若手人材育成部会･研究支援報告書

氏名	秦　圓圓　(シン　エンエン)
所属大学名	大阪大学 大学院連合小児発達学研究科/小児発達学専攻
職位または学年（学生の場合）	D2
申請内容（渡航先、参加学会、ワークショップ等）	参加第48回日本脳科学会(浜松)
実施年月日	2021.12.04-2021.12.05
内容報告	Title: The molecular mechanism by which GPCRs localize to the primary cilia(Implications for ciliopathies with intellectual disability and autism)Background: A subset of GPCRs localize specifically to primary cilia. Genetic damage to primary cilia results in pleiotropic disorders termed ciliopathies, some of which are accompanied by intellectual disability (ID) and autism. It is possible that failure of cilia localization of GPCRs leads to dysfunction of primary cilia in neurons, thereby inducing abnormal neuron activity that is observed in ID and autism. Method: The fourth intracellular domain (i4) of Htr6 fused to CD8α localizes to primary cilia in hTERT-RPE1 cells, suggesting that localization of Htr6 to primary cilia depends on its i4 portion. Therefore, interactors of Htr6-i4 are searched by yeast two-hybrid screening of a mouse cDNA library.　Result:45.5 million clones were screened, and three molecules were identified as candidates that interact with Htr6-i4. Interaction test in yeasts and immunoprecipitation/Western Blot analysis in HEK293T cells demonstrated interaction of Htr6-i4 with two of the identified candidates both in yeasts and in mammalian cells
備考	