1-LBS-15 Late-Breaking Session

Early postnatal lethality of mice lacking mitochondrial protein p13

Norihito Shintani, Hiroki Ueno, Satomi Hara, Sae Ogura, Naoki Inoue, Yohei Morota, Hitoshi Hashimoto

Grad. Sch. Pharmaceut. Sci., Osaka Univ.

p13 is mitochondrial protein *widely expressed in central* and peripheral tissues. Recently, we generated mice lacking p13 (p13-/- mice), and found that p13-/- genotype was smaller than the expected Mendelian ratio at 3 weeks of age (approx. 40% of the expected ratio). Here, we investigated the possible mechanisms underlying the loss of p13-/- mice. At postnatal day 0 (P0), Mendelian segregation of pup genotypes from heterozygous breeding was observed (n = 294, $P = 0.25$, χ^2 analysis), suggesting a significant loss of p13-/- pups specifically during the postnatal period. Kaplan-Meier survival analysis demonstrated that more than half of p13-/- mice died during the first 2 postnatal days. At P0, we observed the presence of milk in p13-/- pups stomach, however, their blood glucose levels were significantly lower than that of wild-type littermates. Taken together, the present results suggest that p13 contributes to early postnatal survival and maintenance of the normal blood glucose levels.