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The syncytin-A envelope gene of retroviral origin is essential for mouse placental development

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In several mammalian species, the fusion of throphoblastic cells into a multinucleated syncytiotrophoblast layer - taking place at the fetomaternal interface-constitutes a key process of placenta morphogenesis. In the last years, two pairs of genes of retroviral origin encoding envelope proteins were identified in the human (*syncytin-1* and *-2*) [1-3] and mouse (*syncytin-A* and *-B*) [4] genomes. These genes, that were independently acquired through ancient retroviral infections of the primate and muridae lineage, 20 to 40 MYA ago, display the characteristics of potential effectors of the trophoblast fusion process. In fact, syncytin genes are specifically expressed in the placenta, at the interhaemal syncytial barrier, display cell-cell fusogenic activity, and have been functionally conserved in evolution.

Here, to definitely assess the role of these genes, we generated knockout mice for *syncytin-A*. We show that homozygous *syncytin-A* null embryos die *in utero* between 11.5 and 13.5 days of gestation. *Syncytin-A*-deficient placentae exhibit disruption of the architecture of the syncytiotrophoblast-containing labyrinth, with overexpansion of trophoblast cells at the expense of fetal blood vessels spaces, and defects in formation of one of the two interhemal syncytial layers. These results demonstrate that *syncytin-A* is essential for placenta development and syncytiotrophoblast morphogenesis, and provide evidence that genes captured from ancestral retroviruses have been pivotal in the acquisition of new important functions during mammalian evolution.

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