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An Investigation of the Roles of Maternal and Fetal Clocks in Determining Gestation Length

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An Investigation of the Roles of Maternal and Fetal Clocks in Determining Gestation Length

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Preterm birth is one of the leading causes of infant mortality in the United States. Shift workers are especially prone to preterm birth, especially those that consistently engage in rotating night shift work, possibly due to their constantly changing schedules that disrupt their circadian rhythms. Circadian rhythms are regulated by a group of clock genes. Per2 (Period 2) is a core clock gene that helps regulate functions about a 24-hour period. A family in Utah has a mutation in the Per2 gene (serine to glycine) that leads to an advanced sleep phase disorder and causes one to be an extreme "early bird." Members of this family consistently go to bed around 7 p.m. and wake at 2 a.m. the human Per2 mutant gene (hPer2) was inserted into a mouse strain to create a mouse model to mimic the human phenotype, the hPer2 short mouse. Previous studies have shown that the circadian periods of hPer2 short mice (21.6 hours) have shorter gestation lengths (17.5 days) compared to wild-type mice (with 24 hour circadian periods and gestation lengths of 19.5 days). We aimed to determine if the circadian period of the mother or fetus drives the timing of the birth. *In vitro* fertilization techniques have an increased risk of shortened gestation length in humans, so we first conducted control studies using wild-type embryos from wild-type donor dams and wild-type recipient dams. We found no significant difference in gestational length between naturally bred dams (19.5±0.257 days, n=13) and dams that received embryo transplants (19.67±0.155, n=7). This result suggests that the IVF protocol does not shorten pregnancy length in mice. Further studies will include transferring wild-type embryos into short dams and short embryos into wild-type dams to better understand whether fetal or maternal clocks determine gestation length.