Use of Crowd-sourced Requests to Inform a Logic Model Analysis of Group B Streptococcus as a Preventable Cause of Fetal/Perinatal Morbidity and Mortality

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Background
Despite progress, Group B Streptococcus (GBS), also known as Streptococcus agalactiae, continues as an important potentially preventable cause of fetal/perinatal (prenatal-onset and early-onset GBS sepsis [POGBSS and EOGBSS]) morbidity and mortality. Mortality includes stillbirth and preivable birth. Morbidity includes sepsis, SIRS, and brain injury, pneumonia, major organ damage as well as chorioamnionitis (IAI), placenta, deciduas, and puerperal infections. The epidemiology and pathophysiology is best studied in developed nations but appears more common in developing countries.

Objective/Goals
1. Analyze requests for information from parents of affected children over the internet (via www.groupbstrepternational.org) to inform creation of a logic model to identify and prioritize unresolved parental questions related to pregnancy-associated GBS pathogenesis, epidemiology, and primary, secondary, and “tertiary” prevention strategies.
2. We constructed a logic model analysis in order to compose and update a community and public-health based review of available information.
3. We created a standard of care model for evidence-based prevention strategies vs. GBS (POGBSS and EOGBSS).

Results
1. The most common categories of enquiries included:
   A. Operational issues
      What are important causes of failure of GBS prevention protocols?
      Why do many countries (UK, Northern Ireland) not implement evidence-based strategies?
   B. Clinical questions
      How can parents and providers recognize uncommon clinical presentations including fetal death (5B);
      “fetal distress” (fetal intrauterine hypoxemia/acidosis and abnormal “fetal monitoring”)
      What are ways to identify increased “risk of invasive disease and/or susceptibilities”?
      What are causes of late-onset GBS sepsis (LOGBSS)?
      Can breast milk be infected with GBS?
      Can colonized breast milk (mother’s or donor’s) mediate disease?
      Does GBS colonization in mothers cause recognizable signs/symptoms?
      What are the best ways to identify mothers/fathers or medical personnel who are at risk for transmitting GBS to newborns or others?
      Can vaccination prevent diseases in mothers and/or newborns?
      Can probiotics or prebiotics reduce risk of maternal carriage and/or newborn acquisition?
      What further new prevention strategies are likely to be adopted by practitioners or parents?
      What are the specific strains of GBS which can be protective rather than using antimicrobial, immune or microecologic therapies?
2. The most common miscellaneous enquiries concentrated on:
   A. Frustration that GBS disease remains an “unheralded threat” to pregnant women and babies
   B. Proposed legislation to fund biomedical research to identify gaps in prevention of GBS disease at all ages.
3. Particular interest in 2010-15 focused on:
   A. Antibiotic resistance of GBS
   B. The effect of GBS IAP on the neonatal microbiome and possible effects of microbe-influenced child development.

Materials, Methods, & Consent
1. We collected and analyzed parent-initiated GBS pregnancy-related questions over a 17-year period (1998-2015). The records and website (www.groupbstrepternational.org) were monitored by one of the authors.
2. We constructed and completed a logic model matrix in order to formulate practicable research suggestions.
3. We used non-statistical measurements to ensure a complete spectrum of parental enquiries.

Summary
There are many gaps in GBS disease prevention even in countries with a universal screening-based approach to help prevent early-onset GBS disease. Clearly there is a need to reduce those gaps and prioritize research.

References