**BACKGROUND**

“Congenital Zika virus infection causes birth defects and developmental deficits. While the features of the immune response that determine the extent of fetal harm is not clearly defined, prolonged viremia during pregnancy may cause more fetal harm. Humans have prolonged ZIKV viremia and vertical transmission even though they produce neutralizing antibodies. This observation means that other features of the antibody response besides neutralization play a role in control of maternal ZIKV infection. To further understand the association between viremia duration and antibody response, we characterize the magnitude of the ZIKV-specific IgG response”

**METHODS**

ZIKV infection of pregnant macaques

- Plasma and Serum preparation
  - Plasma
  - Serum
- Virus quantification by qRT-PCR
- IgG response whole virion ELISA

**RESULTS**

- **Maternal viremia duration at 25-30dpi**
  - Graph showing the relationship between maternal viremia load and duration.
  - Equation: $R^2 = 0.79$

- **Maternal IgG response**
  - Graph showing the antibody response over time.

**CONCLUSIONS**

- IgG seroconversion occurs at 10dpi
- ZIKV-specific IgG response varies across animals.
- ZIKV-specific IgG response is positively associated with its viremia duration.

**ADDITIONAL KEY INFORMATION**

Future Plans:

- Neutralizing antibody assays to understand the relationship between binding antibody responses and viremia duration.
- Analysis of IgM and IgA response to understand breadth of antibody response

Acknowledgements

EC50 = reciprocal of the serum dilution for which virus binding is reduced by 50%

IgG antibody titers increases 7 days post infection and reaches peak at 40-60dpi.
Magnitude of IgG antibody response varies across animals.
Viremia duration is positively associated with ZIKV-specific IgG titers at 25-30 dpi