Yong Fan

Current Position: Postdoctoral investigator in the Division of Immunogenetics, Department of Pediatrics, at University of Pittsburgh School of Medicine

Education: Ph.D. in Developmental Biology from the University of Pittsburgh

Non-scientific Interests: Jogging, reading, tennis – above all, having fun with my wife and children

I still vividly remember my first meeting with Dr. Mark Sperling when he enthusiastically showed me a diagram of glucose-disposal-rate change following an injection of growth hormone (GH) in human subjects. It was one sunny afternoon in early 2005 when my musing about the etiology of diabetes was interrupted by a visit from my mentor, Dr Massimo Trucco, Director of the Diabetes Institute at the University of Pittsburgh, and Dr. Sperling. The task was "simple": help Dr. Sperling solve the mystery of a targeted mutation line, which displayed some weird gene transmission pattern.

It turned out that the line was faulty (a geneticist's nightmare!), and we had to go back to ground zero to generate the exon 4-floxed GHR mouse. Although this was no longer a simple task, my fascination grew ever deeper with the little cytokine molecule that could serve pleiotropic roles in the body, ranging from growth, aging, and reproduction to tumor formation as well as energy homeostasis and metabolism. Once the GHR floxed mouse was generated, the three of us decided to target GH-action in the liver first, because diabetes and metabolism are our common interests, and the liver is the major source of circulating insulin-like growth factor I (IGF-I, also called somatomedin C), which was traditionally considered to be the major GH-mediating factor that promoted long bone growth. Contrary to the "Somatomedin Hypothesis," we did not observe any growth abnormality in our GHR liver-specific deletion mice, which we named GHRLD, even though circulating IGF-I was suppressed to less than 5% of normal—a finding that demanded a serious reconsideration of the hypothesis. A surprising major finding of the study was the observation of hepatic steatosis. In modern society, non-alcoholic fatty liver disease (NAFLD)—a factor considered to be responsible for insulin resistance, metabolic syndrome, and increased cardiovascular risk—is becoming increasingly prevalent. Our study clearly demonstrated an essential role of GH-signaling in hepatic lipid metabolism and export, and we are trying to elucidate its underlined molecular mechanism through ongoing studies. I believe that the GHR exon 4-floxed model we generated will serve as a powerful tool for the scientific community to answer many questions about the physiologic, biochemical, and molecular roles that GH-action has on a range of tissues and organs.

About my mediation over Type 1 diabetes: we recently developed a novel animal model, demonstrating a direct functional link between the ectopic insulin expression in the thymus and the body's immune tolerance towards pancreatic islets: a finding that will advance our understanding of the etiology of Type 1 diabetes and other autoimmune diseases. As a junior investigator who has the luxury to work under the influence of two outstanding role model scientists, I consider myself to be fortunate to commit myself to this research.

Read Dr. Fan's article entitled: Liver-specific Deletion of the Growth Hormone (GH) Receptor Reveals Essential Role of GH Signaling in Hepatic Lipid Metabolism ... http://www.jbc.org/cgi/content/full/284/30/19937