THE GIS
SPEAKER SERIES

Genome Institute of Singapore
A*STAR GIS

Cancer genes beyond chromosomes

Dr. Howard Y. CHANG
Chief Scientific Officer
Amgen

Host: WAN Yue

Monday 16 February 2026
10.00am – 11.00am

GIS L2 – Seminar Room
60 Biopolis Street, Genome, Singapore 138672

About The Speaker

Dr. Howard Y. Chang is Chief Scientific Officer and Senior Vice President of Global Research at Amgen. He is responsible for leading all aspects of discovery research at Amgen.

Prior to joining Amgen, Dr. Chang led a research laboratory at Stanford University. A physician-scientist and board-certified dermatologist, he served as Professor of Dermatology, Genetics, and Pathology at Stanford and Investigator of the Howard Hughes Medical Institute. Dr. Chang discovered a new class of genes, termed long noncoding RNAs, can control gene activity throughout the genome, illuminating a new layer of biological regulation. He invented ATAC-seq and other new methods for defining DNA regulatory elements genome-wide and in single cells. The long term goal of his research is to decipher the regulatory information in the genome to benefit human health.

Dr. Chang is the winner of the Albany Prize, Lurie Prize in Biomedical Sciences, and NAS Award in Molecular Biology for discoveries of regulatory RNAs. He is an elected member of the US National Academy of Sciences, National Academy of Medicine, and American Academy of Arts and Sciences. He is a serial entrepreneur having founded five biotech companies.

Dr. Chang holds a M.D. from Harvard Medical School, a Ph.D. in Biology from MIT, and an A.B. in Biochemical Sciences from Harvard University.

About The Seminar

Cancer patients face an extraordinary challenge when oncogenes unleash themselves from chromosomes. Extrachromosomal DNA (ecDNA) are large, megabase-sized circular episomes containing oncogenes and regulatory DNA elements. EcDNAs have a remarkable transcriptional advantage and rapidly change copy number--a moving target driving accelerated evolution in cancer. EcDNAs are common in many of the most aggressive forms of cancer of women and men, children and adults, and contribute to treatment resistance and shorter survival for patients. EcDNAs lack centromere; yet ecDNAs are somehow not lost in successive rounds of cell division. EcDNAs were first observed to tether to and travel on mitotic chromosomes into dividing cancer cells more than 40 years ago, a phenomenon termed “mitotic hitchhiking”. The mechanism of mitotic hitchhiking is a longstanding mystery. Here we discovered a family of DNA elements in the human genome, termed retention elements, that enable mitotic hitchhiking and thereby confer episome immortality. We uncover the regulatory logic of retention elements, identify their chromosomal contact sites, and show how the co-amplification of retention elements shape ecDNA content and copy number in human cancers.