Insights: Publications

Autophagy as a mechanism of antiviral defense at the maternal-fetal interface

Autophagy

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Mechanisms to protect against viral infections are crucial during pregnancy as maternal-fetal transmission can have serious pathological outcomes, including fetal infection and its sequelae, such as growth restriction, birth defects, and/or fetal death. The trophoblast forms the interface between the feto-placental unit and the maternal blood, and is therefore a critical physical and immunological barrier to restrict the spread of pathogens into the fetal microenvironment. Recently, we found that primary human placental trophoblast (PHT) cells are highly resistant to infection by diverse viruses. In this study, we also found that conditioned medium from PHT cell cultures transferred viral resistance to nonplacental recipient cells, suggesting that a component secreted by trophoblasts and present within the conditioned medium is responsible for this antiviral effect. We found that specific miRNAs from a unique primate- and placental-specific locus the C19MC (chromosome 19 miRNA cluster) are packaged within exosomes produced by PHT cells and confer viral resistance in nonplacental recipient cells. In addition to conveying viral resistance, we found that PHT-derived exosomes and select miRNA members of the C19MC family strongly induce autophagy, which is involved in recipient cell viral resistance. Our findings establish an exciting and novel mechanism by which placental trophoblasts exploit exosome-dependent transfer of placental-specific miRNAs to influence autophagic induction and antiviral immunity at the maternal fetal interface.

Related People



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