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Fumio Takei, PhD Distinguished Scientist, BC Cancer Research Institute, Terry Fox Laboratory; Professor, Department of Pathology & Laboratory Medicine, University of British Columbia

The 2024 CSI – Bernhard (Hardi) Cinader Award Recipient

Presentation: "Long and winding road across the field of innate lymphocytes"

Dr. Fumio Takei was born in Japan and obtained his BSc from University of Tokyo in 1968. After graduation, he worked for industry for 3 years before he started graduate studies at UBC in 1971. He worked in the laboratories of Drs. Doug Kilburn and Julia Levy to investigate suppressor T cells for his PhD. In 1977 he obtained NCIC fellowship and started his postdoctoral work in the laboratory of Dr. Edwin Lennox at the MRC Laboratory of Molecular Biology, Cambridge, England. During his postdoctoral work, he collaborated with Dr. Cesar Milstein and learned the hybridoma technology.

In 1980, he received a scholarship from the Medical Research Council of Canada to start his own laboratory as an assistant professor in the Department of Pathology and Laboratory Medicine, UBC. In 1981, he joined the newly established Terry Fox Laboratory as a scientist. In 1980s and early 1990s, his laboratory generated a number of rat monoclonal antibodies to mouse cell surface proteins, including Ly49A (KLRA1), ICAM-1 (CD54), PC-1 (ENPP1) and CD160. His research initially focused on cloning cDNA for those proteins and characterizing their functions. Subsequently, his research mainly focused on the Ly49 family of NK cell receptors. In collaboration with Dr. Dixie Mager, a colleague at the Terry Fox Laboratory, Dr. Takei's laboratory identified multiple members of the Ly49 family of NK cell receptors and determined their specificities for MHC class I.

While investigating how NK cells acquire the Ly49 family of receptors during their development, his laboratory found a unique population of lymphocytes that produced large amounts of type 2 cytokines in mouse lungs. The population was initially termed natural helper (NH) cells but later renamed group 2 innate lymphoid cells (ILC2s). Following this finding, ILC2s became the main focus of his research. ILC2s were found to drive allergen-induced allergic lung inflammation but also promote initiation of Th2 responses to allergens. The Takei Lab also investigated the development of ILC2s and identified RORα as a key transcription factor and generated ILC2-deficient mice and ILC2 lineage tracer mice. Using those mice, the Takei Lab identified new ILC progenitors in neonatal lung and spleen. Flow cytometry and transcriptome analyses of activated lung ILC2s also revealed multiple functional subsets, including memory ILC2s and migratory ILC2s. Currently, Dr. Takei continues his research on ILC2s and other ILCs in inflammation and fibrosis of the liver and the lung.