Table 3. Multivariate analysis of 30-day infection-related mortality

	OR	p value	95% CI	
Replacement < 2 days	5.908	0.031	1.176	29.679
Hematological malignancy	3.038	0.281	0.403	22.898

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

1779. Prevalence of and Factors Associated with *Clostridium difficile* Co-infection Among Patients with Candidemia, United States, 2014–2016

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Background. Candidemia and *Clostridium difficile* infection (CDI) are two common healthcare-associated infections (HAIs) and share risk factors such as antibiotic use and prolonged hospitalization. CDI and CDI treatment disrupt gut microbial diversity, allowing *Candida* overgrowth and translocation to the bloodstream. We describe CDI co-infection among patients with candidemia.

Methods. Population-based surveillance for candidemia was conducted through CDC's Emerging Infections Program during 2014–2016. A case of candidemia was defined as a blood culture positive for *Candida* species collected from a surveillance area resident. Demographic and medical information, including occurrence of CDI was collected. We defined co-infection as CDI within 90 days of candidemia and performed bivariable analysis to assess factors associated with co-infection.

Results. Among 2129 cases of candidemia, 190 (9%) had CDI co-infection; 116 (5%) had CDI in the 90 days before candidemia (median: 10 days) and 60 (3%) had CDI following candidemia (median: 8 days). The median age of those with CDI-candidemia co-infection was 61 years and 100 (53%) were male. Compared with candidemia alone, the odds of CDI-candidemia co-infection was significantly greater for patients of black race (OR 1.41, 95% CI 1.05–1.90), those with diabetes (OR 1.68, 1.24–2.27), pancreatitis (OR 1.91, 1.01–3.61), or solid organ transplant (OR 4.15, 2.09–8.22). Those with co-infection had higher odds of certain healthcare exposures: hemodialysis (OR 2.27, 1.57–3.28), hospital stay in the past 90 days (OR 1.9, 1.37–2.64), ICU admission in the past 14 days (OR 1.78, 1.20–2.66), and central venous catheter (CVC) at the time of candidemia (OR 1.71, 1.19–2.46). There were no significant differences in 30-day mortality or in type of *Candida* species, although *C. parapsilosis* was less common in the co-infection group (8% vs. 13%).

Conclusion. Nearly one in ten patients with candidemia also had CDI co-infection. Black race, certain underlying conditions, hemodialysis, previous hospitalization, ICU stay, and the presence of a CVC were associated with co-infection. Clinicians should be vigilant for coinfection of CDI and candidemia, particularly in situations with associated risk factors.

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1780. Routine Cryptococcal Antigen Screening in Solid Organ Transplant Recipients: Is it Time to Save Lives and Money?

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Session: 216. The Fungus Among-us – Clinical Advances
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Background. Cryptococcosis affects 1 in 270 solid organ transplant (SOT) recipients with high mortality. In HIV-infected patients, cryptococcal antigen (CRAG) is detectable in blood weeks to months before symptomatic infection and screening is recommended. No screening guidelines exist for SOT recipients.

Methods. We performed a cost-effectiveness analysis of CRAG screening amongst SOT recipients. We estimated costs of screening from Medicare reimbursement of $16.49 for CPT 87899 (Infectious agent antigen detection by immunoassay). We determined the number at risk from a large cohort of 42,634 adult SOT recipients from ICD-9 CM billing data from HCUP State Inpatient Databases of Florida (2006–2012), New York (2006–2011), and California (2004–2010). Cost of screening was compared with the cost of inpatient hospitalization.

Results. Among 42,634 adult SOT recipients, 158 (0.37%) developed cryptococcosis at a median time of 15.5 months (range 0.1 -80) after transplant. During the 43 month follow-up, there was approximately 2.5% annual mortality. The estimated cost of hospital care for cryptococcal meningitis per person is approximately $70,000 in 2016 with current explosive cost of flucytosine at ~$29,000 per 2 weeks. Thus, the total estimated cost of hospital care in the cohort would be $11.0 million in 2016. In comparison, the cost to screen all 42,634 SOT recipients every three months would be $8.8 million. If CRAG screening could detect 75% of asymptomatic cryptococcal antigenemia prior to symptomatic disease requiring prolonged hospitalization, it would be approximately cost neutral ($11.5 million), and even cost saving if above 80% of hospitalizations are averted. Alternatively stated, for every one hospitalization avoided, 4245 persons could be CRAG screened for similar cost and likely better outcome.

Conclusion. Assuming the ability of routine screening to identify 75% of patients who would develop invasive cryptococcosis; CRAG screening every 3 months among SOT recipients likely would be at least cost neutral to the healthcare system. Antecedent duration of cryptococcal antigenemia prior to symptomatic disease in Non-HIV/SOT cohorts to inform optimal screening intervals should be further studied. Prospective SOT cohorts should validate this approach to save lives in a cost-effective manner.

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1781. Galidesivir, a Direct-Acting Antiviral Drug, Abrogates Viremia in Rhesus Macaques Challenged with Zika Virus

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Background. Zika virus (ZIKV) was first isolated from a sentinel rhesus monkey in 1947. ZIKV infection in humans is associated with serious neurological and reproductive complications. No antiviral or protective vaccine is yet available. Galidesivir an adenosine analog is a potent viral RNA-dependent RNA polymerase inhibitor with demonstrated broad-spectrum antiviral activity.

Methods. We have conducted four pre-clinical studies in rhesus macaques to assess the safety, antiviral efficacy and dosing strategies of galidesivir against ZIKV infection. Collectively, we have challenged 70 rhesus macaques by various routes using $1x10^5$ $TCID_{50}$of a Puerto Rican ZIKV isolate. We have evaluated galidesivir therapy administered via IM injection as early as 90 minutes and up to 72 hours after subcutaneous (SC) ZIKV challenge, and as late as 5 days after intravaginal (IVAG) challenge. In these studies, we evaluated the efficacy of a range of loading and maintence doses of galidesivir. The highest dose evaluated has been a loading dose of 100mg/kg BID followed by a maintenance dose of 25mg/kg BID for nine days. We followed multiple endpoints, including ZIKV RNA levels in plasma, urine, saliva, and cerebrospinal fluid. Immune activation, complete blood counts, chemistries and galidesivir pharmacokinetics were also monitored.

Results. Galidesivir was well-tolerated in all studies. All untreated controls developed high-level plasma viremia, and had readily detectable ZIKV RNA in CSF, saliva and urine post-infection. Animals treated in the first 24 hours after SC ZIKV challenge did not develop plasma viremia and were either negative or had significantly reduced ZIKV RNA in bodily fluids. Animals treated with galidesivir later (up to 72 hours) were partially protected; they had detectable plasma ZIKV RNA, but the onset was delayed and/or magnitude significantly reduced compared with controls. Animals infected IVAG were protected by galidesivir treatment up until day 5 after infection, with no blood viremia and significant reductions in ZIKV RNA in the CSF as compared with controls.

Conclusion. Galidesivir dosing in rhesus macaques was well-tolerated and offered significant protection against ZIKV infection. These results warrant continued study and clinical evaluation.

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1782. Outcomes of women with laboratory evidence of Zika infection in pregnancy

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Background. Zika virus (ZIKV) infection in pregnancy is a global health concern. With onset of local transmission, obstetricians in Miami-Dade County, FL,

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.

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

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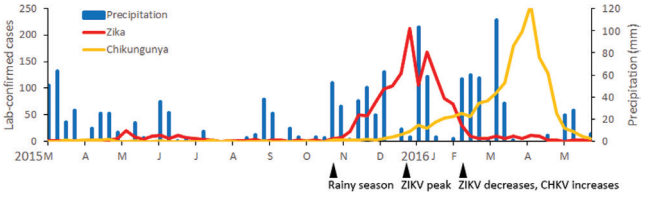
Background. The objective of the present study was to identify drivers of the ZIV epidemic in the state of Rio de Janeiro to predict where the next hotspots will occur and prioritize areas for vector control and eventual vaccination once available.

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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Session: 217. Zika - A to Z

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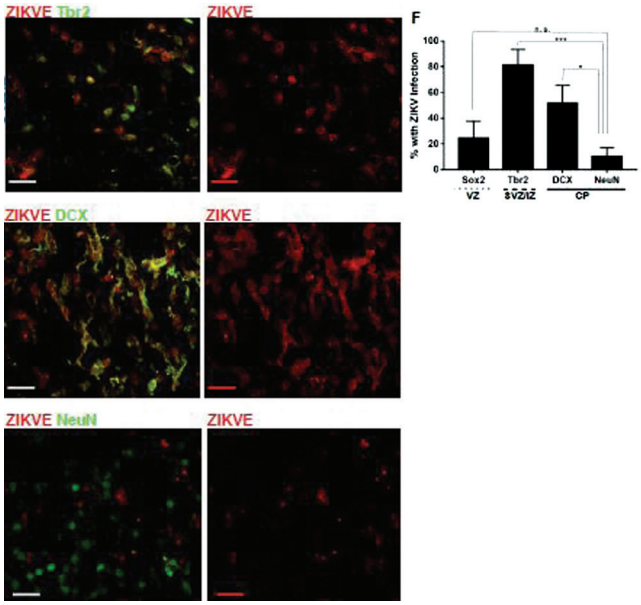
Background. Zika virus (ZIKV) infection during pregnancy may result in severe neurologic injury to the fetus. The mechanisms by which ZIKV injures fetal brain are not fully characterized. Although cell culture and animal models shed valuable insight into pathogenesis, they do not fully recapitulate human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