BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2.
Follow this format for each person. **DO NOT EXCEED FOUR PAGES.**

NAME	POSITION TITLE
Wang, Kai	Professor
eRA COMMONS USER NAME (credential, e.g., agency login) KAIWANG	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	MM/YY	FIELD OF STUDY
Lanzhou University, Lanzhou, P.R. China	B.A.	1986	Mathematics
Nankai University, Tianjin, P.R. China	M.A.	1989	Econometrics
University of Iowa, Iowa City, Iowa	M.A.	1996	Economics
University of Iowa, Iowa City, Iowa	Ph.D.	1999	Statistics

A. Personal Statement.

B. Positions and Honors.

Positions and Employment

1989-1992 Lecturer, Nankai University, Tianjin, P.R. China
1992-1996 Teaching Assistant/Research Assistant, Department of Economics, University of Iowa, Iowa City
1996-1997 Instructor/Research Assistant, Department of Statistics, University of Iowa, Iowa City IA
1997-1998.1 Research Assistant, Department of Preventive Medicine, University of Iowa, Iowa City IA (on the NIH funded Collaborative Genetic Linkage Study of Autism)
1999-1999 Research Assistant Professor, Medical Statistics Section, Department of Medicine, University of Alabama at Birmingham, Birmingham AL
1999-1999 Biostatistician, Biostatistics Unit, Comprehensive Cancer Center, University of Alabama at Birmingham, Birmingham AL
1999-2003 Assistant Professor, Department of Biostatistics, College of Public Health, University of Iowa
2003-2005 Assistant Professor, Program in Public Health Genetics, College of Public Health, Univ. of Iowa
2005-2007 Associate Professor, Program in Public Health Genetics, College of Public Health, Univ. of Iowa
2007-2013 Associate Professor, Department of Biostatistics, College of Public Health, Univ. of Iowa
2010-2013 Associate Professor, Interdisciplinary Graduate Degree Program in Informatics, Bioinformatics Subtrack, University of Iowa
2013 Professor, Department of Biostatistics, College of Public Health, Univ. of Iowa

Other Experience and Professional Memberships

1999-present Member, The American Society of Human Genetics
1999-present Member, International Genetic Epidemiology Society
2000-2001 Chairperson for Departmental Seminar, Department of Biostatistics, College of Public Health
2003-present Director of Graduate Studies, Program in Public Health Genetics, College of Public Health

Honors

1984 Outstanding Student Award, Lanzhou University
1995 Ponder Award, University of Iowa
1999 Travel grant for CBMS Summer Course on Inferences From Genetic Data & On Pedigrees. Michigan Technical University, Houghton, Michigan
2001 New Investigator Research Award, College of Public Health-College of Medicine, University of Iowa

2002 Travel grant for Workshop on Developments and Challenges in Mixture Models, Bump Hunting and Measurement Error Models, Case Western Reserve University, Ohio.

2002 Travel grant for Frontiers of Statistical Research: A Celebration of the 40th Anniversary of the Department of Statistics at Texas A&M University, College Station, Texas.

2003 Finalist in Post-Doctoral Neal Young Investigator Award. Annual meeting of the International Genetic Epidemiology Society. Los Angeles.

C. Selected peer-reviewed publications (in chronological order).

1. Wang K, Huang J. Score test for mapping quantitative-trait loci with sibships of arbitrary size when the dominance effect is not negligible. Genet Epidem 23:398-412, 2002. PMID: 12432506
2. Wang K. Efficient score statistics for mapping quantitative trait loci with extended pedigrees. Hum Hered 54:57-68, 2002. PMID: 12566738
3. Wang K. Mapping quantitative trait loci using multiple phenotypes in general pedigrees. Hum Hered 55:1-15, 2003. PMID: 12890921
4. Wang K. Score tests for epistasis models on quantitative traits using general pedigree data. Genet Epidemiol 25(4):314-326, 2003. PMID: 14639701
5. Wang K. A likelihood approach for quantitative-trait-loci mapping with selected pedigrees. Biometrics, 61:465-473, 2005. PMID: 16011693
6. Wang K, Sheffield VC. A constrained-likelihood approach to marker-trait association studies. Am J Hum Genet, 77:768-780, 2005. PMID: 16252237
7. Wang K, Peng Y. Quantitative-trait-locus mapping in the presence of locus heterogeneity. Ann Hum Genet, 70:882-892, 2006. PMID: 17044863
8. Wang K, Abbott D. A principal components regression approach to multilocus genetic association studies. Genet Epidemiol, 32(2):108-118, 2008. PMID 17849491
9. Wang K. Genetic association tests in the presence of epistasis or gene-environment interaction. Genet Epidemiol,32:606-614, 2008. PMID: 18435472
10. Wang K. Testing for genetic association in the presence of population stratification in genome-wide association studies. Genet Epidemiol, 33:637-645, 2009. PMID: 19235185
11. Wang K. Statistical tests of genetic association for case-control study designs. Biostatistics. 13(4):724-733, 2012. PMID: 22389176
12. Liu J, Wang K, Ma S, Huang J. Accounting for linkage disequilibrium in genome-wide association studies: A penalized regression method. Statistics and Its Interface. 6:99-115, 2013
13. Wang K, Fingert J. Statistical tests for detecting rare variants using variance-stabilizing transformations. Annals of Human Genetics. 76:402-409, 2012. PMID: 22724536
14. Wang K, Hu X, Peng Y. An analytical comparison of the principal component method and the mixed effects model for association studies in the presence of cryptic relatedness and population stratification. Human Heredity. 76(1):1-9, 2013. PMID: 23921716
15. Wang K. Testing Genetic Association by Regressing Genotype over Multiple Phenotypes. PLOS ONE. Accepted 2014

D. Research Support.

<u>Ongoing Research Support</u>

5 P42 ES013661 (Robertson, Larry PI) 04/01/2010-03/31/2015
NIH/NIEHS
Semi-Volatile PCBs: Sources, Exposures, Toxicities (Superfund Research Program Administrative Core)

The Administrative Core is the focal point for the Research Projects and Cores of the Iowa Superfund Research Program and provides administrative oversight, statistical consulting, research results reporting, and serves as a liaison between the stakeholders, University officials, and the SRP.
Role: Co-Investigator

5 T15 HL097622 (Chaloner, Kathryn PI) 08/20/09-02/28/2016
NIH
Iowa Summer Institute in Biostatistics (ISIB)
There is a nationwide shortage of biostatisticians and the shortage is having a negative impact on medical and public health research. The goal of this proposed program is to increase the number of minority undergraduates who enter graduate programs in Biostatistics or related areas. Instruction will be through case-based instruction of real biomedical research; computer laboratory training; projects; and clinical and translational research enrichment activities.
Role: Co-Investigator

5 R01 ES022163 (Rohlman, Diane PI) 03/04/2013-10/31/2017
NIH
Vulnerability of the Adolescent Brain to Organophosphorus Pesticides
Despite evidence from human and animal studies that clearly identifies neurotoxicity as the primary adverse endpoint, the long-term effects of repeated occupational and environmental exposures to organophosphorus pesticides (OPs) remain poorly understood. There is also a critical need to investigate the susceptibility of children and adolescents to pesticides, since the developing brain may be uniquely sensitive to the neurotoxic effects of these agents. We propose a longitudinal study to investigate the relationship between sensitive and specific biomarkers of pesticide exposure, effect and susceptibility and multiple measures of neurobehavioral function in this unique cohort over a 5-year period to assess cumulative and potentially reversible effects.
Role: Co-Investigator

5 R01 EY023187 (Scheetz, Todd PI) 03/01/2013-02/29/16
NIH
Genetic Determinants of Optic Nerve Head Structure
The ultimate goal of this research proposal is identify biomarkers and/or genetic risk factors that accurately predict: (1) primary optic nerve head (ONH) structure (i.e. before age- or disease-related changes), (2) changes in ONH structure, and (3) the development of irreversible glaucomatous optic nerve damage before it occurs. These outcomes will improve the specificity and sensitivity of initial diagnosis of glaucoma, allowing clinicians to determine the proportion of ONH structure change that is damage from this disease, as opposed to normal variations in primary ONH structure. This in turn will allow the application of currently available and effective therapies to be instituted before vision is lost.
Role: Co-Investigator

2 R01 EY017673 (Anderson, Michael) 02/01/14-01/31/18
NIH
Genetic Dissection of Pigmentary Glaucoma
Glaucoma is a leading cause of irreversible blindness and visual disability that has a major impact on the quality of life and productivity of millions of Americans. With no new pharmaceutical classes for treating glaucoma introduced into clinical practice since the 1990s, there remains a continuing need for improved regimes that treat glaucoma more effectively. Our long-term goal is to contribute to the development of these improved therapies by utilizing synergistic genetic approaches with mice and humans. Our objective in this proposal is to utilize and build on these resources to study molecular events contributing to pigment dispersion and its conversion to pigmentary glaucoma. To accomplish this, we propose: (SA1) to identify suppressors of pigmentary glaucoma using hereditary mouse models, (SA2) to define predictors of ocular responses to pigment dispersion using inducible mouse models, and (SA3) to identify genes linked with pigmentary glaucoma using human patient cohorts.
Role: Co-Investigator

2 R 24 DK096518-02 (Engelhardt, John) 08/15/12-06/30/19
NIH
Early Pathogenesis of Cystic Fibrosis Related Diabetes
Cystic Fibrosis (CF) is the most common life-threatening autosomal recessive condition among Caucasians, with over $450 million dollars spent annually on clinical care of CF patients in the U.S. alone. Cystic fibrosis related diabetes (CFRD) is the most common severe complication of CF and is well known to be associated with increased mortality and a decline in lung function. This study will characterize early disease mechanisms that lead to the development of CFRD in animal models and humans, with the long-term goal of developing improved therapies and biomarkers for early diagnosis and treatment of this disease.
Role: Biostatistician