**CURRICULUM VITAE**

**Personal information**

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**Education**

* Bachelor, Public Health, Shandong (Medical) University, China
* Master, Medical Statistics (courses including Linear Algebra/Design of Experiment/Medical Statistics/Multivariate Analysis/Epidemiology at Fudan, and Mathematical Statistics/Optimization Methods/Numerical Methods at Shanghai Jiaotong University), Fudan (Shanghai Medical) University, China
* PhD, Statistical Genetics, King's College London, UK

**Professional positions**

1994.8-1996.5 Visiting scientist, Department of Environmental Science, School of Public Health & Channing Laboratory, Medical School, Harvard University

1996.5-2002.8 PostDoc & Lecturer (2001.3-2002.8), Section of Genetic Epidemiology and Biostatistics, Division of Psychological Medicine, Institute of Psychiatry, King's College London

2002.9-2005.9 Statistician, Social and Genetic Epidemiology, Department of Epidemiology and Public Health, University College London

2005.9-2018.7 Investigator scientist in Genetics, MRC Epidemiology Unit

2018.8- Genetic Analyst / Senior Research Associate, Cardiovascular Epidemiology Unit, Department of Public Health and Primary Care, University of Cambridge

**Research interests**

My work relates to methods and applications in human health-related research. Following earlier efforts on familial aggregation, segregation analysis, candidate genes and genomewide association studies (GWASs, it now capitalises on the meta-analysis at the Cardiovascular Epidemiology Unit (CEU) and within the SCALLOP consortium using the Olink inflammation as well as mass spectrometry (MS) panels measured for the INTERVAL samples. I have led analysis contributed to collaborative projects such as the Host Genetics Initiative and the SCALLOP-Seq(uence) consortium.

I have actively promoted reproducible research through distribution of software on CRAN (https://cran.r-project.org) and GitHub (https://github.com) and through web-based materials from my personal page, https://jinghuazhao.github.io/ and CEU page, https://cambridge-ceu.github.io/. I developed genetic analysis package (gap) as one of the earliest R packages for genetic data and recently pQTLtools for proteogenomic analysis. I have followed closely development in numerical optimization, signal processing, computational statistics, machine learning and expert systems/artificial intelligence. Curations include https://jinghuazhao.github.io/Computational-Statistics/, https://jinghuazhao.github.io/software-notes/, https://jinghuazhao.github.io/Omics-analysis/, and https://cambridge-ceu.github.io/csd3/. My recent exploration features on AI implementations for MS data with InstaNovo & DIA-NN, molecule optimization with DrugAssist, single-cell omics coupled with Seurat, scanpy, scvi-tools, scGPT and C2S-Scale under HPC, e.g., https://cambridge-ceu.github.io/csd3/systems/setup.html#fn:C2S.

**Key publications**

**Zhao JH**, et al. Mapping pQTLs of circulating inflammatory proteins identifies drivers of immune-mediated disease risk and novel therapeutic targets. *Nat Immunol* 2023, **24**(9):1540-1551, 10.1038/s41590-023-01588-w, https://www.nature.com/articles/s41590-023-01588-w.

COVID-19 Host Genetics Initiative. Mapping the human genetic architecture of COVID-19. *Nature* **600**:472–477 (2021); Pathak, G.A. et al. A first update on mapping the human genetic architecture of COVID-19. *Nature* **608**:E1-E10 (2022); The Host Genetics Initiative. A second update on mapping the human genetic architecture of COVID-19. *Nature* **621**:E7–E26 (2023).

**Zhao JH**, Luan JA, Congdon P. Bayesian linear mixed model of polygenic effects. *J Stat Soft.* 2018, **85**(6):1-27. doi: 10.18637/jss.v085.i06

**Zhao JH**, Luan JA. Mixed modeling with whole genome data. *J Prob Stat*. 2012*.* doi: 10.1155/2012.485174.

Xue F, Li S, Luan J, Yuan Z, Luben RN, Khaw K-T, Wareham NJ, Loos RJF, **Zhao JH**. A latent variable partial least squares path modeling approach to regional association and polygenic effect with applications to a human obesity study. *PLoS ONE* 2012, **7**(2): e31927

Loos RJ, *et al*. Common variants near MC4R are associated with fat mass, weight and risk of obesity. *Nat Genet* 2008; **40**(6):768-75

**Zhao JH**. gap: genetic analysis package. *J Stat Soft* 2007, 2**3** (8):1-18*.* doi: 10.18637/jss.v023.i08.

**Zhao JH**, Brunner EJ, Kumari M, Singh-Manoux A, Hawe E, Talmud PJ, Marmot MG, Humphries SE. *APOE* polymorphism, socioeconomic status and cognitive function in later mid-life: The Whitehall II longitudinal study. *Soc Psychiatr and Psychiatr Epidemiol* 2005, **40**:557-563

**Zhao JH**, D Curtis, PC Sham. Model-free and permutation tests for allelic associations. *Hum Hered* 2000, **50**(2), 133-139.

**Publications since 2018**

1. Demenais F, et al. Multi-ancestry genome-wide association study identifies new asthma susceptibility loci that co-localize with immune cell enhancer histone marks. *Nat Genet* 2018; 50(1):42-53
2. Medina-Gomez C, et al. Life-course genome-wide association study meta-analysis of total body BMD and assessment of age-specific effects. *Am J Hum Genet* 2018; 102(1):88-102
3. Sung YJ, et al. A large-scale multi-ancestry genome-wide study accounting for smoking behavior identifies multiple significant loci for blood pressure. *Am J Hum Genet* 2018, 102(3):375-400. doi: 10.1016/j.ajhg.2018.01.015.
4. Turcot V, et al. (2018). Protein-altering variants associated with body mass index implicate pathways that control energy intake and expenditure underpinning obesity. *Nat Genet* 50:26-41
5. **Zhao JH**, Luan JA, Congdon P. Bayesian linear mixed model of polygenic effects. *J Stat Soft* 2018, 85(6):1-27. doi: 10.18637/jss.v085.i06
6. Feitosa MF, et al. Novel genetic associations for blood pressure identified via gene-alcohol interaction in up to 570K individuals across multiple ancestries. *PLoS One* 2018, 13(6):e0198166. doi: 10.1371/journal.pone.0198166.
7. Lee JJ, et al. Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals. *Nat Genet* 2018, 50:1112–1121, https://doi.org/10.1038/s41588-018-0147-3.
8. Ligthart S, et al. Genome analyses of >200,000 individuals identify 58 loci for chronic inflammation and highlight pathways that link inflammation and complex disorders. *Am J Hum Genet* 2018,103(5):691-706. doi: 10.1016/j.ajhg.2018.09.009.
9. Evangelou E, et al. Genetic analysis of over 1 million people identifies 535 new loci for blood pressure traits. *Nat Genet* 2018, 50(10):1412-1425. doi: 10.1038/s41588-018-0205-x.
10. Merino J, et al. Genome-wide meta-analysis of macronutrient intake of 91,114 European ancestry participants from the cohorts for heart and aging research in genomic epidemiology consortium. *Mol Psychiatr* 2018 Jul 9. doi: 10.1038/s41380-018-0079-4.
11. Kilpeläinen TO, et al. Multi-ancestry study of blood lipid levels identifies four loci interacting with physical activity. *Nat Comm* 2019, 10, 376, https://doi.org/10.1038/s41467-018-08008-w.
12. Giri A, et al. Trans-ethnic association study of blood pressure determinants in over 750,000 individuals. *Nat Genet* 2019, 51:51-62
13. Karasik D, et al. Disentangling the genetics of lean mass. *Am J Clin Nutr* 2019, 109(2): 276-287, https://doi.org/10.1093/ajcn/nqy272.
14. de Vries PS, et al. Multi-ancestry genome-wide association study of lipid levels incorporating gene-alcohol interactions. *Am J Epidemiol* 2019, 188(6):1033-1054, https://doi.org/10.1093/aje/kwz005.
15. Justice AE, et al. Protein-coding variants highlight the importance of lipolysis in adipocytes for body fat distribution. *Nat Genet* 2019, https://doi.org/10.1038/s41588-018-0334-2.
16. Shrine N, et al. New genetic signals for lung function highlight pathways and chronic obstructive pulmonary disease associations across multiple ancestries, *Nat Genet 2019,* https://doi.org/10.1038/s41588-018-0321-7.
17. Zhao J, et al. Meta-analysis of genome-wide association studies provides insights into genetic control of tomato flavor. *Nat Comm* 2019, 10, Article number: 1534 (2019), https://www.nature.com/articles/s41467-019-09462-w
18. Sung YJ, et al. A multi-ancestry genome-wide study incorporatinggene–smoking interactions identifies multiple new locifor pulse pressure and mean arterial pressure. *Hum Mol Genet* 2019, doi: 10.1093/hmg/ddz070
19. Clark DW, et al. Associations of autozygosity with a broad range of human phenotypes. *Nat Comm* 2019, NCOMMS-18-33232A-Z
20. Bentley A.R., *et al.* Multi-ancestry genome-wide gene–smoking interaction study of 387,272 individuals identifies new loci associated with serum lipids. *Nat Genet* **51,** 636–648 (2019). https://doi.org/10.1038/s41588-019-0378-y
21. Shah S, et al. Genome-wide association and Mendelian randomisation analysis provide insights into the pathogenesis of heart failure. *Nat Comm* 2020, 11(1):163. Published 2020 Jan 9. doi:10.1038/s41467-019-13690-5
22. Surendran P, et al. Discovery of rare variants associated with blood pressure regulation through meta-analysis of 1.3 million individuals. *Nat Genet* 2020, 52(12):1314-1332, doi: 10.1038/s41588-020-00713-x.
23. Lin W, Ji J, Zhu Y, Li M, Zhao J, Xue F, Yuan Z. PMINR: pointwise mutual information-based network regression – with application to studies of lung cancer and Alzheimer’s disease. *Front Genet* 2020, 11:556259, https://doi.org/10.3389/fgene.2020.556259
24. Cuellar-Partide G, et al. Genome-wide association study identifies 48 common genetic variants associated with handedness. *Nat Hum Behaviour* 2021, 5: 59–70.
25. Gaziano L, et al. Actionable druggable genome-wide Mendelian randomization identifies repurposing opportunities for COVID-19. *Nat Med* 2021, 27:668–676.
26. Chen J, et al. The trans-ancestral genomic architecture of glycemic traits. *Nat Genet* 2021,53:840–860.
27. COVID-19 Host Genetics Initiative. Mapping the human genetic architecture of COVID-19. *Nature* 2021.
28. Zhang Y, et al. Mendelian randomisation highlights hypothyroidism as a causal determinant of idiopathic pulmonary fibrosis. *EBiomed*. 2021, https://doi.org/10.1016/j.ebiom.2021.103669
29. Graham SE, et al. The power of genetically diverse individuals in genome-wide association studies of blood lipid levels. *Nature* 2021,**600**: 675–679, DOI:10.1038/s41586-021-04064-3
30. Jin X, Zhang L, Ji J, Ju T, Zhao JH, Yuan Z. NeRiT -- Network regression in transcriptome-wide association studies. *BMC Genomics* (2022) 23:562, https://doi.org/10.1186/s12864-022-08809-w
31. Pathak, G.A. *et al.* A first update on mapping the human genetic architecture of COVID-19. *Nature* **608**, E1-E10 (2022).
32. Ramdas S, et al. A multi-layer functional genomic analysis to understand noncoding genetic variation in lipids. *Am J Hum Genet*. 2022 Aug 4;**109**(8):1366-1387. doi: 10.1016/j.ajhg.2022.06.012.
33. Wang Z, et al. Genome-wide association analyses of physical activity and sedentary behavior provide insights into underlying mechanisms and roles in disease prevention *Nat Genet.* 2022, **54**:1332–1344
34. Yengo L, et al. A saturated map of common genetic variants associated with human height from 5.4 million individuals of diverse ancestries. *Nature* 2022, **610**:704–712.
35. Kanoni, S., et al. Implicating genes, pleiotropy, and sexual dimorphism at blood lipid loci through multi-ancestry meta-analysis. *Genome Biology* 2022, **23**(1): 268 (medRxiv, DOI:10.1101/2021.12.15.21267852).
36. Shrine N, et al. Multi-ancestry genome-wide association study improves resolution of genes, pathways and pleiotropy for lung function and chronic obstructive pulmonary disease. *Nat Genet* 2023, **55:**410–422
37. van de Vegte JY, et al. Genetic insights into resting heart rate and its role in cardiovascular disease. *Nat Comm* **14**:4646 (2023), https://www.nature.com/articles/s41467-023-39521-2
38. **Zhao JH**, et al. Mapping pQTLs of circulating inflammatory proteins identifies drivers of immune-mediated disease risk and novel therapeutic targets. *Nat Immunol* 2023, **24**(9):1540-1551, 10.1038/s41590-023-01588-w, https://www.nature.com/articles/s41590-023-01588-w.
39. Lagou V, et al. GWAS of random glucose in 476,326 individuals provide insights into diabetes pathophysiology, complications and treatment stratification. *Nat Genet* **55**:1448–1461 (2023), https://www.nature.com/articles/s41588-023-01462-3, *medRxiv*, DOI: 10.1101/2021.04.17.21255471.
40. The Host Genetics Initiative. A second update on mapping the human genetic architecture of COVID-19. *Nature* **621:**E7–E26 (2023).
41. Macdonald-Dunlop E, et al. Mapping genetic determinants of 184 circulating proteins in 26,494 individuals to connect proteins and diseases. *medRxiv*, DOI:10.1101/2021.08.03.21261494.
42. Klaric L, et al. Mendelian randomisation identifies alternative splicing of the FAS death receptor as a mediator of severe COVID-19. *medRxiv*, doi: https://doi.org/10.1101/2021.04.01.21254789.
43. Gaziano L, et al. Transcriptome- and proteome-wide Mendelian randomization to prioritize therapeutic targets for coronary heart disease. m*edRxiv*, DOI: 10.1101/2024.06.27.24309406v1.
44. Kelemen M, et al. Performance of deep-learning based approaches to improve polygenic scores. *MedRxiv*, DOI: 10.1101/2024.10.23.24315973.
45. Koprulu M, et al. Proteogenomics 2.0: Multi-cohort analyses to characterise genetic effects across the proteome and phenome
46. Zhao JH. Genetic association analysis with R (II). The Biomedical & Life Sciences Collection **2025**, (2025).
47. Smit RAJ, et al. Polygenic scores to predict body mass index and obesity across populations and through the life course powered by data from 5.1 million individuals. *Nat Med* (Accepted).