Negative Emotionality and Cortisol during Adolescent Pregnancy and Its Effects on Infant Health and Autonomic Nervous System Reactivity

ABSTRACT: This research examined the relations among maternal emotionality, biology, and infant outcome and autonomic nervous system reactivity (cardiac vagal tone). The sample consisted of 27 pregnant adolescents and their 3-week-old infants. Measures of anxiety, depression, anger, and saliva cortisol were obtained from the adolescents both pre- and postnatally. Infant outcome measures consisted of gestational age at delivery, birth weight, number of risk factors at birth and at 24 hr, Apgar score at 1 and 5 min, abnormalities on newborn physical exam, number of resuscitation measures used on the infant, and cardiac vagal tone. Significant relations were found among the adolescent’s emotionality, infant physical outcomes, and cardiac vagal tone. Higher concentrations of adolescent cortisol were associated with lower infant Apgar scores and an increased need for resuscitation measures performed on the infant. The positive association between negative emotions and better infant outcomes also was found and may reflect the sensitivity of the adolescents to their feelings and needs during pregnancy. Social support during pregnancy mediated the effects of maternal negative emotionality and infant cardiac vagal tone. © 1998 John Wiley & Sons, Inc. Dev Psychobiol 33: 163–174, 1998

Keywords: adolescent pregnancy; emotionality; cardiac vagal tone; social support

INTRODUCTION

Adolescent pregnancy continues to be a major public health problem. The infant outcomes of these pregnancies are often poor with high morbidity and mortality (Hofferth & Hayes, 1987). The potential emotional and social roller coaster of adolescence, in conjunction with the stressors that accompany pregnancy, are believed to be involved in the etiology of poor infant outcomes. Multiple and simultaneous stressors may cause pregnant adolescents to neglect their own physical and psychological health, be non-compliant with medical regimes, and fail to adjust their lifestyles to accommodate to the demands of pregnancy (Menken, 1980). The poor outcomes observed in infants of adolescent mothers may be directly and indirectly attributed to the emotional well being of the adolescent mother.
Maternal physical and psychological characteristics during pregnancy are hypothesized to affect birth outcomes, but few studies empirically document these relationships. Young age is an obvious physical characteristic associated with poor infant outcomes. The younger the mother, the greater her risks of having a low-birth-weight or small-for-gestational-age infant (Amini, Catalano, Dierker, & Mann, 1996; Fraser, Brockert, & Ward, 1995). Lower-birth-weight infants have an increased risk of physiologic and developmental problems at birth and neonatal mortality (McAnaney, 1987; McAnaney & Stevens-Simon, 1990). These infant mortalities are postulated to be a direct result of being a low-birth-weight infant and an indirect result of noncompliance with health care.

The present research considers maternal emotionality within a biosocial theoretical framework that considers the interactions between physiology, emotionality, and contextual influences on adolescent and infant outcomes to teen pregnancy (see Susman, Dorn, Schwab, et al., 1998). The purpose is to examine longitudinally the influence of prenatal psychological, biological, and environmental processes as mechanisms involved in the outcomes of adolescent pregnancy. A longitudinal design is the preferred design for examining links between prenatal risk and protective factors during fetal development and the physiological status of infants. Major shortcomings of previous research include the absence of longitudinal studies and a failure to consider the early pregnancy period. Until the specific early psychological and environmental influences that lead to observed poor outcomes in infants can be identified, even the most successful interventions will continue to provide only minimal reductions in the detrimental consequences of adolescent childbearing.

**Emotional and Biological Processes**

The development and regulation of emotionality is an emerging topic in research on adolescents (Huffman & Hauser, 1994) but very little is known about emotions in pregnant adolescents (Hamburg, 1986). In both animal and human models, negative maternal emotionality appears to affect adversely infant outcomes (Istvan, 1986). Depression during pregnancy is hypothesized to be linked to poor infant outcome (Zuckerman & Beardslee, 1987). The suggested mechanisms involve stress and depression-related maternal physiological processes that directly interfere with fetal development. For instance, placental hypoperfusion, secondary to maternal stress and depression, may lead to growth impairment for the fetus and/or precipitation of preterm delivery (McAnaney & Stevens-Simon, 1990). This speculation is in agreement with findings showing a relationship between cortisol and depression in pregnant adolescents (Dorn, Susman, & Petersen, 1993; Susman, Dorn, Schwab, et al., 1998). Other research reports that mothers classified as “high risk” (those experiencing high anxiety and depression) pregnancies and who also reported low satisfaction for social support had higher levels of norepinephrine than mothers who reported satisfaction with their social support (Kemp & Hatmaker, 1989). Depressed pregnant women also had lower levels of estradiol at 36 weeks’ gestation compared to nondepressed women (Steer, Scholl, Hediger, & Fischer, 1992). Stress can suppress the production of estradiol (River & Vail, 1985). Lower levels of estradiol may reflect placental dysfunction.

The possibility of an indirect effect of maternal depression on infant outcomes is based on clinical observations that pregnant women, both adolescent and adult, who are depressed are likely to participate in adverse health behaviors (Zuckerman, Amaro, Bauchner, & Cabral, 1989). Depressed pregnant women have a higher likelihood of smoking cigarettes and using toxic substances, such as alcohol and cocaine (Zuckerman et al., 1989). These substances may be important mediators of the effects of psychological stress on low birth weight and prematurity in the infant (McAnaney & Stevens-Simon, 1990). Depressed pregnant mothers also may experience weight loss and poor appetite (Steer et al., 1992). The effects of inadequate weight gain increase the risk of giving birth to a low-birth-weight infant, having a preterm birth delivery, and having a small-for-gestational-age infant (Steer et al., 1992). Although the specific mechanisms involved in the direct or indirect effects of depression during pregnancy and infant outcome are unknown, the conclusions are the same across studies: Depression is related to negative infant outcome. Anxiety is reported to have mixed effects on birth outcomes (Istvan, 1986). Infants of anxious mothers cried significantly more than infants of mothers who were less anxious (Ortinger & Simmons, 1964). Maternal anxiety during pregnancy also was linked to a lower Apgar score at 5 min following birth (Crandon, 1979) and a higher incidence of low-birth-weight and premature infants (Dorn, Susman, & McCool, 1998).

The effects of anger and hostility on infant outcomes are rarely examined, particularly in adolescent mothers. The direct or indirect effects of maternal anger on a developing fetus may be difficult to assess considering that anger may be a component of depression (Renouf & Harter, 1990). In the postpartum period, anger and hostility may lead to physical abuse...
of the infant, whereas depression may result in less maternal gazing at their infants, which in turn may lead to delayed or impaired bonding between the infant and mother (Livingood, Daen, & Smith, 1983). Overall, depression, anxiety, anger, and hostility are hypothesized to have negative consequences for a wide range of infant outcomes.

The biological process considered to be important to infant outcome is activation of the hypothalamic–pituitary–adrenal (HPA) axis (see Chrousos & Gold, 1992; Johnson, Kamilaris, Chrousos, & Gold, 1992). Maternal HPA activation may have transplacental consequences, including stimulation of placental hormones (Frim, Robinson, Smas, Adler, & Majzoub, 1988). The cascading reactions that occur in the HPA axis following a disturbance to the organism reflect healthy adaptation processes needed to reestablish the threatened steady state. Individual differences in adaptation, reflected in the secretion of the hormone cortisol (an index of HPA axis activity), are reported in animal (Suomi, 1987) and human model research (Dorn et al., 1993; Susman, Dorn, Inoff-Germain, Notelmann, & Chrousos, 1997). To some degree, a highly reactive HPA axis may place individuals at risk for health problems. Individuals who show greater HPA reactivity tend to have more significant behavioral distress symptoms (Gunnar, Mangelsdorf, Larson, & Hertsgaard, 1989) and more negative emotionality (Susman, Dorn, Inoff-Germain, et al., 1997). Higher concentrations of cortisol in pregnancy predicted pre- and postmature births, suggesting that HPA reactivity may have systemic influences on maternal and fetal health (McCool, Dorn, & Susman, 1994).

A genetic component of HPA reactivity has been demonstrated in primates. Rhesus monkey parents with high HPA axis reactivity were more likely to have offspring with high HPA reactivity (Suomi, 1987). Messenger RNA transcription factors are proposed to mediate the stability of reactivity (Post, 1992). Even though maternal cortisol does not directly affect the infant, a maternal stressor may activate fetal and placental corticotropin-releasing hormone (CRH), which further stimulates the secretion of fetal cortisol (Frim et al., 1988). The adolescent’s lifestyle also has been implicated in the transmission of HPA reactivity. It is speculated that mothers who are highly reactive may use unfavorable coping strategies (e.g., alcohol, smoking) and insensitive parenting styles that may result in higher levels of behavioral reactivity in their children.

**Environmental Processes**

The less optimal a caregiving environment, the more likely unfavorable outcomes will be observed in infants of adolescent mothers (Coll, Volk, Hoffman, & Oh, 1986). Nonetheless, an environmental factor such as social support is believed to buffer the negative impact of adolescent parenting. For instance, social support has been observed to predict both birth-related and postpartum outcomes (Dunst, Vance, & Cooper, 1986). Social support, in the form of family and peer support, is related to positive outcomes for young parents and their children (Wise & Grossman, 1980); better labor progress and babies with higher Apgar scores (Collins, Dunkel-Schetter, Lobel, & Scrimshaw, 1993); and a successful transition to parenthood (Cnice, Greenberg, Ragozin, Robinson, & Basham, 1983). Social support may also reduce the physiological consequences of stressors attendant to pregnancy. In a study by Molfese et al. (1987), perception of social support was found to mediate the effects of maternal anxiety, depression, and stress during pregnancy on gestational age, 5-min Apgar, and Neonatal factors scores.

As previously stated, adolescents with supportive environments are likely to cope better with their negative emotions when friends and family offer assistance in making the transition into motherhood as comfortable (both physically and mentally) as possible (Cnic et al., 1983; Wise & Grossman, 1980). Thus, social support is hypothesized to reduce the effects of negative emotionality on infant outcomes.

**Autonomic Nervous System (ANS) Reactivity**

The effects of maternal emotions on fetal outcomes may affect specific organ systems as well as the diffuse emotional systems discussed earlier. The specific system of interest in this study is the autonomic nervous system (ANS). The ANS is a major component of the stress system (Chrousos & Gold, 1992). Following a stressful stimulus, the ANS is activated, in conjunction with the HPA axis. In children, ANS measures of cardiac vagal tone and heart rate variability (HRV) are linked to behavioral vulnerability (Porges, 1992). Using various methods of assessment, cardiac vagal tone has been observed to be related to behavioral reactivity (Stifter & Fox, 1990), behavioral inhibition (Kagan, 1992), poor attentional responses (Richards, 1987), and behavior problems (Portales, Doussard-Roosevelt, Lee, & Porges, 1992). Low cardiac vagal tone was related to maladaptive and more disorganized behavior in some adults (Mezzacappa et al., 1996). High cardiac vagal tone, on the other hand, was found to be related to increased sociability in later infancy (Fox & Stibler, 1989) and better cognitive performance (Fox & Porges, 1985). Based on his research on healthy and sick
newborns, Porges (1983) concluded that cardiac vagal tone might be used as an index of the integrity of the central nervous system. The connection between maternal psychological characteristics during pregnancy and infant cardiac vagal tone is examined here for the first time. We hypothesize that the emotional characteristics of the mother that predispose infants to poor health outcomes are likely to predict lower infant cardiac vagal tone as well.

In summary, the specific aims of the present study were: (a) to examine the relations between maternal emotionality during pregnancy and birth outcomes of infants; (b) to identify emotional processes (depression, anxiety, anger, hostility) that influence neonatal ANS reactivity; (c) to identify biological processes (HPA reactivity) in the mother during pregnancy that influence the birth outcomes of the infant; and (d) to test whether environmental processes (e.g., social support) mediate the effects of maternal emotionality on infant outcomes (birth outcomes and the ANS of the infants). Four hypotheses were tested: (a) pregnant adolescents who experienced greater negative emotionality (depression, anxiety, anger, and/or hostility) during pregnancy will have infants with less optimal physical outcomes at birth, compared to infants born to adolescents with less negative emotionality; (b) pregnant adolescents with greater depression, anxiety, anger, and/or hostility symptoms will have infants with lower cardiac vagal tone, a marker of developmental risk; (c) pregnant adolescents with higher levels of cortisol during pregnancy will have infants with poorer birth outcomes; and (d) environmental processes (social support) will mediate the effects of maternal emotionality on infant outcome (infant birth outcomes and cardiac vagal tone). Specifically, social support will buffer the adverse effects that negative emotionality may have on infant outcomes.

METHODS

Sample

The sample consisted of 27, primarily Caucasian (96%), Caucasian, 4% African-American), rural and semi-rural, pregnant adolescents. The adolescents ranged in age from 13.9 to 19.4 years ($M = 17.3$ years, $SD = 1.53$). The family socioeconomic status (SES) of the sample was primarily low to middle income. Of the sample, 67% were still in school, 15% had graduated from high school, 11% were out of school and working on their GED, and 7% had dropped out of school. Eighty-nine percent had no prior history of drug abuse and 40% reported the use of cigarettes at the first assessment period.

The current sample was drawn from a larger sample of 78 pregnant adolescents with similar characteristics (ages ranged from 13 to 19 years, $M = 17.4$, $SD = 1.44$ years). The sample is unique because it is one of the few nonurban pregnant adolescent samples that has been studied and it included adolescents prior to 16 weeks gestation (see Susman, Dorn, Schwab, et al., 1998).

The adolescents were referred to the project by a member of the adolescent’s health care team. The criteria for an adolescent’s inclusion in the study were: (a) the first 16 weeks of pregnancy, <20 years old at the expected date of delivery, planning to keep their babies, English-speaking, and free from any chronic illness or serious mental health problems. Pregnant adolescents were recruited from a midsized community hospital prenatal clinic, a family physician group, and community obstetricians.

The project was approved by the Institutional Review Boards of a research university and a cooperating hospital where the data were collected. Adolescents over age 18 years, and a parent or legal guardian of those under age 18 years, gave written consent to the study.

The adolescents were enrolled in the study during or prior to 16 weeks gestation (by dates, examination, or sonogram) ($M = 13.32$ weeks gestation, $SD = 2.98$). All adolescents were seen at 8:30 a.m. to control for diurnal variations in hormonal concentrations. The adolescents were compensated for their time. The average age of infants was 4 weeks ($M = 3.7$, $SD = 1.8$) when they were assessed.

In our sample, we only had one infant born <37 weeks gestation (34 weeks; sample mean = 39.79 weeks, range = 34–42 weeks). This infant was eliminated from the analyses in order to determine if the findings were influenced by it as an outlier. The findings did not differ when the infant was eliminated from the analysis so the infant was included in all analyses.

Procedures

The adolescents were assessed during early pregnancy (less than or equal to 16 weeks gestation (T1)), the 3rd trimester (32–34 weeks gestation (T2)) of pregnancy, and approximately 4 weeks following delivery (T3). The first visit to the research lab (T1) took place within a few days after the first prenatal physician visit. The infants were assessed within 24 hr of birth and at the postpartum visit (T3).

$^3$Tests were run to compare the subsample used in this research to the larger sample it was obtained from. None of the maternal or infant data were significantly different between the two samples.
During the first visit (T1), demographic information and paper and pencil measures of family environment, social support, depression, anxiety, anger, and hostility were obtained from the adolescents. During this time, saliva was obtained for cortisol determination. During the second visit (T2) and the postpartum visit (T3), the adolescents were given the same measures as at T1.

Maternal Emotionality Measures

**Depression.** Depression was assessed by two methods, the Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and the depression subscale of the NEO-AC Personality Inventory (NEO) (Costa & McCrae, 1985). The BDI is a self-report measure consisting of 21 items reflecting symptoms and attitudes of depression. The items are scored from 0 to 3 with 3 indicating the greatest degree of depression symptoms.

The NEO is a measure comprised of 40 items, which are divided into five subscales. The subscales of depression, anxiety, and hostility were used in the current analyses.

**Anxiety.** Anxiety was assessed using the State–Trait Anxiety Inventory (STAI) (Spielberger, Gorsuch, & Lushene, 1970) and the anxiety subscale of the NEO. The STAI is a well-standardized, 40-item, self-report instrument designed to measure both state and trait anxiety. T scores were used in the analysis. The anxiety subscale on the NEO was the second measure of anxiety. It is scored the same as the depression subscale.

**Anger.** The State–Trait Anger Scale (STAS) (Spielberger, Jacobs, Russell, & Crane, 1983) was used to assess anger, which is conceptualized as an emotion that varies in intensity but is a relatively stable personality trait. The STAS consists of 15 items measuring state anger and 15 items measuring trait anger. State anger is defined as an emotional state that includes such feelings as tension, annoyance, irritability, fury, and rage. Trait anger defines individual differences in the frequency that anger is experienced. The item mean scores were used in the analysis as there are no T scores for the STAS.

**Hostility.** A measure of hostility was obtained from the hostility subscale of the NEO. The hostility subscale is scored in the same manner as the depression subscale.

**Social support.** Assessment of social support was obtained from the Inventory of Socially Supportive Behaviors (ISSB) (Barrera & Ainlay, 1983). The ISSB consists of 40 self-report items that make up four subscales (tangible assistance, positive social interaction, nondirectional support, and directive guidance). Our interests were in the overall effect of social support; thus, the total mean score was used in the analysis.

Maternal Biological Measures

**Cortisol.** Five saliva samples were obtained during each of the three visits at 20-min intervals beginning upon arrival at the laboratory (0, 20, 40, 60, and 80 min). The first saliva sample was always obtained at 8:30 a.m. in order to control for diurnal variations in cortisol. The cortisol mean for the adolescents was obtained from these five samples. Cortisol reactivity (cortisol percent change) of the adolescents was obtained by subtracting the value of saliva Sample 1 from saliva Sample 5. Cortisol mean captures differences between individuals whereas cortisol reactivity captures differences within individuals (See Dorn, et al., 1993; Susman, Dorn, Schwab, et al., 1998).

Salivary flow was stimulated by sugarless gum, which does not interfere with assay determination. To maintain consistency across samples, participants rinse their mouths with water prior to beginning salivary sampling to remove particles from the oral cavity. After 5 min, the participants spit a 5-ml sample of saliva into a collection tube. Samples were frozen at −80°C for later analysis in the Behavioral Endocrinology Laboratory, The Pennsylvania State University. Cortisol concentrations were established using the Pantex cortisol radioimmunoassay kit (Santa Monica, CA). All assays were performed by the same laboratory technician in order to quality-control standard techniques. The mean intra- and interassay coefficients of variations for saliva cortisol were: 7.1% and 3.3%, respectively. All samples were done in duplicate. The cortisol mean and reactivity of the five saliva samples were used in the analyses.

Neonatal Assessment

Infant outcome measures were obtained from the medical records at birth (birth weight, number of risk factors at birth and 24 hr, Apgar score at 1 and 5 min, number of abnormalities on newborn profile exam, and the number of resuscitation measures used). Because of the number of variables used to assess infant birth outcomes, an overall “infant risk factor” was created which consisted of health indices at birth (e.g., Apgar score at 1 and 5 min, number of resuscitation methods required, number of abnormalities on...
newborn profile exam, number of risks at 1 hr, and length of stay in hospital). Infants were assigned a value of “one” for scores that reflect risk status and a “zero” for values that do not. Infant outcome values that were assigned a one were: an Apgar of less than 7 on the 1-min assessment, an Apgar of less than 7 on the 5-min assessment, more than one resuscitation method required at birth, more than one abnormality on the newborn profile exam, more than one risk at the 1-hr exam, and more than 2 extra days at the hospital. The maximum possible risk score obtainable was 6. With our sample of infants, the average risk score was 2, with a range from 0–5. High scores on the risk measure reflect greater risks for developmental problems or complications of labor and delivery.

Heart rate data were assessed at the postpartum visit. During the visit, 10 min of resting EKG were recorded while the infants sat quietly in their mother’s lap or in an infant chair. If an infant fussed inconstantly, the EKG was stopped and resumed after the fussing stopped. The majority of infants were in a calm/alert state during the 10-min testing period.2

Infant EKG was recorded by placing three disposable electrodes on the infant’s chest in a triangular pattern. EKG data was collected off-line using a Grass preamplifier (Model P-15) and Vetter FM instrumentation recorder. In order to quantify the data, the EKG pulses were passed through an A/D converter programmed to display the raw EKG signal. These data were then viewed and a threshold was set to trigger at each R spike. The square wave impulses produced by this trigger were timed (in ms) and organized into a heart period file. These data were then analyzed further in accordance with a method developed by Porjes (1985) to compute an index of cardiac vagal tone (the ϕ statistic), which is a noninvasive measure of cardiac vagal tone. The cardiac vagal tone index measures heart rate activity modulated via the vagus nerve and is associated with the frequency of respiration, for example, respiratory sinus arrhythmia (RSA), which is differentiated by variable increases and decreases in heart rate. The frequency of RSA is influenced by right cardiac vagal input to the sino–atrial node of the heart. The aforementioned technique, while scanning the heart period to edit influences due to nonstationarities, uses time series analysis to derive the components of heart period within the respiratory frequency band for newborns (0.3 to 1.3 Hz). The natural logarithm of this variance produces the ϕ statistic. The ϕ statistic was computed on successive 30-s epochs from the baseline recordings. In the event of excessive movements from the infant that may have disrupted the EKG signal, artifacts were edited using the MXEDIT software program (Delta Biometrics). MXEDIT displays the heart period data in graphic and numerical form allowing for the visual identification of artifact. In cases where these periods of data were identified, they were edited using the absolute values with the range of the artifactual data for each individual infant. In cases of significant movement artifact, the data were either deleted and new trials created and averaged or the data were coded as missing.

DATA ANALYSIS

Relationships between maternal independent and infant outcome variables were determined by correlational and regression analyses. To reduce the number of independent variables (maternal emotionality) for further analyses, a factor analysis (principal components) was done to determine if there was an underlying structure for the maternal emotionality variables. The factor analysis produced two composite factors for both T1 and T2. To insure the reliability of the factors, only those factors whose eigenvalues were greater than one were selected. In addition, the emotionality variables were placed within a factor if they had a loading of at least .60 on a factor. The first composite factor consisted of variables that made up “trait” emotionality (STAI-trait, STAS-trait, NEO depression, NEO anxiety, and NEO hostility). The second composite consisted of variables that made up “state” emotionality (STAI-state, STAS-state, and BDI). The correlations between the trait and state composite factors at both T1 and T2 were r = .0000, p = 1.000, indicating that these two factors were measuring different phenomena.

Mediator Model

Based on previous research which indicates the buffering effects of social support during pregnancy, we tested whether an environmental process (social support) mediates the relationship between maternal emotionality and infant outcome. In order to examine this, a series of regression models were utilized following methods proposed by Baron and Kenny (1986). Mediators, as defined by Baron and Kenny (1986), are mechanisms that may causally account for an independent variable’s influence on a dependent variable. 

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Two infants were in a sleep state during their EKG recording. One infant’s cardiac vagal tone was significantly out of range from the other infants and thus was dropped from all subsequent vagal tone analyses. The other infant’s cardiac vagal tone was well within the range of the infants who were in an alert state and thus was retained for all vagal tone analyses. Dropping the infant did not change the stated findings compared to when all the infants were retained for all vagal tone analyses. Dropping the infant did not change the stated findings compared to when all the infants were included in the analyses. A decision was made to leave this infant out contingent on its cardiac vagal measurement compared to the other infants, not on whether it affected the final results.
FIGURE 1  Representation of mediator model with the proposed variable paths.

FIGURE 1  Representation of mediator model with the proposed variable paths.

of interest. The mediator function represents “the generative mechanism through which the focal independent variable is able to influence the dependent variable of interest” (Baron & Kenny, 1986, p. 1173). Figure 1 represents the proposed mediator model with the variable paths. The figure shows the paths for the regressions of the: (a) mediator variable (social support) on the independent variable (maternal trait and state composite factors) \( b_1 \), (b) dependent variable (infant cardiac vagal tone) on the independent variables \( b_2 \), (c) dependent variable on the mediator \( b_3 \), and (d) dependent variable on the independent and mediator variables simultaneously \( b_4 \).

To test for mediation, the following criteria must apply: The independent variable must affect the mediator, the independent variable must affect the dependent variable, and the mediator must affect the dependent variable when the dependent variable is regressed on both the independent and mediator variables together (Baron & Kenny, 1986). That is, the path between the independent variable and the dependent variable will become nonsignificant when the independent variable and the mediator are introduced simultaneously in the regression equation.

RESULTS

Table 1 shows the means and standard deviations for the adolescent predictor variables and Table 2 shows the means and standard deviations for the infant outcome variables used in the analysis.

Maternal Emotionality Variables and Infant Birth Outcome

A positive relation was found between the adolescent’s T1 trait composite score and infant Apgar at 5 min, \( r = .47, p = .01 \). This association indicates that...
higher levels of negative trait emotions during early pregnancy were related to higher scores on their infant’s 5-min Apgar assessment. The finding was in the opposite direction from that hypothesized. No other significant correlations were found between the maternal trait composite score or with the state composite score at T1 and the other infant outcomes. Neither maternal age at conception nor family socioeconomic status (SES) was significantly related to infant outcome as has been reported in previous studies.

The adolescent’s state composite score at T2 was positively correlated with the number of abnormalities on the newborn profile exam, *r* = .59, *p* = .001. The relationship between state emotionality at T2 and the number of abnormalities was in the direction hypothesized. No other significant relationships were found for T2 state or trait composites.

### Maternal Emotionality and Infant Cardiac Vagal Tone

The trait composite score at T1 was the only significant predictor of infant cardiac vagal tone, *r* = −.49, *p* = .01, accounting for 24% of the variance. This relation was in the hypothesized direction, with higher negative emotionality (trait) during early pregnancy predicting lower infant cardiac vagal tone.

No significant relations were found between the state composite score at T1 or the trait and state composite scores at T2 and infant cardiac vagal tone. In addition, there were no significant findings between infant cardiac vagal tone and the infant birth outcomes.

### Maternal Biological Measures and Infant Outcome

Maternal mean cortisol at T1 was a significant predictor of infant Apgar at 1 min, *r* = −.39, *p* = .04, and at 5 min, *r* = −.45, *p* = .01. Higher mean concentrations of maternal cortisol during early pregnancy predicted lower scores on their infant’s 1- and 5-min Apgar assessments. Maternal mean cortisol at T1 also predicted the number of resuscitation measures used on the infant, *r* = .45, *p* = .01. Mothers with higher mean cortisol during early pregnancy were more likely to have infants who needed resuscitation assistance at birth. Cortisol mean at T2 was not associated with any of the infant outcomes. There were no significant findings between maternal cortisol and infant cardiac vagal tone. Higher levels of maternal cortisol during the early months of pregnancy appear to predict which infants will have increased risks during and following birth.

### Environmental Processes as Mediators of Maternal Emotionality on Infant Outcome

In the current analysis, the independent variables were the two composite factors (trait and state emotionality), the mediator was social support (mean of ISSB), and the dependent variables were the overall “infant risk factor” and cardiac vagal tone. The mediator model testing whether social support mediated the relationship between maternal emotionality and the overall infant risk factor did not meet the criteria for mediation set forth by Baron and Kenny (1986). However, the model testing infant cardiac vagal tone as the dependent variable did. The results of this regression analysis are presented in Table 3.

As can be seen in Table 3, when the mediator was regressed on the independent variables (β1), the trait composite score at T1 approached significance, *p* = .06. Regression of the dependent variable on the trait emotionality composite factor showed a significant negative relation. As reported earlier, higher levels of trait emotionality were associated with lower levels of cardiac vagal tone (β2). The regression of the dependent variable on the mediator variable showed that a higher mean score on the ISSB was significantly negatively associated with cardiac vagal tone (β3). When the dependent variable was regressed on the independent and the mediator variable simultaneously, the relation between the trait composite factor at T1 and infant cardiac vagal tone became nonsignificant (β4). These regression findings fulfill the criterion that social support mediates the effects of adolescent trait emotionality on infant autonomic reactivity. As can be seen from Table 3, no significant mediator findings

### Table 2: Means and Standard Deviations for Infant Outcomes

<table>
<thead>
<tr>
<th>INFANT OUTCOMES</th>
<th>M</th>
<th>(SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational Age (weeks)</td>
<td>39.79</td>
<td>1.97</td>
<td>34–42.6</td>
</tr>
<tr>
<td>Infant Weight (g)</td>
<td>3259.97</td>
<td>441.86</td>
<td>2553–4199</td>
</tr>
<tr>
<td># of Risk Factors at Birth</td>
<td>.26</td>
<td>.52</td>
<td>0–2</td>
</tr>
<tr>
<td># of Risk Factors at 24 Hr</td>
<td>.15</td>
<td>.36</td>
<td>0–1</td>
</tr>
<tr>
<td>1-Min Apgar</td>
<td>7.56</td>
<td>2.04</td>
<td>2–9</td>
</tr>
<tr>
<td>5-Min Apgar</td>
<td>8.52</td>
<td>1.15</td>
<td>4–9</td>
</tr>
<tr>
<td>Abnormalities on Newborn Profile</td>
<td>.33</td>
<td>.73</td>
<td>0–3</td>
</tr>
<tr>
<td>Resuscitation</td>
<td>.92</td>
<td>.87</td>
<td>0–3</td>
</tr>
<tr>
<td>Vagal Tone</td>
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<td>.67</td>
<td>1.76–4.07</td>
</tr>
<tr>
<td>Infant Risk Factor</td>
<td>1.70</td>
<td>1.54</td>
<td>0–5</td>
</tr>
</tbody>
</table>

Maternal mean cortisol at T1 was a significant predictor of infant Apgar at 1 min, *r* = −.39, *p* = .04, and at 5 min, *r* = −.45, *p* = .01. Higher mean concentrations of maternal cortisol during early pregnancy predicted lower scores on their infant’s 1- and 5-min Apgar assessments. Maternal mean cortisol at T1 also predicted the number of resuscitation measures used on the infant, *r* = .45, *p* = .01. Mothers with higher mean cortisol during early pregnancy were more likely to have infants who needed resuscitation assistance at birth. Cortisol mean at T2 was not associated with any of the infant outcomes. There were no significant findings between maternal cortisol and infant cardiac vagal tone. Higher levels of maternal cortisol during the early months of pregnancy appear to predict which infants will have increased risks during and following birth.
were found with the state composite score at T1 or either of the composite scores at T2.

DISCUSSION

Four hypotheses were tested that examined the relations among adolescent maternal emotionality and biological processes during pregnancy and the birth outcomes of their infants. The first hypothesis examined effects of adolescent emotionality during pregnancy on infant physical outcomes at birth. Adolescent mothers who reported greater negative trait emotionality during pregnancy, particularly during early pregnancy (T1), had infants with more positive birth outcomes. These findings are contrary to what we hypothesized. As was stated previously, research shows that mothers who experience negative emotions during pregnancy increase their chances of delivering a low-birth-weight infant (<2500 g) and delivering a preterm delivery at <37 completed weeks gestation (Steer et al., 1992). So what is buffering our adolescent sample from these expected negative birth outcomes? We have two hypotheses for this observed “buffering effect.” The first, which we tested in our analyses and had to reject, was the hypothesized mediating effect of maternal social support on newborn risk. The other hypothesis concerns the demographics of the adolescent mothers in our sample. Most of the adolescents were in some form of community support that offered them preterm parenting classes. The preparation for childbirth they received from these classes may have provided them with psychological coping skills that assisted them with their negative emotionality. This type of support was not tapped by our support measure, which reflects the amount of social support the adolescent perceived from family and friends. Thus, certain types of support programs may be better at “buffering” the effects of prenatal stress on infant birth outcomes (Collins et al., 1993; Turner, Grindstaff, & Phillips, 1990).

An alternative explanation for these contradictory negative-emotion findings is that mothers may be using the way they feel as a catalyst for positive behaviors (e.g., eating a good diet, refraining from drugs, controlling stress) in order to improve the status and outcome of their pregnancy. It may be that an adolescent who responds with negative emotions (anxious, ashamed) rather than lackadaisically is reacting in a responsible, mature manner, instigating behaviors that will assist in enhancing the outcomes of this new situation rather than just doing nothing and avoiding responsibilities.

Although the mother’s trait emotionality was observed to predict positively infant Apgar scores, maternal trait emotionality predicted negatively infants with lower cardiac vagal tone. These relationships may provide an indication of the differential effects that the mother’s emotionality during pregnancy may play during the fetus’ development.

This brings us to our second hypothesis that examined the effects of adolescent maternal emotionality during pregnancy and infant cardiac vagal tone. The findings support our hypothesis: Mothers who reported greater negative trait emotionality during early pregnancy had infants with lower cardiac vagal tone. This finding was significant only for mother’s trait emotionality measured in the 1st trimester. One interpretation of these findings may be explained by the activation of the mother’s HPA axis during early pregnancy. The adolescent mothers who are experiencing high negative emotionality, reflective of stress, may have an HPA axis that is highly reactive (Gunnar et al., 1989; Sussman, Doran, Inoff-Germain, et al., 1997). Greater behavioral distress and negative emotionality is associated with greater HPA reactivity in both infants (Gunnar et al., 1989) and young adolescents (Sussman, Doran, Inoff-Germain, et al., 1997). During the 1st trimester (≤16 weeks gestation) a developing fetus is undergoing rapid differentiation of various organ and physiological systems, including the central nervous system.

Table 3. Regression Statistics for Environmental Mediators of Emotionality and Infant Cardiac Vagal Tone

<table>
<thead>
<tr>
<th></th>
<th>Time 1</th>
<th>Time 2</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>( \beta_1 )</td>
<td>( \beta_2 )</td>
</tr>
<tr>
<td>COMPOSITE MODEL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trait Model</td>
<td>.39††</td>
<td>-.49**</td>
</tr>
<tr>
<td>State Model</td>
<td>.10</td>
<td>-.37</td>
</tr>
</tbody>
</table>

Note. \( \beta_1 \) = regression of environmental mediator on Trait or State composite; \( \beta_2 \) = regression of dependent variable on Trait or State composite; \( \beta_3 \) = regression of the dependent variable on environmental mediator; \( \beta_4 \) = simultaneous regression of dependent variable on Trait or State composite and environmental variables. \( \beta_5 \) shows the association between the Trait and State composite scores and the dependent variable when the Trait or State composite and environmental variables are entered simultaneously.

\( \hat{p} \leq .06, \* \hat{p} \leq .05. ** \hat{p} \leq .01. \)
nervous system. By the end of the 3rd week of development, the fetus' heart is beating independently of outside stimulation. Any maternal biochemical perturbations (i.e., excessive secretions of hormones related to stress) at this critical developmental period may have adverse effects on the future development of the fetus. This interpretation was supported by our third hypothesis showing that infants born to mothers with higher concentrations of mean cortisol during early pregnancy were more likely to score lower in both of their Apgar assessments and also require resuscitation assistance compared to infants born to mothers with lower mean cortisol.

There are many proposed mechanisms that link maternal HPA reactivity and infant outcomes. A genetic component has been demonstrated in primates (Suomi, 1987). An event with a strong emotional experience associated with it can incite the proto-oncogene c-fos and other transcription factors to influence the expression of neurotransmitters that affect the mother and fetus (Post, 1992). Another mechanism involved in the transmission of maternal stress reactivity to the fetus involves placental corticotrophin-releasing-hormone (CRH) mRNA. A maternal stressor may activate fetal and placental CRH, which further stimulates the secretion of fetal ACTH and cortisol (Frim et al., 1988). Understanding these mechanisms will assist further research in the incorporation of intervention strategies that may help to control these maternal effects from negatively influencing the developing fetus.

Although it was found to be predicted by maternal emotionality at the first time of measurement, infant cardiac vagal tone was not observed to be related to other infant birth outcomes. This is contrary to the literature in which infants classified as high-risk had significantly lower cardiac vagal tone (Porges, 1992). One possible explanation for this null finding is that the infants in this sample were all primarily healthy. There was very little variability in the birth outcomes observed in the infants. In addition, the birth outcome variables particularly at this low level of risk are generally transient and are not predictive of future outcomes in infants. On the other hand, the measure of cardiac vagal tone is considered to be a sensitive physiological index of an individual’s vulnerability to stress (Porges, 1992) and predictive of long-term outcomes (Fox & Porges, 1985).

The disruptive challenges of an unplanned adolescent pregnancy may bring forth a dilemma that an adolescent may not have the skills or resources to manage, which may lead to a feeling of lack of control. The challenge for many adolescents is to find an effective strategy that alleviates the difficult situation and helps to establish the feeling of control in their lives. Previous research findings have described significant effects of environmental factors on the risks and consequences of adolescent childbearing. One of these factors, social support, is reported to mediate, or buffer, the adverse affects of maternal psychological stress and depression on infant outcomes (McAnarney & Stevens-Simon, 1990). This is represented in our proposed mediator model (see Figure 1). In support of our fourth hypothesis, the environmental process of social support did appear to mediate the effects of maternal emotionality on infant cardiac vagal tone. Mothers who reported greater emotionality during pregnancy but perceived receiving more support from family and peers were less likely to have infants with low cardiac vagal tone than similar mothers who received no support. The perceived social support in the mothers accounted for the relation found between maternal emotionality and infant outcome. Although social support in our model may not have had a “strong” direct mediating effect (did not reduce the correlation of the independent and dependent variable to zero (path $P_1$)) it does influence the strength of this correlation by reducing its significance (from $r = -0.49$ to $r = -0.30$). It may be that social support is one of a group of influential variables that mediates the effects of maternal negative emotionality and infant outcome. Through consistent social support, the adolescent mother may learn better means to cope with stress during pregnancy.

The results of this study reinforce the need to continue research on the mechanisms involved in adolescent pregnancy that influence poor infant outcomes. Though the current study was based on a small sample size which limited statistical power and generalizability, the results suggest that early infant outcome appear to be the result of the complex interplay of biological and social factors occurring during early fetal development. Future longitudinal studies with larger samples that measure multiple biobehavioral variables (contextual, physiological, and psychological) as we did, should find similar results, supporting the urgency of researchers to implement and continue addressing studies of this biopsychosocial nature. Once a clearer understanding of the complex interplay of these processes is attained, researchers will have a better understanding of the relationships between maternal mental and physical health and infant outcomes. Understanding these relationships will assist in the design of appropriate prenatal interventions that are directed toward reducing both maternal and neonatal health risks.

The issues pertaining to the effects of community social support should be examined further. Although our research measures failed to assess directly the influence that community social support groups may offer, our small sample did provide evidence for the
mediating effects of social support networks encompassed of family and friends in parenting adolescents. Larger studies attempting to assess further these effects may find stronger significant findings if they include the measurement of extended support networks that are available to the adolescent mothers.

NOTES

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