Background

Maternal-fetal interactions have been largely investigated within the scope of maternal stress negatively impacting fetal development. There has been relatively little research on utilizing maternal-fetal interactions non-invasively to stimulate fetal development. Theoretically, a greater shift towards maternal homeostasis can result in an altered inter-uterine environment that can promote optimal fetal development. This study investigated the use of a maternal intervention, HRV biofeedback, to promote improved physiological maternal homeostasis with the ultimate aim of targeting fetal development.

Objective

The main objective was to document changes in fetal movement in response to maternal HRV biofeedback. Fetal movement provides an indicator of neurological development and therefore, evidence of a non-invasive maternal intervention that can stimulate fetal movement would provide the basis for further investigation to use a HRV biofeedback paradigm to promote optimal fetal development.

Study Design/Methods

There were two components of the study, the first utilizing an ABA design (N=22) and the second utilizing an ABABA design (N=23). Fetal movement amplitude (measured in arbitrary
units, AU; recorded by Toitu Software), which ranges from 0-50 AU and maternal HRV (LFHF and SDNN) were monitored continuously throughout both designs.

**Results**

Fetal movement was split into two groups based on the direction (increase or decrease) of fetal movement from baseline to biofeedback. A repeated measures ANOVA for mean fetal movement for the ABA design was significant for the decrease group (F(2, 20) = 6.401, \( p = 0.007, \eta_p^2 = 0.390; \)), with a high observed power (0.852). Post hoc tests indicated that fetal movement amplitude decreased with introduction of the intervention (large effect size). A repeated measures ANOVA for mean fetal movement for the ABA design was significant for the increase group (F(2, 10) = 4.868, \( p = 0.033, \eta_p^2 = 0.493 \)), with a medium observed power (0.665). Repeated measures ANOVA for fetal movement amplitude in the ABABA design was not significant, for both decrease and increase groups respectively, (F(4, 24) = 1.243, \( p = 0.319, \eta_p^2 = 0.172 \); observed power= 0.328) and (F(4, 12) = 2.039, \( p = 0.153, \eta_p^2 = 0.405 \); observed power= 0.442). A case-by-case individual analysis of fetal movements in the ABABA design using MATLAB revealed shifts in mean and trend of fetal movement associated with implementation and withdrawal of the biofeedback stimulus with individual variations in fetal movement that were not captured by the group level repeated measures analysis.

A repeated measures ANOVA for maternal LFHF ratio was significant for both ABA: (F(1.401, 25.210) = 22.504, \( p=0.000, \eta_p^2 = 0.556 \); observed power= .999) and ABABA: (F(1.474, 14.743) = 10.510, \( p = 0.003, \eta_p^2 = 0.512 \); observed power= 0.929). Post hoc analysis for both designs indicated that maternal LFHF ratio increased with initial implementation of the intervention (large effect size) and the ABA design noted a decrease during the non-intervention phase (large effect size).
A repeated measures ANOVA for maternal SDNN was significant for both ABA: $(F(2, 36) = 25.478, p= 0.000, \eta^2_p= 0.586; \text{observed power}= 1.000)$ and ABABA: $(F(4, 40) = 11.781, p= 0.000, \eta^2_p= 0.541; \text{observed power}= 1.000)$. Post hoc analysis indicated that maternal SDNN increased from baseline to biofeedback for both ABA and ABABA designs with large effect sizes. In the ABA design, maternal SDNN remained increased from biofeedback to post-biofeedback (large effect size) while in the ABABA design, maternal SDNN remained increased from baseline to post-biofeedback II (large effect size).

**Conclusion**

The main goal of testing the HRV biofeedback manipulation was to determine the feasibility of shifting baseline fetal movement trajectory thereby stimulating fetal neuro-motor pathways. The data on HRV biofeedback and fetal behavior (fetal general movement) is characterized by individual differences and necessitate further investigation on the implications of using HRV biofeedback as a stimulus to activate fetal neuro-motor pathways. The group level fetal movement data masks individual changes in fetal responding. The consistent activation of maternal baroreceptor and change in maternal SDNN provide positive indicators for the potential use of HRV biofeedback to target maternal ANS pathology such as preeclampsia that has detrimental effects on fetal development. The neurotrophic hypothesis outlines a balance between programmed cell death and strengthening of neural pathways during fetal development (Buss, Sun, & Oppenheim, 2006). Given the association between HRV biofeedback and changes in fetal movement, it is feasible that this manipulation can be used to activate fetal neural-motor pathways, thereby shifting the balance towards strengthening of these pathways and increasing the fetus’ range of responding to stimulus. Greater activation of a neural pathway provides these pathways a greater chance of survival during the prenatal period, allowing the fetus a broader
repertoire of neurobehavioral responses with the potential of more adaptive functioning post-birth. A broader range of neurobehavioral responding post-birth has the potential to impact areas of physiological and psychological adaptation such as emotion regulation and cognitive flexibility.

Fig 1.
Fig 2. Maternal Power Spectrum Changes during HRV Biofeedback (sample of one participant)