

COMMENT

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The role of the vaginal microbiome in pregnancy loss and preterm birth: a commentary

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Abstract

Background Eubiosis or dysbiosis of the vaginal microbiome may influence the rate of pregnancy loss and preterm birth, the major cause of neonatal and perinatal mortality worldwide.

Methods This was a comparison of two vaginal microbiome studies; one a cohort study and the other a multinational randomised controlled feasibility study. Both studies used cultivation-independent molecular microbiological techniques that together have implications on the risk of pregnancy loss and preterm birth in association with vaginal dysbiosis.

Results The cohort study identified a risk-associated vaginal microbiome signature in association with early pregnancy-loss that comprised an increase in the relative abundance of potentially dysbiotic organisms such as *Lactobacillus iners*, *Sneathia* and *Prevotella* spp and a concomitant decrease in the abundance of eubiotic microorganisms such as *Lactobacillus crispatus*. Convergent evidence across the two studies demonstrated that a synbiotic intervention was able to shift the vaginal microbiome from the signature demonstrated by Skaft-Holm et al., to a decrease in the abundance of *Prevotella*, *Gardnerella* and *Atopobium* spp, while simultaneously increasing the abundance of eubiotic vaginal *Lactobacillus* spp.

Conclusions We concluded that there is convergent evidence across the two studies which might otherwise have gone unnoticed. While neither study was powered to demonstrate clinical endpoints and did not establish causal relationships between microbiome modulation and pregnancy outcomes, when administered regularly, vaginal commensal probiotics in ice-cream were effective in optimizing both the vaginal and intestinal microbiota in pregnant women at increased risk of pregnancy loss, particularly preterm birth. This emphasises the need for adequately powered trials to test whether early pregnancy vaginal microbiome modulation can improve clinical outcomes.

Keywords 16S, Food fortification, Ice cream, Lactobacilli, Microbiome, Miscarriage, Preterm birth, Pregnancy loss, Probiotics, Synbiotics, Vaginal microbiome

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Background

Worldwide, spontaneous preterm labour (SPTL) that leads to preterm birth (PTB) is the major cause of death and handicap in neonates and a significant financial burden on healthcare [1–3]. A significant proportion of SPTL and PTB is due to infection and historically, the earlier in pregnancy at which SPTL and PTB occur, the more likely this is to be due to infection [4] and the greater is the risk of perinatal death and neurodevelopmental handicap [5, 6]. With the advent of new technology using cultivation-independent, molecular-based techniques as part of the Human Microbiome Project rather than traditional cultivation-dependent techniques, there has been markedly influential new information with respect to human vaginal eubiosis or dysbiosis and its influence on the rate of pregnancy loss and PTB [7]. The human vaginal microbiome has a stable core microbiome together with a variable microbiome that is influenced by the host lifestyle, genotype, pathobiology, physiology, environment, immune response, and transient community members [8]. Accordingly, the microbiota of the gut and genital tract in women displays complex biological ecosystems that continuously communicate with each other and the crosstalk between these two ecosystems affects host physiological, immunological and metabolic homeostasis [9]. The evolutionary perspective of the microbiome is also important but is beyond the scope of this commentary. For those interested in the evolutionary perspective of the human microbiome and how this might apply to the prediction and prevention of PTB, the following reviews are recommended [10–12]. In this commentary, we highlight two vaginal microbiome studies that together have implications on the risk of pregnancy loss in association with vaginal dysbiosis assessed using such molecular microbiological technology.

Findings

The first of these studies was a randomised controlled feasibility study investigating the relationship between daily intake of dietary fibre-enriched ice-cream containing vaginal commensal probiotics (*L. crispatus*, *L. gasseri*, *L. jensenii*, *L. rhamnosus* GR-1) throughout pregnancy and the impact on the gut and vaginal microbiomes, in women at high risk of PTB [13]. Subsequently, we read with interest, an article by Skaft-Holm et al. on the role of the vaginal microbiome in involuntary pregnancy-loss [14]. This cohort study identified a risk-associated vaginal microbiome signature in early pregnancy-loss comprising an increase in the relative abundance of *Lactobacillus iners*, *Sneathia* and *Prevotella* spp concomitant with a decreased abundance of *Lactobacillus crispatus*. We feel that the earlier study complements the conclusions of Skaft-Holm et al. and demonstrates that a synbiotic intervention was able to shift the vaginal microbiome

from the signature demonstrated by Skaft-Holm et al., to a decrease in the abundance of dysbiotic *Prevotella*, *Gardnerella* and *Atopobium* spp. Simultaneously the abundance of eubiotic vaginal *Lactobacillus* spp was increased.

Conclusion

In the randomised controlled feasibility study [13], it was concluded that, when administered regularly, vaginal commensal probiotics in ice-cream were associated with favourable shifts in both vaginal and intestinal microbiota in pregnant women at increased risk of PTB. Although late involuntary pregnancy-loss and PTB represent distinct clinical entities, they are often considered part of a biological continuum that share infectious and inflammatory aetiologies. Together, the finding of these studies strengthen the biological case that: (i) vaginal communities skewed towards *L. iners* and dysbiotic bacterial vaginosis BV-associated anaerobes (*Prevotella*, *Sneathia*, *Gardnerella*, *Atopobium*, spp.) are associated with worse pregnancy outcomes such as miscarriage and PTB; (ii) targeted supplementation with eubiotic candidates for probiotics in ice-cream or perhaps other vectors, administered throughout pregnancy can shift the microbiome from a risk-like pattern of vaginal dysbiosis to one with eubiotic *Lactobacillus* spp with reduced abundance of gut BV-associated taxa and (iii) probiotic strategies that focus on *L. crispatus*, *L. jensenii*, and *L. gasseri* and suppression of BV-associated bacteria warrant further investigation to mitigate infection-mediated pregnancy loss and PTB.

We emphasise that neither study was powered to demonstrate clinical endpoints and did not establish causal relationships between microbiome modulation and pregnancy outcomes. In addition, while changes in the abundance of *L. iners* were observed, the primary effects of intervention were seen in other taxa, which may not be generalisable beyond the relatively homogeneous Danish/European population of the included cohorts. Nevertheless, we believe that there is convergent evidence across the two studies which might otherwise go unnoticed and emphasises the need for adequately powered trials testing whether early pregnancy vaginal microbiome modulation can improve clinical outcomes.

We recommend that future studies should validate these risk signatures across diverse populations, particularly comparing white, African American, Caribbean and African cohorts to distinguish the impact of genetics and social stressors on the vaginal microbiome. This approach is essential to determine if the findings reflect biological ancestry or environmental factors and may identify which interventions are applicable across wider population variants.

Abbreviations

BV	Bacterial Vaginosis
PTB	Preterm Birth
SPTL	Spontaneous Preterm labour

Author contributions

R.F.L. and J.S.J. conceived the commentary. All co-authors contributed to and approved the final manuscript submitted for publication.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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