東医大誌　**79**(1)：86-117, 2021

総 会 記 事

第 186 回東京医科大学医学会総会

日　　時：2020 年 11 月 7 日（土）12 時 00 分～14 時 30 分
開　　催：ウェビナー開催
当番分野：循環器内科学分野、泌尿器科学分野

ポスター発表（Zoom 開催）：1-1～1-10、2-1～2-6、③-①-1～8、③-②-1～8、4-①-1～4-①-5、4-②-1～4-②-7、5-①-1～5-①-7、5-②-1～5-②-6

1-1.
Exploration of pathogenesis for central nervous system dysfunction and changes in glial cells of sporadic ALS employing model mice

（社会人大学院博士課程 4 年、神経学分野）
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［Objective］ ALS is a common neurodegenerative disease of unknown etiology characterized by selective degeneration of both UMN and LMNs. In the LMNs of ALS, ADAR2（adenosine deaminase acting on RNA2）mediated RNA editing of GluA2 mRNA at the Q/R site was profoundly deficient. Since genetically modified mice（ADAR2 $^{flox/flox}$/VCAhT-Cre. Fast or AR2 mice）in that the ADAR2 gene is selectively knocked out in cholinergic neurons using Cre-loxP system underwent progressive loss of LMNs in an ADAR2-deficient manner, deficient RNA editing at this site is considered to be a cause of death of LMNs. However, the cause of UMNs in ALS is largely unknown. It has been revealed that pathological changes in glial cells are actively involved in the pathology of ALS. Based on these results, to test whether the loss of LMNs influences UMN survival, we examined UMN dysfunction and changes in astrocytes and microglias in the central nervous system of AR2 mice.
［Methods］ We investigated the changes in morphology, size, and the number of Betz cells in AR2 mice at the age of 12 months（n=3）, compared with wild type mice at comparative age（n=5）and tested the significance of the change using Mann-Whitney's U-test. All counted Betz cells had full soma and a prominent apical dendrite in the layer V of primary（M1）and secondary motor cortex（M2）.
［Results］ The number of large Betz cells was significantly decreased both in M1（the number of Betz cells/section, mean ± SEM: for AR2 mice, 22.2±4.1 ; for control mice, 27.2±2.8, $p<0.001$）and M2（the number of Betz cells/section, mean ± SEM : for AR2 mice, 41.6±5.1 ; for control mice, 49.6±8.7, $p < 0.001$）of AR2 mice. The number of reactive astrocytes of M1, M2, and spinal cord was increased in AR2 mice.
［Conclusions］ These results indicate that loss of ADAR2 is likely involved in the pathogenesis of UMN dysfunction of ALS.