

令和7年度 共同研究報告書

研究区分		一般共同研究		
研究課題名		Role of histone deacetylases for anti-tumor immune responses		
新規・継続の別		新規・継続		
研究代表者	所属	Institute of Immunology, Center for Pathophysiology, Infectiology and Immunology Medical University of Vienna	35歳 以下○	40歳 以下○
	職名・氏名	Associate Professor, Shinya Sakaguchi		○
研究分担者 (適宜行を追加し て下さい)	所属		/	/
	職名・氏名			
	所属		/	/
	職名・氏名			
受け入れ教員	職名・氏名	Prof. Takaoka Akinori		
概要 (100～150字程度)		I have visited the group of Prof. Takaoka at the Institute for Genetic Medicine (IGM), Hokkaido University from 3 <sup>rd</sup> to 6 <sup>th</sup> of February 2026. During my stay at the IGM, we had very active discussion on potential collaborative projects. In addition, I had a unique opportunity to present our ongoing projects in a seminar hold at the IGM and received valuable feedbacks for the projects.		
研究目的 (300字程度)		Histone deacetylases (HDACs) remove acetyl groups from lysine residues on histones and thereby control gene expression. Owing to their essential function, HDACs orchestrate various biological processes, including immune cell differentiation and tumorigenesis, and several HDAC inhibitors are already under clinical trial for cancer patients. However, the role of HDACs during anti-tumor immune responses still remains largely elusive. Our group is currently investigating their roles in T cells during tumorigenesis (e.g. T cell exhaustion). With the support of IGM joint research program, we planned to have a kick-off meeting with Prof. Takaoka's group, a world-leading group in the field of innate immune responses, to initiate elucidation of the role of HDACs in anti-tumor immune responses in a comprehensive manner.		
研究内容・成果 (1000字程度・Web会議の回数も記載)		Firstly, we introduced ongoing projects in each group. While I mainly introduced the role of HDACs for T cell exhaustion in the settings of chronic viral infection and tumor, the members of Prof. Takaoka's group explained several projects addressing the role of RNA sensors in the innate immune system and tumorigenesis. In particular, their genome-wide CRISPR screening system to identify novel molecules involved in		

	<p>RNA-sensor signaling was very insightful, since our group is also establishing such system to elucidate the molecular mechanisms underlying T cell exhaustion. After then, we had a very intensive discussion on how to merge and synergize our expertise to address unexplored research questions in the field. We came up with the idea that we shall investigate the potential role of RNA sensor-signaling for T cell exhaustion. Moreover, since Prof. Takaoka's group is currently studying immune responses in type I diabetes (T1D) where CD8+ T cells play a critical role for the disease progression, we think it would be highly interesting to examine if molecular mechanisms controlling T cell exhaustion exert similar or different functions for CD8+ T cell differentiation in T1D. The resultant knowledge could be leveraged for designing novel therapeutic approaches for T1D patients. Based on our discussion, my group will test the role of RNA-sensor signaling molecules for T cell exhaustion using CRISPR-KO approach. Moreover, we may compare the transcriptome of CD8+ T cells isolated from NOD mice spontaneously bearing T1D and from mice infected with LCMV inducing chronic viral infection. This will allow us to get first insight into similarity and difference in mechanisms underlying CD8+ T cell differentiation in these two settings.</p>
<p>成果</p>	<p><b>【学会報告】</b>  参加者名、講演タイトル、学会名、開催場所、開催日時入力のこと  None</p> <p><b>【論文発表】</b>  著者、論文名、掲載誌名、号・年・ページ等、IF 入力のこと  None</p> <p><b>【新聞報道】</b>  None</p> <p><b>【学位取得者】</b>  学部名・学年（または職位）・氏名を入力のこと  None</p>