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Recommended Citation

Puri, Tanvi, "Reproductive Tissues in Pregnant MiceShow Sustained Intrinsic CircadianOscillations and Clustering of PhasePrior to the Onset of Labor" (2018). *Spring 2018*. 99.
https://openscholarship.wustl.edu/wushta_spr2018/99

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Reproductive Tissues in Pregnant Mice Show Sustained Intrinsic Circadian Oscillations and Clustering of Phase Prior to the Onset of Labor

Tanvi Puri

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Preterm birth is the leading cause of mortality in newborns and infants. In 2016, 9.54% of live births in the U.S. were premature. However, approximately half of all preterm births have no known cause. Previous studies involving pregnant shift workers have reported a correlation between disrupted circadian rhythms, and premature labor-onset and birth. This led us to hypothesize that circadian rhythms in the mother play a role in preterm birth. Prior studies have revealed that reproductive tissues in mice have circadian expression of mPeriod2, a mammalian clock gene. We used knock-in mice with the firefly luciferase gene fused to the open reading frame of the Period 2 gene (PER2::LUC) to test whether circadian rhythms intrinsic to reproductive tissues change through the course of normal pregnancy. We recorded bioluminescence for five days from uterine, cervical, and placental tissue harvested at pregnancy days 9.5 (P9.5), P11.5, P15.5, or P18.5. We found all cultured tissues were circadian at all stages of pregnancy. We found that the intrinsic circadian period and amplitude remained constant over the course of pregnancy in the uterus, cervix, and placenta. The time of peak PER2 expression in P18.5 uterine and placental tissue reliably peaked in early subjective day. Tissues collected at all other ages had less reliable phases. We conclude that pregnancy does not change the intrinsic circadian period or amplitude of maternal reproductive tissues, but events around the day of delivery coordinate the time of peak circadian expression in uterine and placental tissue. These changes may underlie the initiation of normal parturition.