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Female sex hormones enhance Neisseria gonorrhoeae colonization on the human endocervix

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Background

Gonorrhea, caused by Neisseria gonorrhoeae (GC), is the second-most common sexually transmitted infection worldwide. While GC infections in women are often asymptomatic, they can lead to pelvic inflammatory disease and fallopian tube damage. However, the mechanism(s) underlying various clinical outcomes remain unclear. Due to their role in regulating the mucosal surface of the female reproductive tract during the menstrual cycle, the sex hormones estradiol and progesterone potentially contribute to the infection process and clinical outcomes.

Aim/Methods

Using the human cervical tissue explant model, we examined the effect of different hormone conditions on GC infection in the human cervix, the gate of the female reproductive tract. Cervical tissue explants were treated with either no hormones or biologically relevant concentrations of hormone (50 nM estradiol or 50 nM estradiol together with 100 nM progesterone) to mimic the menstrual, proliferative, and secretory phases of the menstrual cycle. This hormone treatment was carried out 48 hours prior to infection with GC and continued throughout the 24-hour infection period.

Results

We found that hormone treatment increased GC colonization along the surface of the endocervix. Estradiol treatment alone increased the number of GC colonies on the luminal surface of the endocervix, while a combination of estradiol and progesterone increased both the colony number and colony size.

Conclusions

These data support the hypothesis that variations in hormonal conditions in the female reproductive tract can alter the interactions between GC and the cervical epithelium, potentially driving different clinical outcomes.