





Methods to dissect cell-type heterogeneity in bulk and single-cell data with applications to cancer, cancer-risk and aging

Dr Andrew Teschendorff

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Host: Shyam Prabhakar





GIS Seminar Room (Level 2) 60 Biopolis Street, Genome, Singapore 138672

About The Speaker

Andrew Teschendorff is currently a Principal Investigator at the Shanghai Institute of Nutrition and Health, part of the Chinese Academy of Sciences, leading a lab in Computational Systems Epigenomics, with a research focus on aging and cancer-risk. He trained as a Mathematical Physicist at the University of Edinburgh (1990-1995) and Cambridge University (1996-2000) where he obtained his PhD. From 2003 to 2008 he held Cambridge-MIT and Isaac Newton Fellowships to conduct research in Statistical Cancer Genomics at the University of Cambridge. From 2008 to 2013 he held the Heller Research Fellowship at the UCL Cancer Institute in London. In 2013 he moved to Shanghai where he is currently a PI at the CAS Key Lab of Computational Biology, part of the Shanghai Institute for Nutrition and Health. From 2015 to 2019 he held an International Newton Fellowship from the Royal Society in association with UCL London. In 2023 he received a Highly Cited Researcher award from Clarivate in recognition of how his work published in premier journals has influenced the epigenomics, aging and cancer systems biology fields. He is an associate editor for Genome Biology and holds patents on algorithms for cancer risk prediction and cell-type deconvolution.

About The Seminar

My lab is interested in elucidating the role of epigenetic changes, notably DNA methylation, in aging, cancer-risk and cancer itself. To attain this goal, we have been developing computational methods that address some of the key emerging challenges, such as how to infer cell-type specific DNAm changes from large bulk-tissue epigenome datasets, how to identify stem-like cells in preneoplastic lesions from scRNA/snRNA-Seq data, or how to identify disrupted regulatory networks in aging at cell-type resolution. In this talk I will describe 4 different computational methods (EpiSCORE, SCIRA, CancerStemID, stemTOC) we have recently developed to tackle these challenges, including the construction of a pan-tissue DNAm-atlas to allow cell-type deconvolution of bulk-tissue DNA methylomes, an epigenetic mitotic clock for cancer risk prediction and an algorithm to detect single preneoplastic cells at elevated cancer-risk. Overall, the results obtained by these methods are supportive of an epigenetic stem-cell model of oncogenesis and highlight the value and importance of DNAm as a sensitive, and potentially causal, marker for quantifying cancer-risk.