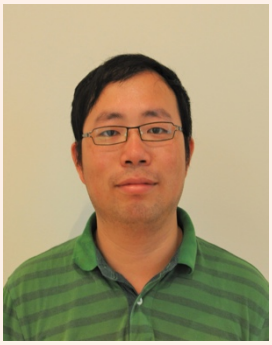
Department of Biomedical Informatics

Arizona State University

SPRING 2017

Biomedical Informatics Weekly Seminar Series

Hypothesis Testing for Mediation Effects in the Context of Epigenomic Studies



Jincheng Shen, PhD
Postdoctoral Research Fellow, Department of Biostatistics
Harvard University

March 3, 2017 (2:45 p.m. – 4:00 p.m)
Room Sj 1-149 , Samuel C. Johnson Research Bldg.
Mayo Clinic Scottsdale

Speaker's Bio: **Jincheng Shen** is a Postdoctoral Research Fellow at Harvard University. He received his PhD degree from the Department of Biostatistics at the University of Michigan under the joint supervision of Dr. Jeremy Taylor and Dr. Lu Wang. Currently he is working with Dr. Xihong Lin at Harvard University. His research focuses on developing causal inference methodologies for personalized treatment regime estimation for clinical studies. He also explores hypothesis testing for genetic mechanisms and pathways that mediates the effect of environmental exposures to complex diseases.

Talk Abstract: It is of increasing interest to study the underlying mechanisms of whether the effect of environmental exposures on disease outcome is mediated through genomic and epigenomic markers, e.g., DNA methylations. Mediation analysis provides a powerful tool for mechanism studies, and has gained great success in social science research. There has been growing interests in employing mediation analysis technologies in genetic and epigenetic studies to look at causal pathways and mechanisms. We have worked on addressing challenges of developing proper tests for single mediator, as well as multiple mediators in epigenomic setting. The proposed methods can effectively account for the composite nature of the null hypothesis and allow for weak and sparse signals. Extensive simulation studies have been conducted to assess the type I error rates and powers under various practical settings. The proposed tests have also been applied to the Normative Aging Study and identified putative DNA methylation CpG sites as mediators in the causal pathway of smoking behavior to lung functions.