ID No.	K2007	
Project Title	Analysis of plethora of ASXL functions under physiological and	
	pathological conditions	
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Project Members		
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Report	•	

Kitamura's group have been collaborating with Abdel-Wahab's lab for nearly 10 years. This year, based on the collaboration with Abdel-Wahab's lab, we found that the mutant ASXL1 induces age-related expansion of phenotypic hematopoietic stem cells through activation of Akt/mTOR pathway (Fujino et al. Nat Commun, 2021). Through this collaboration, Takeshi Fujino, the first author of this paper, decided to move to Abdel-Wahab's late this year as a postdoctoral fellow to further enhance the collaborative status between the two labs.

Inoue's lab recently reported in collaboration with Abdel-Wahab's lab that BRD9 (Bromodomain-containing protein 9) is post-transcriptionally degraded in substantial subsets of cancer, especially in myeloid malignancies (Inoue et al. Nature 2019). Following the successful project, we collaboratively investigated the role of BRD9 in normal and malignant hematopoiesis in terms of chromatin three-dimensional regulation and promoter-enhancer chromatin loops (Xiao et al. in preparation). Moreover, we are working together to elucidate the functional consequences of the novel EVI1 isoform caused by the aberrant splicing machinery (Tanaka et al. resubmitted). These projects are supported by mutual discussions in monthly virtual meetings.