tionship between CL and time to birth (p=0.03), but this effect was null after controlling for maternal age. In logistic regression models, CL did not significantly predict PTB < 24, < 28, < 35, or < 37 weeks. Stratifying CL by 25-29 mm versus 30 mm or greater also had no significant predictive value for these 4 preterm birth cutoffs.

**CONCLUSION:** Women at high risk for recurrent preterm birth, but whose cervical length at < 23 weeks′ gestation remains ≥ 25 mm are still at increased risk of recurrent PTB (16% delivered < 35 weeks); however, CL measured before 23 weeks, whether considered on a continuum or stratified to consider CL′s near the 25 mm cutoff (25-29 mm), does not predict PTB.

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## 17 Epithelial protection and repair: the role of trefoil factor 1 in the mouse cervix through pregnancy

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**OBJECTIVE:** The cervix remodels throughout pregnancy in preparation for parturition and includes stages of softening, ripening and dilation. Understanding the molecular processes that regulate each phase is critical for identification of preterm birth risk factors due to cervical malfunction. Trefoil factor 1 (Tff1) is a member of a family of secreted proteins that play an important role in the protection and restitution of the gastrointestinal mucosal epithelium. We previously determined a temporal and robust mRNA expression of Tff1 in the pregnant cervix with maximal expression prior to birth and rapid decline postpartum. Mice deficient in Tff1 will be used to understand the role Tff1 may play in barrier protection and repair of cervical epithelia during pregnancy.

**STUDY DESIGN:** Cervical Tff1 protein abundance and cell localization was evaluated by western blotting and immunohistochemistry. The role of Tff1 in epithelial cell function during cervical remodeling was assessed by morphological assessment of Tff1−/− cervices and evaluation of genes normally expressed in the epithelia during cervical ripening.

**RESULTS:** Protein expression of Tff1 mirrors mRNA expression and is confined to differentiated squamous epithelia. Morphology of the Tff1−/− cervix appears normal before and during cervical ripening. Gene expression of hyaluronan synthase 2, keratin 16, and serine protease inhibitor kazal type 5 were suppressed on gestation d15 and d18 compared to wild type controls but recovered postpartum. Claudin 2 had elevated expression in Tff1−/− mice on day 18 compared to wild types, but claudin 1 had normal expression.

**CONCLUSION:** These studies confirm the expression of Tff1 in the cervical squamous epithelia and suggest that some but not all aspects of barrier regulation are compromised in the absence of Tff1. The altered barrier properties of the cervical epithelia in Tff1 null mice suggest that these mice may be a useful model to evaluate susceptibility to infection mediated preterm birth upon epithelial insult when there is a reduced capability to repair cervical epithelia.

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