Inefficient development of syncytiotrophoblasts in the Atp11a-deficient mouse placenta.

Yuki Ochiai, Katsumori Segawa (present: Tokyo Medical and Dental University), Shigekazu Nagata (Biochemistry & Immunology, IFReC), and the research group showed that a flippase Atp11a at the plasma membrane plays an important role in the formation of syncytiotrophoblasts in placental development.

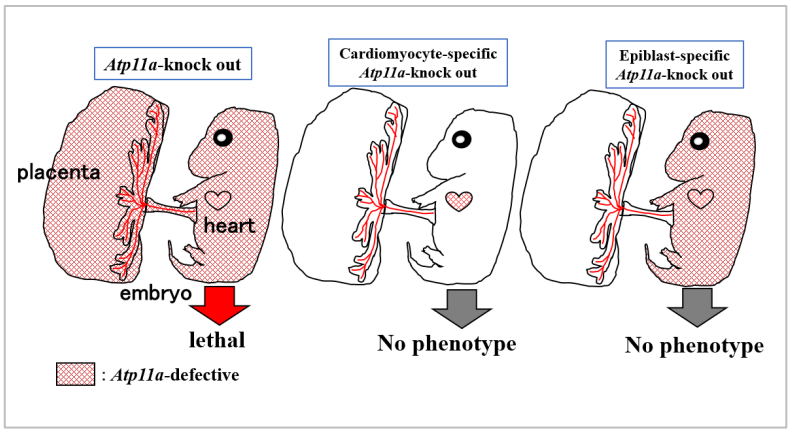

Figure 1. Embryonic lethality of Atp11a-/- mice. Atp11a-/- mouse embryos died at E14.5 with thin-walled heart ventricles. However, the cardiomyocyte- or epiblast-specific Atp11a deletion did not affect mouse development, suggesting the inability of Atp11a-/- placentas to support the embryos.

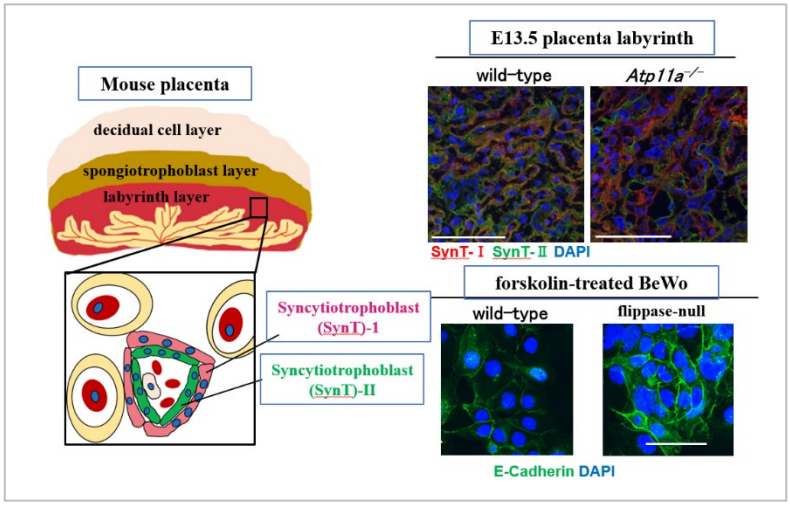

Figure 2. Poor development of the labyrinth layer of the Atp11a-/- placenta. (left) A scheme of the mouse placenta consisting of decidual cells, spongiotrophoblast, and labyrinth layers. Two syncytiotrophoblasts (SynT-I and SynT-II) tightly adhere and separate the maternal and fetal blood compartments in the labyrinth layer. (right) The two syncytiotrophoblasts were not well developed, and the labyrinth was sparse in the Atp11a-/- placenta. Treatment of BeWo cells with forskolin causes cell fusion accompanied by the reduction of E-cadherin at the plasma membrane. The flippase-null mutation blocks the process.

Journal: Proc. Natl. Acad. Sci. USA (April 28, 2022 online)

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