functionally. In the same time, the mechanism of redifferentiation was studied by Flow cytometer and Laser confocal microscopy, and we found that $[Ca^{2+}]_i$ and mitochondria membrane potential may be important in differentiation. TDZ could also inhibit the proliferation of tumors *in vivo*.

2003.9-Present

Working as a doctor candidate of cell biology, molecular biology and free radical biology. Thesis is about *DNA damage and repair in cells*.

DNA damage is implicated in mutagenesis, carcinogenesis, aging and other degenerative diseases. Damaged DNA is encountered in cells by cellular repair mechanisms thus can be repaired; and genomic stability is maintained, in part, by reversing and/or repairing these lesions before mutations occurring. However, if DNA damage cannot be repaired, mutagenesis and carcinogenesis may occur.

In our experiment, cells were treated with H_2O_2 , a well-known strong oxidant, to cause DNA damage. Oxidative damage to individual DNA bases is repaired primarily via the base excision repair (BER). It has been shown that single-strand breaks of DNA are induced very rapidly after the addition of oxidants, which leads to the activation of DNA repair mechanisms. We use these BER inhibitors to block the BER pathway and see whether there exists the enzymatic-independent fast repair in cells. After DNA repair mechanism (BER pathway) inhibited, H_2O_2 led to a 2-5 fold increase of DNA damage level. Natural polyphenols are able to recover the DNA damage significantly. In our study, we provide the first evidence that non-enzymatic, fast repair may be a universal form in cells.

Other work

Participate *the study of Heroin Administered Mice and Antioxidants Protection*

Heroin addiction is a phenomenon with complex physiological and social causes and consequences. Despite a great deal of research, the exact mechanisms of dependence and withdrawal remain unclear. Most of phenomena occurring in heroin addicts, such as aging, abscess; arthritis, other rheumatologic disorders and immunity disorders are related to

degenerative diseases. However, the very important factor caused degenerative diseases is reactive oxygen species (ROS) (Zheng et al., 2001; Ames, 1983). There are some indirect evidences showed the relationships between heroin addiction and the products of free radical reactions. We guess that oxidative damage may be an important pathological factor in heroin addiction, therefore, antioxidant maybe a useful agent in the release of withdrawal syndrome. Therefore, my work was explored whether there was oxidative stress in heroin-addicted mice, and if it was existence, whether antioxidants could block those oxidative stresses and therefore be a new therapy for opiate abusing. Till now, we have already gained some merit and interesting results.

International Experiences

During the course of my studying, I have participated in some International meeting and international cooperation,

The 27th Symposium of Free Radical Research-Japan (Okayama, Japan)

The grant of “ Chunhui” Project of the Ministry of Education of China for the research cooperation between Chinese scientists in Australia and in the West of China.

Summary of Skills

* **Cell culture and test:** Cell culture, separation, freezing and defreezing, Cell apoptosis and cycle analysis, MTT, SRB, cell growth curve, scanning electronic microscopy (SEM), fluorescence microscopy, laser confocal microscopy, flow cytometer, etc.

* **Nucleic acid manipulation:** isolation and purification of DNA and RNA, PCR, RT-PCR technique, transformation, transduction.

* **Protein extraction, purification and analysis:** Protein isolation, SDS-PAGE, Western blotting, Co-IP.

* **Animal experimentation:** construction of mouse liver-toxicity model, mouse tumor model, heroin-addiction model etc., operation and handle of animal.

* **Computer:** Word, Excel, PowerPoint, Photoshop, and Using of bioinformation in Internet database.

Main Publications:

Zhang Q, Pan J, Zhao CY, Zheng RL, Wang. Qin. (2005) Redifferentiation of human hepatoma cell induced by synthesized 6-(p-chlorophenyl)-3-[1-(p-chlorophenyl)-5-methyl-1H-1,2,3-triazol-4-yl] -s-triazolo[3,4-b]-1,3,4-thiadiazole (TDZ). *Die Pharmazie*. 60(5):378-82.

Qi Zhang, Jing Pan, Chenyang Zhao, Yuan Wang, Zhongjian Jia, Rongliang Zheng. (2006) Fast Repair of Oxidative DNA Damage may exist in Cells. *DNA Repair*, submitted.

Pan J, **Zhang Q**, Zhang YT, Ouyang ZQ, Zheng QS, Zheng RL. (2005) Oxidative Stress in Heroin Administered Mice and Antioxidants Protection. *Life Sciences*. 77: 183–193.

Zhang Q, Pan J, Zheng RL. (2005) Oxidative stress caused by heroin and protection mechanisms by AOB in mice. The thesis of the 27th Symposium of Free Radical Research-Japan. 31

Pan J, **Zhang Q**, Zhao CY, Zheng RL. (2004) Redifferentiation of Human Hepatoma Cell Induced by Synthesized Coumarin. *Cell Biol Int*. 28: 329-333

Zhang Q, Guo GN, Miao RD, Chen NY, Wang Q. (2004) Studies on the Chemical Constituents of *Artemisia sieversiana* and the Activities of Anticancer. *J Lanzhou University (NATURAL SCIENCES)*

Zhang Q, Wang Q, Han Y, He YG, Hui XP. (2003) Study on Antibacterial Activity of s-thiazolo [3,4-b]-1,3,4-thiadiazine Derivatives and Structure Activity Relationships. *Microbiology (China)*. 30: 61-64.

Qi Zhang, Jing Pan, Yuan Li, Xinqing Xu, Rongliang Zheng. (2006) Oxidative stress caused by heroin and protection mechanisms by natural antioxidants in heroin-addiction mice. submitted.