United States, are now in the unique position of providing care to both pregnant women with locally-transmitted and travel-associated ZIKV infections. This study provides data regarding the testing and pregnancy outcomes of women with laboratory evidence of ZIKV infection in pregnancy.

Methods. A retrospective chart review was conducted using laboratory records of ZIKV testing (PCR and IgM) completed from January through December 2016 at multiple tertiary care centers located in Miami-Dade County. Testing was based on CDC guidelines at time of testing, leading to heterogeneity in tests performed. Data was extracted from charts of women with positive ZIKV PCR in serum and/or urine or positive ZIKV IgM with confirmatory, pending, or insufficient PRNT results. Routine obstetrics parameters and the presence of fetal or neonatal abnormalities were recorded.

Results. Of the 2327 pregnant women screened for ZIKV, 88 (3.8%) screened positive with PCR and/or IgM in serum or urine. Of those women with positive ZIKV testing, 53 (60%) had no documented ZIKV symptoms and 40 (45%) had no known travel history outside of Miami-Dade County during their pregnancy. Sixty-six women had antenatal ultrasounds, 14 (21%) of which ever had a head circumference or biparietial diameter measurement less than the third percentile, but none showed evidence of intracranial calcifications. Fifty-four women with positive testing have delivered: 46 at term and 8 preterm. Fifty-four infants have been born to women with positive ZIKV testing; 2 infants (1.98%) had documented congenital abnormalities. One infant was born with clinically-defined microcephaly (1.9%) and intracranial calcifications and the other had only intracranial calcifications. Ninety-four positive IgM tests were sent to the CDC for confirmatory plaque reduction neutralization testing (PRNT). 49 PRNT tests returned positive (ZIKV titer ≥10), while 28 returned negative (ZIKV titer < 10), representing a false-positive rate of 30.4%.

Conclusion. As this epidemic persists, data from this unique cohort of pregnant women with both local and travel-associated ZIKV exposure contributes to the growing knowledge base regarding implications of ZIKV in pregnancy.

Disclosures. **All authors:** No reported disclosures.

1783. Environmental and Climatic Risk Factors for Zika and Chikungunya Virus Infections in Rio de Janeiro, Brazil, 2015–2016

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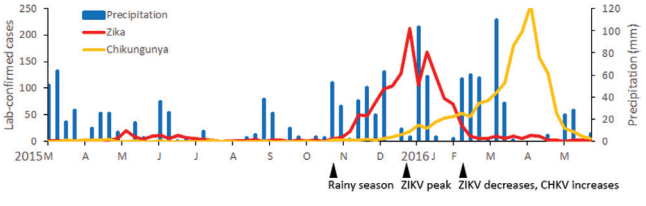
Background. The objective of the present study was to identify drivers of the ZIV epidemic in the state of Rio de Janeiro to predict where the next hotspots will occur and prioritize areas for vector control and eventual vaccination once available.

Methods. To assess climatic and socio-economic drivers of arbovirus epidemics, we mapped rainfall, temperature, and sanitation infrastructure in the municipalities where individuals with laboratory confirmed cases of arboviral infection resided using our spatial pattern risk model.

Results. From March 2015 to May 2016, 3,916 participants from 58 municipalities in the state of Rio de Janeiro were tested for dengue, Chikungunya (CHKV), and ZIKV by RT-PCR and enzyme immunoassays. During the same period, 69,256 suspected cases of dengue, CHKV, and ZIKV were reported to the Rio Health Department, including 23,983 of dengue, 44,572 of ZIKV, and 701 of CHKV. Laboratory confirmed cases included 29 cases (0.7%) of dengue, 1,717 of ZIKV (43.8%), and 2,170 of CHKV (55.4%). Rains in Rio began in October 2015 and were followed one month later by the largest wave of the ZIKV epidemic (Figure 1). ZIKV cases markedly declined in February 2016, which coincided with the start of a CHKV outbreak. Rainfall predicted ZIKV and CHKV in Rio with a lead-time of 3 weeks each time. Social and environmental variables predicted the number of cases. The temporal dynamics of ZIKV and CHKV in Rio de Janeiro are explained by the shorter incubation period of the viruses in the mosquito vector; 2 days for CHKV vs 10 days for ZIKV.

Conclusion. The association between rainfall and ZIKV reflects vector ecology, as the larval stages of *Aedes aegypti* require pools of water to develop. Rainfall in October 2015 would have produced such pools resulting in increased mosquito abundance likely contributing to the ZIKV epidemic in humans the following month. The decrease in ZIKV in February 2016 and the increase in CHKV likely arose due to within-vector competition. The Pan American Health Organization's ZIKV Strategic Plan states that controlling arboviruses requires mapping their social and environmental drivers. Our findings contribute to such control efforts.

Figure 1. Lab-confirmed cases of ZIKV and CHKV per week in the state of Rio de Janeiro, March 2015 to May 2016.



Disclosures. **All authors:** No reported disclosures.

1784. Differential Neuronal Susceptibility and Apoptosis in Congenital Zika Virus Infection

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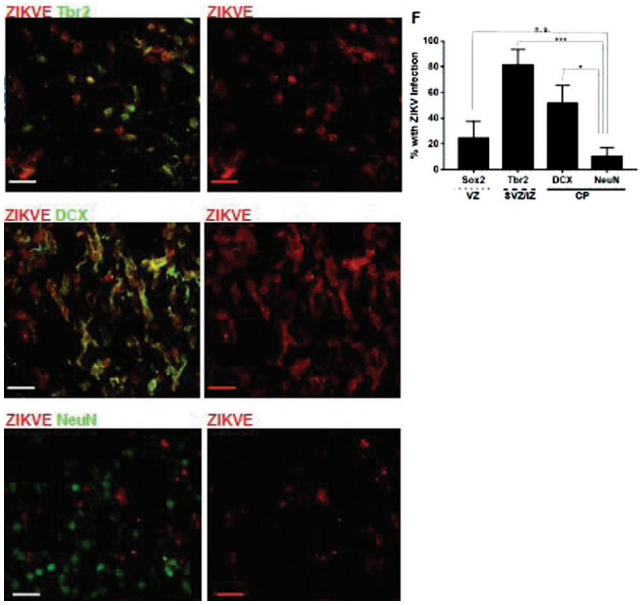
Background. Zika virus (ZIKV) infection during pregnancy may result in severe neurologic injury to the fetus. The mechanisms by which ZIKV injures fetal brain are not fully characterized. Although cell culture and animal models shed valuable insight into pathogenesis, they do not fully recapitulate human disease.

Methods. To characterize the mechanism of ZIKV-induced human brain injury, we performed immunolabeling on brain tissue from a 20-week fetus with intrauterine ZIKV infection. Formalin-fixed sections of brain tissue were co-immunostained with ZIKV envelope antibody, as well as neuronal and non-neuronal lineage cell markers to assess infection within populations. Apoptosis was assessed by quantifying activated caspase 3 -positive staining cells. Minimum 3–5 random microscopic fields per brain region were photographed and quantified in an automated fashion using the ImageJ Cell Counter plug-in. GraphPad Prism and Microsoft Excel software were used for data analysis.

Results. ZIKV demonstrated a wide range of neuronal and non-neuronal tropism. However, infection rate was highest in Tbr2+ - Intermediate Progenitor Cells (IPC; 81.4±12%) and DCX+ Immature Neurons (IN; 51.5±13.9%), followed by SOX2+/ Nestin+ Neural Precursor Cells (NPC; 26.6±13.4%). NeuN+ Mature Neurons had the lowest frequency of infection (MN; 10.0±7.0 %) (Figure). Apoptosis was observed in both infected and uninfected bystander cortical neurons. A high infection frequency was also observed in non-neuronal cells (astrocytes, microglia, macrophages, lymphocytes).

Conclusion. Our study provides valuable insights into ZIKV pathogenesis in the fetus; it is the first to demonstrate differential infectivity/susceptibility of neuronal lineage cells to ZIKV, and evidence of apoptosis in and around these cells. The high frequency of ZIKV+ IPC and IN implies that that infection can be supported until the immature stage of neuronal differentiation. The resistance of mature neurons to ZIKV infection may also explain why ZIKV infection in the third trimester poses less risk of microcephaly in infants. The high infection rate of non-neuronal cells also suggests potential contribution of immune-mediated mechanisms of brain injury in the setting of congenital ZIKV infection.



Disclosures. **All authors:** No reported disclosures.

1785. Risk Factors Associated with Persistence of Zika Virus Nucleic Acid in Serum and Semen

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