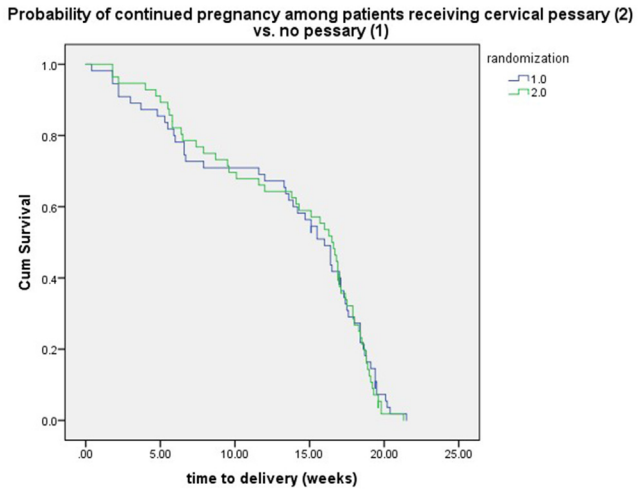
age at delivery, birth weight and composite neonatal outcome (Table).

CONCLUSION: Our study showed that treatment with a cervical pessary did not prevent PTB in women with singleton gestations with a TVU CL $\leq$ 25 mm at 18^0 - 23^6 weeks and without a prior spontaneous PTB. Though study recruitment was stopped before we reached our enrollment goal, our findings are consistent with the recent studies that failed to show efficacy of pessaries in similar clinical settings.

Outcomes according to study group

	PessaryN=56	No pessaryN=55	RR (95% CI)	P-value
PTB<37 weeks	24 (43%)	23 (42%)	1.02 (0.70,1.48)	
PTB<34 weeks	20 (36%)	17 (31%)	1.11 (0.76,1.62)	
PTB<28 weeks	11 (20%)	14 (25%)	0.84 (0.52,1.37)	
PTB<24 weeks	2 (4%)	5 (9%)	-	
Mean GA at delivery (weeks)	34.7 ±5.7	34.4 ±6.5		P=0.83
Birth weight(g)	2359 ±1073	2404 ±1143		P=0.83
Composite neonatal outcome	14 (25%)	16 (30%)	0.90 (0.58, 1.39)	



7 Potential mechanisms of contemporary strain Zika virus replication in human placental trophoblasts

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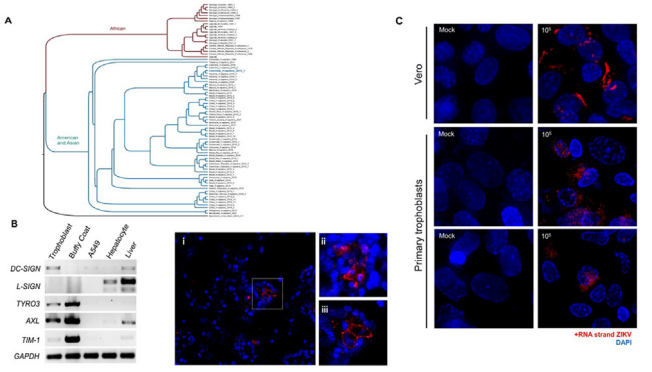
OBJECTIVE: Zika virus (ZIKV) is an emerging mosquito-borne flavivirus. A causal link explaining the delay between maternal symptoms and fetal infection is missing, and why prior African outbreaks were not associated with fetal malformations is unknown. In this study, we sought to answer these questions using state-of-the-science approaches with a single passage contemporary ZIKV strain and infection of high purity trophoblasts from uninfected donors.

STUDY DESIGN: ZIKV-FLR was isolated by inoculating *A. albopictus* C6/36 mosquito cells with serum from a non-pregnant subject infected in Barranquilla, Colombia. Subsequent deep sequencing on the Illumina platform was performed, and alignments to all 77 sequenced genomic strains was made. We isolated n=20 uninfected human primary trophoblasts, and examined expression of putative ZIKV cell entry receptors. We documented intracellular infection in trophoblasts using single molecule RNA FISH to both negative and positive viral strands. Finally, we examined the potential role of miRNAs in modulating trophoblast infection.

RESULTS: Phylogenetic trees were generated from all currently available complete ZIKV sequences (n of 77; **Fig 1A**). The resultant phylogenetic tree demonstrates phylogenetic delineation noted between African strains (non known to cause microcephaly) and the current Americas and recent Asian strains. Moreover, primary human trophoblasts express putative cell entry receptors for ZIKV (**Fig 1B**) prior to infection with ZIKV. Upon co-culturing with a current strain of ZIKV, we observe intracellular localization of ZIKV as evidenced by single molecule FISH (**Fig 1C**). Finally, ZIKV infection is associated with a significant diminution of the ssRNA-ligand sensing miRNA, mir21 (0.7 fold lower, $p<0.01$) but not the C19 cluster miRNA species.

CONCLUSION: Contemporary ZIKV strains are genomically distinct from historic epidemic strains, mirroring their association with microcephaly and fetal malformations. Placental trophoblasts express putative ZIKV entry receptors, and cytoplasmic replication can visualized by single molecular RNA FISH. Select placental miRNAs (mir-21) are significantly and specifically down-modulated following ZIKV infection. We speculate that these findings are of potential mechanistic interest to ZIKV perinatal pathogenesis.



8 Whole exome sequencing in the evaluation of fetal structural anomalies: A prospective study of sequential patients

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OBJECTIVE: Small studies have reported the use of whole exome sequencing (WES) in the prenatal evaluation of fetal structural anomalies and have reported pathogenic variant rates of 10-30%. These studies however have all used selected patients that were felt to have a high likelihood of having a genetic etiology. We sought to evaluate the incremental value of WES in routine prenatal diagnosis including all structural anomalies.

STUDY DESIGN: Under an IRB protocol, all sequential patients with a fetal structural anomaly were offered WES as part of the fetal genetic evaluation. Those having diagnostic prenatal testing had WES, karyotype, and chromosomal microarray done on amniotic fluid or CVS and those not having PND had cord blood obtained at birth for testing. All results were returned to the patients.