The psychophysiology of the maternal–fetal relationship

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Abstract
The enigmatic quality of the maternal–fetal relationship has been extolled throughout history with little empirical support. We apply time series analysis to data for 137 maternal–fetal pairs collected at 20, 24, 28, 32, 36, and 38 weeks gestation. Maternal heart rate and skin conductance data were digitized in tandem with fetal heart rate and motor activity. No temporal relations between fetal heart rate and either maternal variable were found, although averaged maternal and fetal heart rates were correlated from 32 weeks. Consistent temporal associations between fetal movement and maternal heart rate and skin conductance were detected. Fetal movement stimulated rises in each parameter, peaking at 2 and 3 s, respectively. Associations did not change over gestation, were unaffected by a maternal stressor, and showed within-pair stability. The bidirectional nature of the maternal–fetal relationship is considered.

Descriptors: Fetal heart rate, Fetal movement, Pregnancy, Maternal stress

There is perhaps no relationship more profound yet enigmatic than the first one. The bond between mother and fetus has been extolled throughout history and literature, with supposition on its nature ranging from the material to the metaphysical. Yet there has been surprisingly little academic examination of the maternal–fetal physiologic connection beyond that providing direct obstetric relevance.

The most common indicators of fetal functioning are heart rate and heart rate variability. There is a substantial body of knowledge concerning the neural genesis of both over the course of gestation (Dalton, Dawes, & Patrick, 1983; Martin, 1978; Parer, 1999; Yoshizato et al., 1994) but there are few reports of associations with maternal parameters under baseline conditions. Early reports using relatively primitive methods of maternal–fetal monitoring failed to detect associations between maternal and fetal heart rate (Sontag & Richards, 1938). A brief (15 min) recording using more sophisticated monitoring techniques also failed to detect significant correlations (Lewis, Wilson, Ban, & Baumel, 1970). However, subsequent implementation of 24-hr recording of both maternal and fetal electrocardiogram using subcutaneous electrodes resulted in a strong association when mean values were averaged over the course of the day (r = .78) or hour (r = .71; Patrick, Campbell, Carmichael, & Probert, 1982). Although fetal heart rate has a circadian rhythm with lowest levels occurring in the middle of the night (Patrick et al., 1982), it does not appear to be affected by maternal sleep states per se (Hoppenbrouwers et al., 1981).

Cursory understanding of the psychophysiological association between mother and fetus has been generated by studies that monitor the fetus during periods of maternal arousal. Physical exertion is associated with elevation in fetal heart rate (Artal et al., 1986) and declines in both variability (Macphail, Davies, Victory, & Wolfe, 2000; Manders, Sonder, Mulder, & Visser, 1997) and motor activity (Manders et al., 1997) measured shortly after the termination of exercise. Induced psychological stress, as effected through the Stroop Color–Word test, has been associated with suppressed motor activity and increased fetal heart rate variability (DiPietro, Costigan, & Gurewitsch, 2003). Another study reported increased fetal heart rate in women with high, but not low, anxiety (Monk et al., 2000).

There are a handful of studies that have evaluated the relations among maternal dispositional attributes, including anxiety and stress perception, and fetal neurobehavior. The most consistent, although not universal, findings indicate that persistent emotional arousal is associated with greater levels of fetal motor activity (DiPietro, Hilton, Hawkins, Costigan, & Pressman, 2002; Ianniruberto & Tajani, 1981; Sontag, 1941; Van den Bergh et al., 1989). Examination of fetal heart rate measures, including variability, has yielded less consistent results: negative associations with maternal stress over the second half of gestation in one cohort (DiPietro, Hodgson, Costigan, Hilton, & Johnson, 1996),

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but positive associations with pregnancy-specific stress at term only in another (DiPietro et al., 2002).

The goal of the current study was to examine, for the first time, the temporal nature of the maternal–fetal relationship by applying time series analysis to fundamental features of maternal and fetal functioning. This is an initial step toward ascertaining whether the maternal–fetal associations detailed above are mediated through immediate and direct stimulation of the fetus through maternal physiologic processes. Our method of antepartum fetal monitoring generates two streams of Doppler-based fetal data: fetal heart rate and fetal motor activity. Maternal data collection included measures of heart rate and electrodermal activity. Given the small but growing literature indicating that induced and chronic maternal sympathetic activation is associated with fetal heart rate and/or motor activity, we expected that changes in maternal physiological measures would be followed by fluctuations in fetal heart rate and motor activity. Prediction of the direction of effects was difficult given the limited and inconsistent body of knowledge in this area. We have previously proposed a biphasic relation between maternal arousal and motor activity based on other data collected from these participants (DiPietro et al., 2003), such that transient suppression in response to acute stress is followed by rebound in motor activity. Although the unit of analysis in this study is small (i.e., minutes), the data are aggregated over a longer period. Thus we expected that evidence of maternal sympathetic activation would be associated with increased fetal motor activity and faster fetal heart rate. Because maternal psychological factors have been shown to yield both immediate and prolonged effects on fetal behavior, we evaluated the degree to which both induced maternal arousal and chronic perception of stress, anxiety, and depression may modify any observed temporal relation.

Methods

Participants
Eligibility was restricted to nonsmoking women with uncomplicated pregnancies carrying singleton fetuses. Accurate dating of the pregnancy was required and based on early first trimester pregnancy testing or examination and/or confirmed by ultrasound. A total of 185 self-referred pregnant women were enrolled; 48 were either prospectively or retrospectively excluded as follows: preterm labor, preterm delivery, or both (21; 11%); gestational diabetes (6; 3%); congenital malformation (2; 1%); fetal death in utero or nonviable delivery (2; 1%); growth retardation or other condition of antepartum origin detected in the newborn (6; 3%); and lack of completion of protocol due to scheduling difficulties, moving, and so forth (12; 6%). The final sample comprised 137 maternal–fetal pairs representing a population of healthy, relatively well-educated women (M years education = 16.7, SD = 2.1, range 12–20; M age = 31.3, SD = 4.1, range 21–39). Fifty percent of the fetuses were female and this was the first child for 56% of the sample.

Design and Procedure
Maternal–fetal monitoring commenced at 20 weeks gestation and was repeated at 24, 28, 32, 36, and 38 weeks. Testing occurred at the same time of day during each visit (either 1:00 or 3:00 p.m.). Women were instructed to eat 1.5 hr prior to testing, but not thereafter. A brief real-time ultrasound scan was conducted to determine fetal position as a consideration in locating the Doppler transducer, measure amniotic fluid using a common index, and provide photographs to parents. Fetal monitoring proceeded for 50 min, with the mother resting comfortably in a semirecumbent, left-lateral position. At 32 weeks, monitoring duration was shortened to 30 min to accommodate a maternal stress manipulation. The manipulation consisted of a 29-min labor and delivery video in which women recounted their children’s births, interspersed with explicit delivery scenes in black and white (Birth Stories, The Cinema Guild, NY). Women were monitored in the same semirecumbent position and viewed the video wearing headphones, without the presence of companions. Individual response patterns of women and fetuses to this manipulation are presented elsewhere (DiPietro, Ghera, Costigan, & Gurewitsch, 2004).

Fetal data. Fetal data were collected using a Toitu (MT320) fetal actocardiograph. This monitor detects fetal movement and fetal heart rate through the use of a single wide array transabdominal Doppler transducer and processes this signal through a series of filtering techniques. The actograph detects fetal movements by preserving the remaining signal after bandpassing frequency components of the Doppler signal that are associated with fetal heart rate and maternal somatic activity. Reliability studies comparing actograph-based versus ultrasound-visualized fetal movements have found this monitor to be highly accurate in detecting both fetal motor activity and quiescence (Besinger & Johnson, 1989; DiPietro, Costigan, & Pressman, 1999; Maeda, Tatsunuma, & Utsu, 1999).

Fetal data were collected from the output port of the monitor and digitized at 1000 Hz through an internal analog-to-digital board using streaming software. Data were analyzed off-line using software developed in our laboratory. Digitized heart rate data underwent error rejection procedures based on moving averages of acceptable values as needed; fetal movement data represent raw voltage values generated from the actograph, scaled from 0 to 100 in arbitrary units. Mean values for fetal neurobehavioral measures are presented elsewhere (DiPietro, Caulfield, et al., in press).

Maternal physiological data. Maternal physiological signals were amplified using a multichannel, electrically isolated bioamplifier. Electrocardiogram was recorded from three carbon fiber disposable electrodes in triangulated placement (right mid clavicle, left mid axillary thorax, and upper left thigh for ground lead). Electrodermal activity (skin conductance) was monitored from two silver–silver chloride electrodes with a gelled skin contact area placed on the distal phalanxes of the first and index fingers of the nondominant hand affixed with adhesive collars to limit gel contact to a 1 cm diameter circle and velcro. Maternal data were time synchronized and analyzed in conjunction with fetal data. ECG data underwent R-wave detection, manual editing for artifact, and interbeat interval computation. To maintain consistency with fetal measures, interbeat interval values were prorated to second-by-second heart rate in beats per minute (bpm). Skin conductance was measured by administering a constant 0.5-V root-mean-square 30-Hz AC excitation signal and detecting the current flow. Skin conductance level was scaled from 0 to 25 μS and detrended to remove the mean, thereby amplifying the signal-to-noise ratio (–2.5 to +2.5 μS).

Maternal psychosocial data. Five validated self-report scales were administered at either one or two gestational ages. The schedule of administration was selected to minimize participant burden. Perceived daily stress was recorded at 24 and 36 weeks...
(Brantley, Waggoner, Jones, & Rappaport, 1987), state and trait anxiety at 28 and 38 weeks (Spielberger, 1983), and depressive symptoms at 32 weeks (Radloff, 1977). Pregnancy-specific stress was measured at 32 and 38 weeks using a scale validated, in part, on this sample of participants (DiPietro, Ghera, Costigan, & Hawkins, in press).

**Data Analysis**

The data were reduced by resampling at 1 Hz producing time series measurements \((X_1, Y_1), (X_2, Y_2), \ldots, (X_T, Y_T)\) for each pair of measures. Fifty minutes of data, sampled once per second, generates a time series of 3000 data points (1800 at 32 weeks). Each pair of values can be considered outcomes of a two-component stationary time series. A standard way of measuring the association between two components of a time series is through the cross-correlation function (Diggle, 1990) obtained by computing the correlation coefficient between each series at various time lags. The mathematical formula is included in the Appendix. Cross-correlations were conducted for each of four maternal-fetal variable pairs at each lag from \(-100\) s to \(100\) s: maternal heart rate with both fetal heart rate and movement, and maternal skin conductance with fetal heart rate and movement. Two descriptive statistics were extracted. The first is the individual lag, in seconds, at which the strongest association occurs. The second is the magnitude of the cross-correlation function at the overall peak lag.

Because this is an unfamiliar analysis to many readers, a few comments on the interpretation of the ensuing values are in order. Peak magnitude associations should not be interpreted in the same manner as Pearson correlations between mean values of two variables within a cohort of individuals. Fetal movement data are comprised of a continuous signal generated by the actograph; thus both small values representing minor movements (including some noise) are not distinguished from large values denoting gross motor activity. These lower amplitude signals were not filtered out of the analysis to avoid a priori decisions relating to movement size that were inconsistent with the exploratory nature of these analyses. Numerous sources of variation, including stochastic variability in the true lags, shape of responses, and measurement error can make a relationship that is perfectly paired have peak cross-correlation coefficients of values much less than one. Although some of these issues are also germane to Pearson correlations computed between variables, their influence is magnified in time series analyses that rely on multiple associations between streams of data.

Weighted least squares analysis was used to model the developmental trends in peak magnitudes of each variable pair from 20 to 38 weeks. This method estimates the correlation structure generated by the repeated measurements on the same fetus and uses the estimate to appropriately weight the observations in the regression analysis and produces appropriate estimates of regression parameters and their variances (Diggle, Liang, & Zeger, 1994). Although there were few instances of missing data due to technical problems or noncompliance at any visit prior to the final one (two instances of missing data at 28 weeks, one at 36 weeks), a substantial proportion (33%) of women who delivered at term did so prior to their scheduled 38 week recording. However, cases with missing values are not excluded from weighted least squares analysis. Fetal sex and parity (nulliparous vs. multiparous) effects were examined by comparing both peak magnitudes and lags by t tests at each gestational age. Pearson correlations were used to evaluate temporally independent associations between each maternal and fetal measure and within-fetal stability of the time series pairs over gestation.

Changes in response to the stress manipulation for maternal heart rate, skin conductance, and peak magnitudes of the maternal-fetal relations were evaluated using repeated-measures analysis of variance comparing baseline values to those generated during the video. Two series of Pearson correlation analyses were conducted to examine associations between self-report psychosocial measures and time series peak magnitudes. In the first, correlations were computed within specific gestational ages (e.g., 24 week stress with 24 week maternal heart rate/fetal heart rate peak magnitude). The second set of analyses was based on mean values of each time series pair averaged across gestation and mean values of each psychosocial measure. Given the large number of individual correlations, a Bonferroni correction was applied and only correlations that achieved significance of \(<.01\) were considered significant.

**Results**

Figures 1 and 2 illustrate results from the time series analysis for each fetal measure with maternal heart rate at the six gestational periods evaluated. Plots reflect mean peak magnitudes (darkest shaded areas) at each lag ranging from \(-40\) to \(+40\) s for display purposes although analyses included values through \(\pm 100\) s. Lighter shadings indicate interquartile ranges. Examination of Figure 1 indicates no systematic relations between maternal and fetal heart rate, but Figure 2 depicts consistent associations between maternal heart rate and fetal movement beginning at 20 weeks gestation. The peak lag is stable at \(2\) s throughout gestation. Positive lag values generated by the time series analyses indicate that the fetal variable precedes the maternal variable; thus the strongest association is found \(2\) s after a signal indicating fetal movement. Because it is difficult to ascertain the mean values of the peaks from visual inspection of Figures 1 and 2, Figure 3 provides means and 99% confidence intervals for each over time. Peak magnitudes are significantly different from 0 for maternal heart rate/fetal movement at each gestational age, but it is clear that the maternal heart rate/fetal heart rate values hover around 0. No significant linear trends over gestation were detected.

Similar results were found between fetal measures and maternal skin conductance; these are depicted in Figures 4 and 5. There are no systematic relations between maternal skin conductance and fetal heart rate, but consistent associations were detected with fetal movement beginning at 20 weeks gestation. Peak lags were stable at \(3\) s; again, a positive lag indicates that the fetal movement preceded changes in maternal skin conductance. Peak magnitude data presented in Figure 6 include maternal skin conductance/fetal movement values that are significantly different from 0 but maternal skin conductance/fetal heart rate values are near 0. No significant linear trends over gestation were detected. Analyses involving skin conductance were conducted with and without African-American participants (\(n = 17\)) because race has a well-known suppressive effect on skin conductance reactivity (Vrana & Rollock, 2002). Exclusion of this subsample made little difference to results. African-American women had significantly lower peak values for skin conductance/fetal movement, \(F(1,119) = 5.04, p < .05\), but not skin conductance/fetal heart rate.

**Maternal Parity and Fetal Sex**

Peak magnitude or lags did not differ by fetal sex or maternal parity at any gestational age.
Intrafetal Stability

Evident in Figures 3 and 6 is the degree of variability among maternal–fetal pairs in peak magnitude of maternal heart rate/fetal movement and maternal skin conductance/fetal movement. Peak values ranged from −.11 to .34 for the former and −.10 to .31 for the latter. In general, individuals with higher associations on one variable had higher associations on the other, rs at each gestational age = .62, .66, .52, .62, .59, and .43 at each gestational age from 20 to 38 weeks, respectively (ps < .001). Correlations computed between peak magnitudes for each of these maternal–fetal variable pairs over gestation are presented in Table 1.

Figure 1. Times series analysis results showing no association between maternal heart rate and fetal heart rate at six gestational ages.
Two correlational analyses were conducted to determine whether local uterine factors (i.e., amniotic fluid and fetal size) were associated with peak magnitudes for either maternal heart rate/fetal movement or maternal skin conductance/fetal movement. Fetal size, approximated by using weight at birth, was unrelated to these values. Amniotic fluid volume, measured at each gestational age, was also unrelated.

Figure 2. Time series analysis results showing a consistent relation between maternal heart rate and fetal movement from 20 to 38 weeks gestation. Note peak lag time of 2 s.
Women responded to the video with a decrease in heart rate (although these were inconsistent over gestation).

Positive associations were detected with fetal movement, were found between maternal skin conductance and fetal heart movement beginning at 32 weeks. No significant associations heart rate beginning at 28 weeks and maternal heart rate and fetal age. Significant associations emerged between maternal and fetal variables averaged over the entire recording at each gestational age. Bivariate Maternal–Fetal Associations

Maternal heart rate with fetal heart rate and fetal movement. Vertical cross-correlation (peak magnitude) function values for maternal heart rate with fetal heart rate and fetal movement using similar time series analysis employed is quite accurate (Dawes, 1993).

In contrast to the lack of significant time-based associations, beginning at 32 weeks gestation, women with higher overall heart rates had fetuses with higher heart rates and greater motor activity. Although there is inconsistency in the literature regarding whether maternal and fetal heart rates are correlated, the magnitude of the correlations we detected were below those reported following 24-hr recordings (Patrick et al., 1982) but significant despite reports of no relations by others (Lewis et al., 1970). Despite its apparent sensibility, this remains a phenomenon that is difficult to explain (Patrick et al., 1982). Because we were unable to document time-dependent associations, such correlations must be explained through characteristics of the uterine milieu that were not measured in this study, including genetic, metabolic, endocrine, or other unidentified environmental factors. For example, cortisol levels are known to be correlated between mother and fetus (Gitau, Cameron, Fisk, & Glover, 1998), and thus to the extent that activation of the maternal sympathetic pathways would involve changes in cortisol level, there may be coactivation of the fetal sympathetic pathway by the same mechanism. An alternative explanation may be that the fetal heart rate entrains to the maternal heart rate through acoustic sensory channels. This proposal is consistent with the developmental change in the size of this association, because fetal auditory capacities continue to develop over the third trimester (Querleu, Renard, Boutetville, & Crepin, 1989).

Unlike fetal heart rate, fetal movement was temporally associated with both maternal heart rate and skin conductance. The direction of this effect was in direct contrast to expectations, in that the fetus precipitated a maternal response. Although maternal heart rate is subject to dual autonomic innervation, electrodermal activity is singly innervated by the sympathetic branch (Venables, 1991). The detected lag times indicate that maternal heart rate responds maximally after 2 s and skin conductance responds maximally after 3 s. Both latencies are consistent with expectations based on observations of elicited physiological responses (Dawson, Schell, & Filion, 2000; Stern, Ray, & Quigley, 2001; Turpin, 1983; Venables, 1991). The magnitudes of the cross-correlations between maternal and fetal measures were consistent over gestation, but relatively small in magnitude. As previously discussed, the time series analysis employed is more likely to underestimate associations. To put these findings in some context, a report of the well-known, robust relationship between fetal heart rate and fetal movement (Dawson, Schell, & Filion, 2000) was unrelated to responsiveness. None of the four maternal–fetal synchrony measures changed in response to the manipulation.

Psychosocial Measures
Of all the associations evaluated, only one significant, modest correlation emerged between maternal anxiety at 38 weeks and the degree of association between skin conductance and fetal movement at that time, \( r(87) = .30, p < .01 \). Of the remaining correlations, the majority (79%) were less than \( r \pm .10 \) in magnitude.

Discussion
These results illustrate how little is known about psychophysiology of the earliest relationship. No time-dependent associations between fetal heart rate and either maternal heart rate or skin conductance were detected; thus we conclude that fast-acting aspects of maternal sympathetic activation do not directly affect fetal heart rate. These data are consistent with findings of relative noncorrespondence of fetal heart rate to induced maternal heart rate and blood pressure changes (Monk et al., 2000). However, we temper this conclusion with a caveat. Fetal heart rate was measured using Doppler technology, which quantifies heart rate by detecting motions of the fetal heart. Commercially available fetal electrocardiography devices function only with the use of scalp electrodes applied after rupture of membranes; existing experimental systems that extract fetal from maternal ECG are challenged by a period of increased electrical isolation of the fetus during the middle period of the third trimester (Oostendorp, van Oosterom, & Jongsma, 1989). However, although the sampling rate of fetal monitors is necessarily lower than that used to quantify interbeat intervals, the autocorrelation techniques underly modern Doppler detection of heart rate are quite accurate (Dawes, 1993).

Figure 3. Cross-correlation (peak magnitude) function values for maternal heart rate with fetal heart rate and fetal movement. Vertical lines indicate 99% confidence intervals.
series techniques established the strength of that relation to range from approximately .10 to .25 over the same gestational period (DiPietro, Irizarry, Hawkins, Costigan, & Pressman, 2001).

Because of this unexpected result, a number of confirmatory post hoc manipulations were conducted, including reversing the order of the sequencing of variables entered into the time series and randomly examining individual movements in relation to

Figure 4. Time series analysis results showing no association between maternal skin conductance and fetal heart rate at six gestational ages.
maternal measures. All supported the temporal linkage of the findings. The magnitude of the cross-correlations did not change from mid- to late gestation, although this does not rule out the possibility that a developmental trend might have been detected had monitoring commenced earlier. However, the emergence of fetal behavioral states begins within the period of study and maturity of state patterns is evident at term (Nijhuis & van de Pas, 1992). The lack of developmental trends in maternal–fetal

Figure 5. Time series analysis results showing a consistent relation between maternal skin conductance and fetal movement from 20 to 38 weeks gestation. Note peak lag time of 3 s.
synchrony suggests that these processes are not dependent on fetal neuromaturation in general, and unrelated to state development in particular. It should be noted that there are multiple influences on both fetal behavior and maternal sympathetic arousal and the fact that shared variance between the two was detected does not preclude the possibility of other mediating influences on either aspect.

How does fetal motor activity affect maternal functioning? Although women perceive most large amplitude or prolonged movements, they are not good detectors of smaller spontaneous or evoked fetal movements (Kisilevsky, Killen, Muir, & Low, 1991) and detect as few as 16% of all movements at term (Johnson, Jordan, & Paine, 1990). This suggests that the maternal sympathetic response is evoked in the absence of perception of movement. On average, the fetus moves approximately once per minute during the second half of gestation (DiPietro, Caulfield, et al., in press; DiPietro, Costigan, Shupe, Pressman, & Johnson, 1998; Manning, Platt, & Sipos, 1979; Nasello-Paterson, Natale, & Connors, 1988; Roberts, Griffin, Mooney, Cooper, & Campbell, 1980; Roodenburg, Wladimiroff, van Es, & Prechtl, 1991). Thus, despite the ubiquity of the stimulus, the lack of a change in the degree of maternal responsiveness over gestation suggests that women neither habituate nor become sensitized to fetal movements.

The question must be answered, at least in part, by mechanisms that do not require conscious maternal mediation. This is further supported by our failure to detect any influence of maternal physiological arousal or psychosocial stress characteristics on these associations. Other internal stimuli unrelated to the fetus, such as tachygastria, generate autonomic response in pregnant and nonpregnant individuals (Koch, Stern, Vasey, & Dwyer, 1990). Perhaps the most obvious mechanism through which fetal movement may generate an autonomic response involves the perturbations to the uterine wall. The normal response of the uterus to distension is contraction. Contractions are known to be stimulated by beta adrenergic pathways and modulated by noradrenergic pathways (Andersson, Ingemarsson, & Persson, 1973). It is possible that distension of the uterine wall caused by fetal movement may stimulate neural fibers that would effect a contraction, but may also stimulate a noradrenergic response in the mother that would modify this to prevent excessive contractions. Such modulation of the normal physiologic response is not a unique maternal physiologic adaptation to pregnancy. For example, the renin–angiotension–aldosterone system is markedly enhanced throughout pregnancy, yet the effect of angiotension is blunted in normal pregnancies as a protective mechanism against hypertension (Parisi & Creasey, 1992).

Although physiologic feedback systems may provide local mechanisms that explain these associations, these findings also have broader implications of the role of the fetus in pregnancy. There is precedent for considering the fetus as an active agent in arenas ranging from ontogeny (Smotherman & Robinson, 1987) to stimulation of labor (Challis et al., 2001). We suggest that the observed phenomenon of maternal sympathetic activation by the fetus may serve a signal function to the pregnant woman in preparation for the consuming early demands of child rearing and redirecting maternal resources directed at competing but less relevant environmental demands. Pregnancy has been associated with a dampening of physiological activation on a variety of levels to laboratory challenges including cognitive stressors (Matthews & Rodin, 1992), postural changes and isometric exercise (Barron, Mujais, Zinaman, Bravo, & Lindheimer, 1986), and cold pressor (Kammerer, Adams, von Castelberg, & Glover, 2002). This pattern of findings can be interpreted as reflecting diminished responsivity to external stressors that may jeopardize pregnancy. Transient deficits in cognitive performance have also been documented (Buckwalter et al., 1999; deGroot, Adam, & Hornstra, 2003). Although the well-known variations in sex steroids or glucocorticoids that occur during pregnancy are putative culprits, no such linkage in human pregnancies has been established (Buckwalter et al., 1999; Keenan, Yaldoo, Stress, Fuerst, & Ginsburg, 1998). We suggest that sympathetic activation of the mother by the fetus is an unidentified mechanism that may contribute to observed decrements in cognitive performance in

Table 1. Intrafetal Consistency in Maternal Heart Rate/Fetal Movement and Maternal Skin Conductance/Fetal Movement Time Series Peak Magnitude over Gestation

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Note: Values above the diagonal are for maternal heart rate/fetal movement; values below the diagonal are for maternal skin conductance/fetal movement.
*p < .05; **p < .01.
pregnancy by interfering with parasympathetic processes that are required for maintenance of attention. Moreover, periodic sympathetic surges generated by the fetus may serve to entrain maternal arousal patterns to the behavior of the fetus with implications for the impending needs of newborn care. Among these is the potential that the periodic declaration through movements of the fetal presence and resultant sympathetic surge provides the rudimentary basis of the strong maternal protective response.

A seminal paper by Bell (1968) challenged the notion that child rearing was a unidirectional phenomenon and introduced the now well-accepted construct that the relationship between mothers and children is bidirectional. More recently, a similar model has been applied to understanding the effects that rodent offspring have on the development of the maternal nervous system (Kinsley et al., 1999). The complex nature of the most fundamental human relationship raises questions regarding the longer term implications of this phenomenon. The degree of stability within individuals in the magnitude of the fetal movement associations with skin conductance and heart over the course of gestation is large, particularly when considered in relation to most other developmental phenomena. These correlations are comparable to or exceed those for fetal heart rate and motor activity alone (DiPietro, Hodgson, Costigan, & Johnson, 1996), suggesting that synchrony is a stable characteristic of individual maternal–fetal pairs.

A distal, but intriguing question is whether maternal–fetal synchrony sets the stage for postnatal synchrony in maternal–child interaction. Are women who are more physiologically responsive to fetal movements more responsive to infant behavior? Does success at fetal stimulation of a maternal response translate to an infant’s success at serving an elicitor of care giving? Efforts to address these questions are currently underway in our laboratory as we follow these maternal–child pairs through the first years of life and examine the continuing intricacies of the earliest relationship.

**REFERENCES**


Assuming that the measurements are the outcome of a stationary stochastic process, we define the cross-correlation function with

\[
\rho(h) = \frac{E[(X_t - \mu_X)(Y_{t+h} - \mu_Y)]}{\sigma_X \sigma_Y}, \quad h = 0, \pm 1, \pm 2, \ldots
\]

with \( \mu_X = E(X_t) \) and \( \mu_Y = E(Y_t) \) the expected value of \( X_t, \ldots, X_T \) and \( Y_t, \ldots, Y_T \), respectively and \( \sigma_X^2 \) and \( \sigma_Y^2 \) the variance of \( X_t, \ldots, X_T \) and \( Y_t, \ldots, Y_T \), respectively. With the assumption of stationarity, none of these quantities depend on time \( t \). If fetal motor activity tends to increase whenever there is a change in maternal heart rate, we expect \( \rho(0) \) (the common definition of correlation between \( X_t, \ldots, X_T \) and \( Y_t, \ldots, Y_T \) is the cross-correlation at lag \( 0 \)) to be relatively high. However, if fetal motor activity tends to increase some time \( h \) after maternal heart rate change occurs, then it is \( \rho(h) \) that we expect to be high. Notice that \( \rho(h) \) has an intuitive definition as the correlation between \( X_t, \ldots, X_{T-h} \) and \( Y_{h+1}, \ldots, Y_T \). The sample cross-correlation function is

\[
\hat{\rho}(h) = \frac{1}{T-h} \sum_{t=h+1}^{T} (X_t - \bar{X})(Y_{t+h} - \bar{Y})
\]

with \( \bar{X} \) and \( \bar{Y} \) the sample averages of \( X_t, \ldots, X_T \) and \( Y_t, \ldots, Y_T \), respectively, and \( \sigma_X^2 \) and \( \sigma_Y^2 \) the sample variances of \( X_t, \ldots, X_T \) and \( Y_t, \ldots, Y_T \), respectively. The sample cross-correlation is the standard estimate of the cross-correlation function.

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