SUPPLEMENTAL MATERIAL

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Figure S1.  **ZIKV infection in human trophoblasts.** (A) Western blots for LC3-I/II show enhanced autophagy flux upon ZIKV infection at 6 hpi. ZIKV-infected JEG-3 (multiplicity of infection 0.1, 2 h) were cultured for 6 h and harvested. Baf A1 was applied for 30 min before harvesting to monitor autophagic flux. Images represent data from four independent experiments. (B) Cell viability assays of uninfected JEG-3 cells cultured in medium supplemented with indicated autophagy modulators (at same dosage as described in Fig. 1F) for 48 h; n = 4 for each group. Data depict mean ± SEM. *, P < 0.05; ns, not significant (ANOVA with a Dunn’s multiple-comparison test). Rap, rapamycin.

Figure S2.  **ZIKV infection induces autophagic activity in mouse placentas.** (A) Immunoblot and quantification of LC3 and p62 show increased autophagic activity in mouse placentas (at E14.5) infected with ZIKV compared with uninfected controls. GAPDH, a loading control; n = 3–5. Data depict mean ± SEM. *, P < 0.05, Mann-Whitney test. (B) Representative immunohistochemical staining of p62 in WT placentas with or without ZIKV infection at E14.5. Images represent data from five independent dams. Bars, 100 µm.
Figure S3.  **HCQ treatment reduces placental and fetal ZIKV infection specifically via autophagy.** (A) Litter size at E14.5 with or without HCQ treatment; n = 6 per group. Results represent mean ± SEM, three independent experiments. ns, not significant, Mann-Whitney test. (B) Representative ISH images of ZIKV RNA in indicated maternal uterine decidua. Bars, 100 µm. (C and D) Pregnant HM female mice were treated with HCQ (40 mg/kg/day via intraperitoneal route) or DMSO as a mock control from day +1 post–ZIKV infection (E10.5) to E14.5. Viral burden of ZIKV in placentas (C) and fetal head (D) were measured by quantitative RT-PCR; n = 8 from three independent dams. ns, not significant, Mann-Whitney test.