Name: Pei-Jane Tsai
Lab place: 5773B room 3F, Department of Medical Laboratory Science and Biotechnology
E-mail: peijtsai@mail.ncku.edu.tw
Phone: 866-6-235-3535 ext. 5762
Fax: 866-6-236-3956

We study on the molecular toxicology and cancer biology.

Research Introduction

Bacterial pathogenesis ~ Host responses ~ from cells to knockout mice

My long term research goal is to study the host defending system, particularly focused on the innate immune system, in human inflammatory disease with an emphasis on solving the pathogenesis using mouse genetics as a tool. In the past few years, I have applied varies gene deficient animals, including Toll-like receptor (TLR)-, iNOS-, and cystatin C-deficient mice, to study how host deficient in certain defending system responds to the inflammatory diseases (Kuo et al., 2011; Tseng et al., 2011).

Pathogenesis of Streptoccocus pyogenes From 2000, I joined a research group focused on the pathogenesis of Streptococcus pyogenes and we have some fruitful results (Chang et al., 2007; Tsai et al., 2008; Chang et al., 2009a; Chang et al., 2009b; Chiang-Ni et al., 2011). In addition, our previous results showed that the inflammatory responses regulated by NF- B and MAP kinase were activated after streptococcal infection (Tsai et al., 2006). This paved the way to additional studies that address the next important questions of how these inflammatory responses are activated; ie, through which innate immune receptors, particularly on TLRs. In 2003, we collaborated with Dr. Kuei-Tai A. Lai (賴葵太) in DCB to import the TLR2-, TLR4-, and MyD88-deficient mice and established them in Tzu-Chi University, where I was working in 2000~2006, to continue our study. In 2010, we demonstrated that a novel streptococcal collagen-like surface protein 1 has the ability to promote bacteria adherent to epithelial cells (Chen et al., 2010).

Microbial diagnosis In addition, I also devoted to the development of functionalized nanoparticles for probing pathogenic bacteria in collaboration with Dr. Yu-Chie Chen (陳月枝) and obtained promising results (Chen et al., 2007a; Chen et al., 2007b; Li et al., 2007; Liu et al., 2008; Chen et al., 2008a; Chen et al., 2008b; Liu et al., 2009; Chen et al, 2009; Cheng et al., 2009). I developed a Beijing and non-Beijing differentiated multiplex PCR method to identify Beijing and/or non-Beijing strains of Mycobacterium tuberculosis directly from patient sputum in a single step, and demonstrated that mixed infection is present in patients with active pulmonary TB in eastern Taiwan (Huang et al., 2010； Huang and Tsai, 2011).

Molecular imaging Molecular imaging is a rapidly emerging field, providing noninvasive visual quantitative representations of fundamental biological processes in intact living subjects. Due to the family issue, in 2007, I moved to National Laboratory Animal Center (NLAC, 國家實驗動物中心南部設施) and developed an in vivo light reporting system, NF- B-RE-luciferase transgenic mice that express luciferase under the control of NF- B, to monitor the inflammatory responses in vivo. This mouse serves as a platform to analyze NF- B activation noninvasively in a variety of pathological conditions, particular on inflammation. During this period, I jointed a ENU project and identified a novel leptin gene mutation site in a obese mice (Hong et al., 2011).

After creating several light reporting mouse models in NLAC, in 2009, I moved back to the academic environment, which I particularly enjoy working with, in NCKU. For the next stage of my career, I plan to initiate and continue my research work on dissecting the contribution of TLR-mediated innate inflammation to infectious disease using animal model. This idea was initiated in 2005, while I was a visiting scholar in the laboratory of Dr. Oliver Smithies and Dr. Nobuyo Maeda at University of North Carolina at Chapel Hill. We have continued our collaboration for years together with Dr. Yau-Sheng Tsai (蔡曜聲) (Tsai et al., 2009； Jheng et al., 2011). I believe a combined basic and applied approach, with the focus on the inflammatory responses in human disease, is worth of such an investment in time and effort to better understand the innate immune system in host defending mechanisms.