Mini Review Article

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Micro Biome during Pregnancy and Preterm Birth

Abstract

Despite improvements in obstetric and neonatal care, preterm birth remains a major cause of newborn illness and mortality, which is a global health problem. The underlying etiology is complex and is still not fully understood. The intricate interactions between the vaginal micro biome throughout pregnancy and its link to preterm birth are thoroughly described in this paper. The understanding of the vaginal micro biota in both health and disease has advanced as a result of advancements in the study of bacteriology and the human micro biome.

Keywords: Preterm birth • Vaginal micro biota • Bacteriology • Preterm birth

Jeffery Alexander*

Department of Gynecology, Greenwich University, UK

*Author for correspondence: alexander@gu.ac.uk

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Introduction

Preterm Birth (PTB), a multi-aetiological illness state, is the leading cause of mortality in children under the age of five globally. It results in almost 1 million fatalities annually. At least one-third of these instances are thought to be caused by infection. While systemic maternal infection and colonization of the lower reproductive tract by well-known pathogens like Chlamydia trachomatis and Trichomonas vaginalis have long been linked to an increased risk of PTB, recent applications of molecular-based profiling methods have revealed new information regarding the part that microbe-host interactions during pregnancy play in determining PTB risk. In this review, we look at the evidence that connects high-risk PTB phenotypes to the host response and maternal micro biota makeup [1].

Profiling of microbial populations using culture and DNA analysis

In the late nineteenth and early twentieth century, bacteriology was essentially restricted to the study of germs that could be quickly isolated and cultured outside of the human body. Microscopy innovations, however, soon made it clear that many diverse microbial mor-photypes of a complexity that culture-based approaches alone failed to capture were colonising the niches of the human body. High-throughput DNA sequencing techniques, for example, have revolutionised how quickly and thoroughly we can characterise polymicrobial communities in the twenty-first century. This has also increased our understanding of the enormous diversity of bacteria, viruses, fungi, and archaea that live inside the human body. The total number of microorganisms found in a specific metagenomics and metataxonomics are two sequencing-based approaches that are frequently used to research the microbiota. Shotgun sequencing of the entire DNA in a sample yields the metagenome, which is a collection of genomes and genes from the individuals who make up a microbial community [2].

Nine "hyper-variable" sections (V1-V9) and nine "highly conserved" regions make up the bacterial 16S rRNA gene. By mapping the resulting sequences to existing 16S rRNA gene databases, PCR primers made to bind to the conserved regions enable amplification of the variable regions, which are typically diverse and distinctive, and facilitate classification of bacterial taxonomy to species, and in some cases strain level. Therefore, utilising metataxonomics to accurately identify bacterial species is subject to bias depending on the primer design, amplification region selection, and even the database to which the sequence reads are mapped. For instance, because certain of the 'conserved' portions of the 16S rRNA gene are not completely conserved, some bacterial DNA may only be preferentially bound by some PCR primers [3].

Vaginal micro biome in preterm birth

It is well believed that pathogen ascent from the vagina is a secondary cause of intrauterine infection that results in PTB. This idea is corroborated in humans by the similarities

between the bacterial species discovered in PTB cases' placentas and foetal membranes (amnion and chorion) and those discovered in the vagina. This idea is further supported by the fact that histological chorioamnionitis is most frequently seen at the site of membrane rupture in the lower portion of the uterus near the cervix and in twin pregnancies, where microbial invasion of the amniotic cavity and histological chorioamnionitis are most frequently seen in the presenting and first born twin [4].

A relationship between spontaneous PTB and decreased vaginal bacterial diversity was found in a study of a small sample of nulliparous African American women, albeit this was not statistically significant. A decrease in vaginal variety was linked to PTB in African American women, although a lack of specific Lactobacillus species did not connect to risk or protection from PTB in a cohort of Black women. African American and non-African American women differ from one another in terms of bacterial taxa and PTB, and this has recently been investigated. Again, even though there were less Lactobacillus species in the blood of African Americans, this was only a significant risk factor for PTB in White women. The discovery of particular bacterial taxa that were substantially linked to spontaneous PTB was also reported in this investigation [5].

Evidence of a preterm birth assisted with oral-placental micro biome axis

Another potential secondary pathway of invasion and infection that could result in PTB is the haematogenous dissemination of microorganisms and subsequent colonisation of the placenta. The oral cavity has been proposed as the origin of these germs, and mouse models have shown that pathogens can be transmitted from the mouth to the placenta. The discovery of typical oral pathogens, such as *Fusobacterium nucleatum, Dialister spp., Prevotella spp.*, and *Porphyromonas gingivalis*, in the placenta of women with periodontal disease, which is linked to an increased risk of PTB, provides additional support for an oral-placental axis involved in PTB. PTB and several bacterial species that were histopathogenic during pregnancy are not prevented by therapies that enhance periodontal health [6, 7].

Vaginal microbiota manipulation to alter PTB risk

It is not unexpected that many studies have looked at the effectiveness of antibiotics for the treatment and prevention of PTB given the extensive data that shows a connection between a specific vaginal microbiota composition and higher PTB risk. The findings of these trials have been wildly uneven and have largely been aimed at pregnant women with Bacterial Vaginosis (BV). This is partially caused by the variability of study designs (e.g., variations in treatment schedule, antibiotic kinds utilised, administration routes, patient cohorts chosen, and false indications). In any such future trial, there are a number of things that should be carefully taken into account [8]. Treatment of BV-positive women suggests that ascending infection or activation of the inflammatory system may have already started. Additionally, some medications, like as metronidazole, can lyse bacteria and release endotoxins, which are potent pro-inflammatory stimulators in gestational tissues and may thus exacerbate an inflammatory phenotype. Certain antibiotic therapies may be more efficient against commensal species like Lactobacillus but ineffective against infections linked to BV, accidentally enriching these organisms in the vaginal niche [9,10].

Conclusion

High-throughput DNA sequencing techniques have substantially improved our ability to investigate communities of microorganisms and have validated what was already known from classical microbiology based on culture. Although the existence of a physiologically necessary placental microbiome is questionable, it is nevertheless possible that some premature labours are caused by the haematogenous spread of organisms to the placenta and uterus. Studying how the gut microbiota affects pregnancy and whether or not gut microbial inflammation is associated with an increased risk of PTB will be difficult, but it has the potential to shift thinking. The vaginal microbiota is the source of the most compelling data linking the microbiome to premature birth.

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