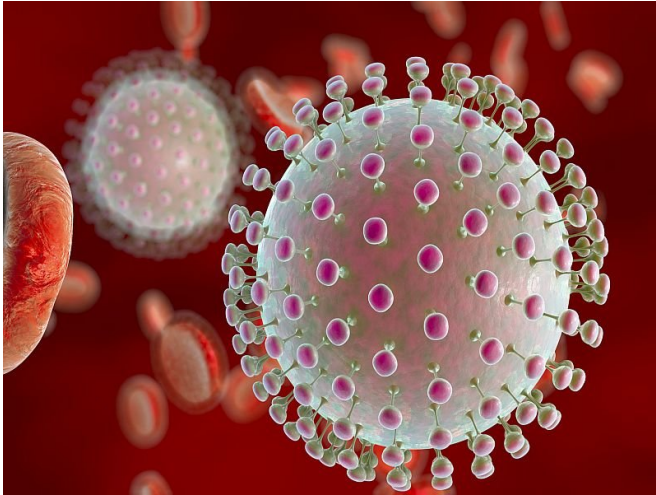


# Type 1 IFNs may mediate Zika pregnancy complications

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signaling in the conceptus was found to inhibit development of the placental labyrinth, resulting in abnormal maternal-fetal barrier architecture after ZIKV infection. Exposure of midgestation human chorionic villous explants to type I IFN resulted in altered placental morphology and cytoskeletal rearrangements within the villous core; this was not seen with exposure to type III IFN.

"Our results implicate type I IFNs as a possible mediator of [pregnancy complications](#), including spontaneous abortions and growth restriction, in the context of congenital viral infections," the authors write.

**More information:** [Abstract](#)  
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(HealthDay)—Type 1 interferons (IFNs) may mediate pregnancy complications in the context of congenital Zika virus (ZIKV) infection, according to a study published in the Jan. 5 issue of *Science Immunology*.

Laura J. Yockey, from the Yale University School of Medicine in New Haven, Conn., and colleagues examined the role of type 1 IFNs in limiting or mediating ZIKV disease within a model of [congenital infection](#), observed after ZIKV [infection](#) of type I IFN receptor knockout (*ifnar1*<sup>−/−</sup>) dams mating with wild-type sires, resulting in fetuses with functional type 1 IFN signaling. Pregnant dams carried a mixture of fetuses that expressed or did not express IFN- $\alpha$  receptor (IFNAR) (*ifnar1*<sup>+/?</sup> and *ifnar1*<sup>−/−</sup>) within the same uterus.

The researchers found that the titer of virus replication was higher in the placenta of *ifnar1*<sup>−/−</sup> than *ifnar1*<sup>+/?</sup>. Only *ifnar1*<sup>+/?</sup> fetuses were resorbed after ZIKV infection during early pregnancy; *ifnar1*<sup>−/−</sup> littermates continued to develop. IFNAR

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