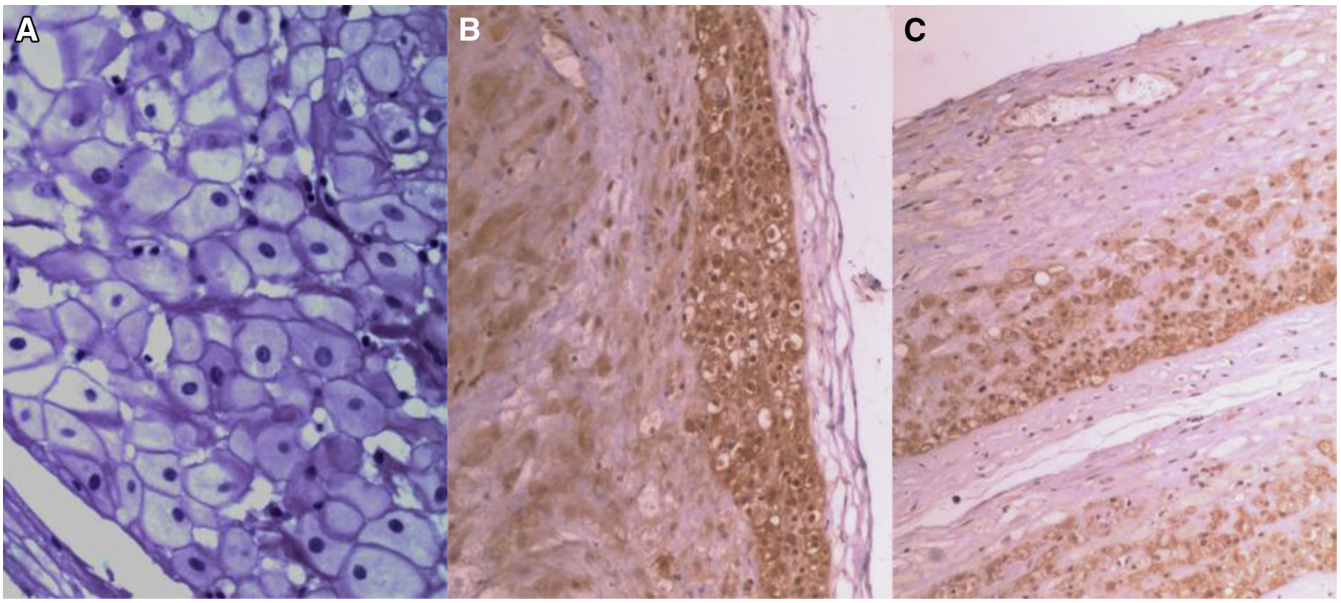
**FIGURE**
**Inflammatory cells, P2X3-positive cells, and de-layering of P2X3-positive layer of cells**




**A,** Sheets of inflammatory cells obliterate the membranes adjacent to the site of membrane rupture in a rolled membrane preparation (hematoxylin and eosin, magnification, ×200). **B,** A discrete layer of P2X3-positive cells in maternal chorion after cesarean delivery at term **(B)** (anti-P2X3, magnification, ×100). **C,** De-layering of the P2X3-positive layer of cells following spontaneous labor at term (anti-P2X3, magnification, ×100).

*P2X3*, P2X purinoceptor 3.

*Zhou. Purinergic, P2X3 positive cells in amniotic membranes. Am J Obstet Gynecol 2019.*

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## REPLY



We appreciate the thoughtful comments of Dr Quinn and colleagues regarding our recent article.[1] In response to their first question, we demonstrated only the results in vitro cell or tissue explants. The cause-effect is an important question; however, our model is not designed to answer a cause-effect relationship but primarily to show how infection can exaggerate a response with thrombin, a known agent that weakens membranes. Depending on the specific type of underlying pathology, thrombin may come first or in other cases infection may arise first.

It is also difficult to predict that denervation of vaginal epithelium is predisposing ascending infection. Vaginal epithelial cell exfoliation has been reported as a mechanism of ascending infection with Group B streptococcus but unsure that can be the reason for genital mycoplasma ascension.

Finally, congratulations on your new discoveries. ■

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The author reports no conflict of interest.

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