Cologne Seminar Series on Ageing

Jan Vijg
Department of Genetics, Albert Einstein College of Medicine, NY, USA
Center for Single-Cell Omics in Aging and Disease, Jiao Tong University School of Medicine, Shanghai, CN

Monday, 27th June 2022
at 2:00 pm

hybrid

Host: Björn Schumacher
(CECAD)

Scientific Background:
Jan Vijg, Ph.D., is Professor and Chairman of the Department of Genetics at the Albert Einstein College of Medicine in New York since July, 2008. He received his Ph.D. at the University of Leiden, The Netherlands, in 1987. From 1990 to 1993 he was founder and Scientific Director of Ingeny B.V., a Dutch Biotechnology company. In 1993 he moved to Boston, to take up a position as Associate Professor of Medicine at Harvard Medical School. In 1998 he accepted an offer from the University of Texas Health Science Center in San Antonio, Texas, to become a Professor in the Department of Physiology. From 2006 to 2008 he was a Professor at the Buck Institute for Age Research in Novato, California. He is a founder of several biotech companies as well as the founding Director of the Center for Single-Cell Omics (CSCOmics) at Jiaotong University School of Medicine in Shanghai, China (2019).

Aging of the genome: A single-cell approach

About Vijg's talk:

Genome instability, i.e., genetic alterations or mutations that can vary from small base substitution mutations to large chromosomal changes, is a hallmark of both aging and cancer. Mutations are inevitable due to errors in genome replication and repair of DNA damage. They are also irreversible except through cell or organismal death. While mutations in the germline are responsible for the great diversity and perpetuity of life, mutations in the soma of multicellular organisms such as humans over the lifetime result in genome mosaicism, i.e., gradual dissimilarities in genome sequence information. In turn this can have adverse effects, resulting in diseases such as cancer and, possibly, aging in general. Until recently our insights into genome instability were limited to germline variation across individuals or clonal lineages, such as tumors. However, de novo somatic mutations in normal tissues remained beyond reach. We and others developed methods for analyzing DNA sequence variation by whole genome sequencing of single cells or nuclei. Using these methods we studied genome instability in human blood, liver, lung and mammary gland. I will first review the concept of genome mosaicism and the various approaches to study the phenomenon. I will then present data from our lab about somatic mutations in the context of the aging process and cancer risk and discuss the possibility that somatic mutation accumulation is a basic cause of aging, thereby increasing the risk of cancer and other age-related diseases.

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