Sending and Receiving: Biochemical Communication of Emotions Between Prenate and Mother: A Call for Early Intervention

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Full Text: Headnote ABSTRACT: This review article presents evidence for prenatal biochemical communication involving the mother, the placenta, and the prenate, and calls for prenatal intervention for at-risk dyads. The concept of prenatal biochemical communication is based on the view that the develoment of the self starts prenatally and is continuous and incremental. The study of prenatal programming has led to an understanding that the prenate and mother reciprocally influence each other via the placenta, which also provides many of its own contributions to the biochemistry of pregnancy, and that these effects may have long-range consequences in determining the course of adult health. Recent research has expanded this understanding to include the biochemicals associated with emotions and their transmission between the prenate and the mother, mediated by the placenta. After briefly touching on prenatal stress, which has been extensively studied, the review focuses on recent studies of maternal depression and PTSD. Hyperactivity, which may be a generic marker of prenatal stress response, is also briefly considered. The review concludes with a call for prenatal intervention for at risk mother-prenate dyads. KEY WORDS: Biochemical communication, prenatal programming, pregnancy, maternal depression, PTSD, prenatal intervention. INTRODUCTION Psychological research has historically framed questions concerning the influence of the caregiver's emotions on the child in terms of the child's postnatal experience. More recently, however, evidence has been accumulating that the mother's responses in the prenatal environment play a significant role in the development of the child, both pre- and postnatally. This paper examines the accumulating psychobiological evidence for prenatal physiological transmission of maternal emotional traits to the prenate. The research presented highlights the possibility of, and the need for, early intervention with at-risk individuals. After a summary of the arguments for the incremental development of the self beginning in the prenatal period, and hence for prenatal learning, evidence will be reviewed for the transmission of maternal biological markers for depression and PTSD to the prenate, increasing the prenate's risk for those disorders. In addition, the effects of maternal stress will be briefly reviewed, as well as evidence for fetal hyperactivity in response to various stressors. A REVIEW OF THE LITERATURE Scholars from disciplines as diverse as neurobiology, developmental psychoneuroendocrinology, and psychoanalysis have expressed the notion that the sense of self exists prenatally, in some early, inchoate form (Bion's proto-mind, Winnicott's psyche-soma, Damasio's mammalian proto-self) (Bianchedi et al., 1997; Damasio, 1999, pp. 16-19, 154-156; Winnicott, 1958, p. 191), quite different from the reflexive, self-aware, cognitive, verbal self that is often implied in discussions of the development of the self. These scholars also believe that this early self develops in interaction with its environment. Just as the postnatal development of the brain's neural pathways is seen as shaped by experience during and after birth (Schore, 2001a, 2001b, 2001c, 2003a, 2003b), learning also occurs prenatally: "Study of the prenatal ontogenesis of behavior suggests that the mind will emerge in an immature form and that stimulation received in utero, and the behavior emitted, will play an important role in the development of the fetus" (Hepper &Shahidullah, 1994); although the experiences may be different from our classic understanding of learning (for example, habitual washes of hormones from the mother's emotions that result in long-term, possibly lifelong, changes to the prenate's response patterns). Rather than verbal and cognitive, this learning is nonverbal, probably biochemical as well as neural, and is stored as cellular or procedural rather than explicit memory. The development of a sense of self can be viewed as being like an everexpanding sphere, starting with the prenatal psyche-soma and the physiological core of

hormonal and other exchanges between prenate/ infant and mother, adding new layers as the infant/child continues to develop new capabilities (emotional/limbic maturity, ego and selfawareness, awareness of others, cognition, boundaries, somatic awareness, etc.), eventually resulting, if all goes well, in a multivalent, nuanced individual who interweaves transmission and reception with other selves in a similarly expanding and deepening intersubjective field, in all the modalities an individual is capable of (Trevarthen, 1993). This developing prenatal being is constantly subject to the influence of its environment, which consists of several worlds within worlds. The local environment, immediately surrounding the prenate, is bounded by the amnion containing the amniotic fluid and the prenate and its umbilical cord, which links the prenate to the placenta. The amniotic sac, surrounded by the placenta, is inside the uterus into which the placenta has extended countless fingers, or villi. The placenta acts both as an independent source of biochemical responses and as the intermediary between the internal maternal environment and the prenate (Albrecht &Pepe, 1990; Castellucci, Kosanke, Verdenelli, Huppertz, &Kaufmann, 2000; Cross, 1998; Cross, Baczyk et al., 2003; Cross, Simmons, &Watson, 2003; Maranghi, Macri, Ricciardi, Stazi, &Mantovani, 1998; Mayhew, 2001; Page, 1993; Rurak, 2001; Steinborn, Rebmann, Scharf, Sohn, &Grosse-Wilde, 2003; Wadhwa, Culhane et al., 2001). The uterus is in turn inside the mother's body, whose sounds, movements, and biochemistry directly affect the prenate. The final environment that affects the prenate is the larger world outside the mother's body. None of these environments is self-contained: they all interact, directly or indirectly with one another. As the prenate's senses develop, the prenate has two sources of experience: its own direct sensory experience, which includes stimuli inside and outside the mother's body, and the mother's constant stream of biochemical responses to her environment, almost all of which pass through the placenta and umbilical cord to create biochemical responses in the prenate. In this sense, you could say that the prenate has twice as much experiential input as a postnate, since it receives information from another being's biochemistry in addition its own direct sensory experience and its own developing biochemical responses to that experience (Wirth, 2001, p. 40). Once again, science confirms folk wisdom, which has viewed the prenate as deeply influenced by the mother's experiences during pregnancy, in stark contrast to current practice in the USA that calls upon the modern mother-to-be to carry a full workload as long as possible, regardless of the stress she might be experiencing. If these environmental experiences are favorable, development proceeds in an optimal manner. However, the environment may be problematic, as a result of possible detrimental influences affecting the mother, and hence the prenate, such as: * Substance abuse (O'Leary, 2004; Pullen, 2004), smoking (Ernst, Moolchan, &Robinson, 2001; Hellstrom-Lindahl &Nordberg, 2002; Klesges, Johnson, Ward, &Barnard, 2001; Zeskind &Gingras, 2005) * Emotional problems, including PTSD (Field, Diego, HernandezReif et al., 2004; Van den Bergh, Mulder, Mennes, &Glover, 2005; Yehuda et al., 2005) * Medication (Jacqz-Aigrain &Koren, 2005) * Domestic violence (Corry, 1999; Gilliland &Verny, 1999; Huth-Bocks, Levendosky, &Bogat, 2002; Lent, Morris, &Rechner, 2000) * Poor prenatal medical care (Goffinet, 2005) * Malnutrition (Aerts &Van Assche, 2003; Roseboom et al., 2001; Ross &Desai, 2005) * Social Stressors-e.g. poverty, legal problems, isolation (Owen, Andrews, &Matthews, 2005; Van den Bergh et al., 2005; Wadhwa, Dunkel-Schetter, Chicz-DeMet, Porto, &Sandman, 1996) * Pollution (Houlihan, Kropp, Gray, &Campbell, 2005). These risk factors are interrelated (for a more complete list of interrelated risk factors, see Gold, 2000, pp. xxi, 25; Martinez, Partridge, &Taeusch, 2004, p. 107), and they in turn create a high risk of being premature, which in its turn increases risks of: * Brain damage * Defects in sight and hearing and other sensory issues * Feeding difficulties * Attachment problems (Martinez et al., 2004, p. 110) Detrimental prenatal influences are cumulative and interact with each other. For example, oxygen deprivation caused by the mother's smoking will make the prenate more fragile and more at risk for premature birth (Martinez et al., 2004, pp. xxi, 25). Prenatal Programming For the period from birth to two years, research is bringing attention to the importance of the countless microinteractions that can be as influential, if not more so, as more visible, large-scale trauma, in setting the direction of an individual's development. One researcher, Karlen Lyons-Ruth, has called these microinteractions "the silent traumas", which result in greater risk of dissociation at age 19 than

overt traumas such as abuse (Lyons-Ruth, 2003). It seems likely that in the prenatal period also, the repetitive daily patterns of maternal biochemicals, activities, and sounds shape the prenate as much as overtly traumatic events. The nine months from conception to birth encompass the most rapid period of change the human organism ever experiences. During this time the prenate changes physically from day to day, developing new strengths and corresponding vulnerabilities. In addition, researchers are increasingly discovering that prenatal experience has long-range effects on adult life. Evidence for prenatal programming is growing with regard to the impact of maternal nutrition, stress, substance abuse, medications, and emotions on the prenate's life trajectory. Research on prenatal humans is highly sensitive, given the potential impact on both the mother and the prenate, with the result that much of the work has used other placental mammals as animal models. Unfortunately, in placental mammals there is considerable interspecies variability in the processes leading up to birth, so that it is necessary to exercise caution in extrapolating from animal results to human prenatal development. For example, the rat's brain is only 12% adult weight at birth, meaning that much development takes place postnatally; the brain of rhesus monkeys is more or less mature at birth; and the human brain has a protracted postnatal development (A. C. Huizink, Mulder, &Buitelaar, 2004). Rodents, sheep, and nonhuman primates have been the main species studied, and it is becoming clear that the molecular mechanisms of transduction vary from species to species. In particular, information concerning placental interactions with the maternal and fetal biochemistry is still not at a level of detail sufficient to permit accurate cross-species biological comparisons, though there is enough commonality that research in one species provides insight and raises questions for similar research in a different species (Anja C. Huizink, Mulder, &Buitelaar, 2000)]. Historically, prenatal research has taken place in two different disciplines: biology and psychology. These disciplines have traditionally had little contact with each other, so that research on physiological and physical development has been independent of research on behavioral development. The information from these two fields is now beginning to converge, as the findings of biological research are integrated into behavioral and psychological studies. Studies are beginning to incorporate both behavioral measures, such as neonatal Brazelton scores, with physiological measures, such as autonomic arousal. The mechanisms by which the biochemical exchanges between the prenate and the mother shape the development of the prenate, not just in utero, but well into adult life, are receiving increasing attention from developmental biologists. In particular, the placenta both mediates and originates this biochemical "conversation" by transmitting or secreting a large number of substances, each of which has its impact on both the mother and the prenate: * Hormones, including stress hormones; other biochemicals such as neuropeptides * Oxygen, carbon monoxide, carbon dioxide * Most medications, including psychotropics * Cells from the prenate, such as red blood cells * Wastes from the prenate * Nicotine, alcohol, caffeine * Heroin, methadone, cocaine, marijuana * Viruses for chicken pox, mono, HIV, hepatitis B * Syphilis bacterium * Pollutants * Nutrients, vitamins, minerals For a full understanding of the impact of prenatal experience, not only would each of these exchanges need to be studied independently, in the classic scientific method, but all of their (exponentially complex) interactions would be investigated in a yet-to-be-established methodology of complexity. There are, however, a number of areas in which single-focus research is taking place: the effects of maternal alcohol consumption (Zhang, Sliwowska, &Weinberg, 2005), the effects of maternal substance abuse (Huestis &Choo, 2002) the effects of maternal smoking (Cornelius &Day, 2000), the effects of maternal malnutrition (Ross &Desai, 2005), the effects of maternal stress (Huizink, Rubles de Medina, Mulder, Visser, &Buitelaar, 2003), the effects of maternal PTSD (Yehuda et al., 2005), and the effects of maternal depression (Field, Diego, Dieter et al., 2004). In some instances, there is already a substantial body of work; in other instances, work is just beginning. Transduction of Emotions This paper will focus on the emotional biochemistries of prenate and mother, in particular the more recent developments concerning the transduction of maternal depression and PTSD, with additional material on stress, hyperactivity, and the prenatal effects of antidepressants. The field of psychoneuroedocrinology has shown that there is no separation between the emotional and the somatic: Every emotional response is inseparable from its

accompanying physiological concomitants: "I view emotions as biological functions of the nervous system" (LeDoux, 1998, p. 12). "Emotions are complicated collections of chemical and neural responses, forming a pattern; all emotions have some kind of regulatory role to play...emotions are biologically determined processes, depending on innately set brain devices" (Damasio, 1999, p. 51). Even though the prenatal infant has traditionally not been seen as having emotions, physiological processes taking place both within the infant and between the mother and infant are similar to processes that are identified as concomitants of post-natal emotions. Wirth explicitly states that the pre-nate communicates with her/his mother biochemically as well as through the senses (Wirth, 2001, p. 6) and goes on to say, "The impact of this prenatal communications system is greater on emotional tone than on cognitive abilities, because the emotional responses originate in the older parts of the brain structures that are relatively more mature at an earlier stage of fetal development" (Wirth, 2001, pp. 47-48). Although this metaphor of a biochemical dialogic communication between mother and prenate captures the essence of transduction, it must be remembered that in fact there is a third participant in this conversation, the placenta. From the perspective of infant-mother communication, these neuroendocrinological feedback mechanisms might be considered to be the prenatal equivalent of caregiver-child contingent communication, in which the nature of the caregiver's response to a communication from the child is contingent on the nature of the communication to which it is responding, and vice versa. When, for example, the caregiver amplifies the happy baby's body language and sounds in an attuned fashion, with the result that the infant in turn manifests a heightened pleasure, which again is reflected by the caregiver, contingent communication has taken place. Similarly one could say that when the mother's cortisol rises in response to the infant's rise in cortisol, contingent communication has taken place. It is important, however, to remember that the prenate is not a totally passive recipient of maternal biochemicals, although recent work on the intergenerational transmission of stress, depression, and PTSD has focused on the flow from mother to prenate, rather than from the prenate to the mother. In addition, the placenta is an extremely active third party in the conversation, sometimes communicating with the mother, sometimes with the prenate, and sometimes with both (Rurak, 2001), resulting in a far more complex flow of signals and feedback loops than a simple one-on-one model would indicate. Stress This complexity is clearly seen in the area of stress, which is the best-studied and will be briefly touched on in this paper. On the biological side, work is still in progress to understand the intricacies of maternal-placental-prenate biochemistries. Again, much of this research has employed animal models, requiring caution in extrapolating the results to humans. The role of the adult hypothalamic-pituitary-adrenal (HPA) axis in stress is fairly well understood, but in pregnancy, its functioning changes over time. For example, there is a surge in corticoreleasing hormone (CRH, also known as corticoreleasing factor or CRF) and cortisol in the late third trimester, prior to birth, that originates with the placenta rather than the mother (Thomson, 1998). Adding to the complexity is the fact that the fetal HPA axis does not function in the same manner as the adult and also changes how it functions over the course of development (Gitau, Fisk, Teixeira, Cameron, &Glover, 2001; Mesiano &Jaffe, 1997). In addition, it has become common to administer synthetic glucocorticoids (e.g. dexamethasone) to mothers at risk for preterm delivery. On the one hand, this intervention speeds up the development of the prenate's lungs, thus increasing its chances of survival in case of premature birth; on the other hand evidence is mounting for altered development of the nervous system (Weinstock, 2005) and of the fetal HPA axis (Matthews, Owen, Banjanin, &Andrews, 2002; Wadhwa, Sandman, &Garite, 2001), and for negative long-range effects such as increased risk of cardiovascular disease, insulin resistance, and diabetes in later life (Owen et al., 2005). Another effect of maternal antenatal stress and anxiety is constriction of the uterine blood vessels, decreasing the flow of oxygen and nutrients through the placenta to the fetus, possibly accounting for the association of stress and low birthweight (Glover, 1999). In rats, stress decreased the invasiveness of the placental trophoblasts into the wall of the uterus, possibly setting the stage for later preeclampsia (Kanayama, Tsujimura, She, Maehara, &Terao, 1997). The blood chemistries of their neonates reflected the blood chemistries of those mothers who scored high on trait anxiety: the mothers had high

norepinephrine and low dopamine; the neonates had low dopamine and serotonin (Field et al., 2003), indicating a possible prenatal diathesis towards anxiety in the neonate. This study will be discussed in more detail in the section on depression. Antenatal maternal stress also has effects on behavior, both immediately and later. To date there have been over 15 ultrasound studies recording fetal behavior during maternal stress. These studies had varied results, depending on the age of the prenate. In the first half of pregnancy, spontaneous motor activity showed no significant effect, but near-term fetuses (27-28 weeks gestational onwards) demonstrated increased wakefulness, increased fetal heart rate variability, and increased motor activity (Van den Bergh et al., 2005). Antenatal maternal stress also has long-range effects on behavior, as demonstrated by observation or ratings by teachers or parents. These effects manifest generally in regulatory issues in all spheres: behavioral, cognitive, and emotional. Neonates of antenatally stressed mothers, for example, have lower scores on the Neonatal Behavior Assessment Scale; infants are highly reactive and more irritable and difficult; children have more attention problems and hyperactivity as well as lower grades; adolescents demonstrate poor impulse control and poorer performance on the Child Behaviour Checklist (Van den Bergh et al., 2005). Depression In contrast to the focus on the biology and physiology of the HPA axis in studies of prenatal transduction of anxiety, depression in mothers has been more studied on the psychological and behavioral side, where its negative effects on infants were attributed to postnatal interactions with the depressed mother (Cohn, Campbell, Matias, &Hopkins, 1990; T. Field, 1995; Field, Healy, Goldstein, &Guthertz, 1990; T. M. Field, 1995) but research on the physiological aspects, or maternal-fetal transduction, of depression, is more recent, following on earlier studies using animal models to suggest how neurotransmitters interact to produce depression (Weiss, Bonsall, Demetrikopoulos, Emery, &West, 1998). The work of Tiffany Field at Touch Research Institutes, University of Miami School of Medicine, has focused on the behavioral, physiological, and biochemical aspects of the transduction of depression from mother to fetus. In prior work, Field et al. explored the postnatal characteristics of infants of depressed mothers (see above), and in 1995 Field proposed a model in which the prenatal environment was implicated in the phenomenon of depressed-seeming neonates (T. Field, 1995). A preliminary note published in 1998 reported that lower vagal tone and right frontal EEG asymmetry characterized infants of depressed mothers. In a study published in 1999, she and her associates tested neonates of depressed mothers vs. neonates of non-depressed controls within 24 hours of birth, and found that the infants' biochemistry paralleled the mothers', with depressed dopamine and elevated cortisol and norepinephrine levels (Lundy et al., 1999). These results were confirmed in a subsequent study, in which Field compared the biochemistry of 80 mothers who were categorized as depressed or intrusive to 40 non-depressed mothers with respect to their levels of cortisol, dopamine, norepinephrine, and epinephrine in the late midtrimester of pregnancy (18-24 weeks gestational age) (Field et al., 2001). The results indicated that the depressed/ intrusive mothers had higher levels of cortisol and lower dopamine (the "feel-good" neurotransmitter) than the control subjects. The neonates born to these mothers had the highest cortisol levels, the lowest dopamine and serotonin levels, the most asymmetrical EEG patterns, lower Brazelton scores (in particular on habituation, depression, motor activity, and orientation), more complications surrounding their birth, and more irregular sleep patterns than the neonates of non-depressed mothers. These results were further refined by a 2002 study that focused on the influence of socio-economic status (SES) and ethnicity on the effects of depression on the fetus and neonate in 86 mothers of who were Latina or African-American (Field, Diego, Hernandez-Reif, Schanberg, Kuhn et al., 2002). Not surprisingly, the neonates of mothers who were of middle SES had lower norepinephrine and fewer postnatal complications and were less excitable than the neonates of mothers in the lower range of SES. Antenatally, the middle SES mothers had higher norepinephrine levels (in contrast to their infants' lower postnatal levels of norepinephrine), were older, had more social support, and lower anxiety, depression, and anger scores. In terms of ethnicity, Latina mothers had a higher SES and higher antenatal norepinephrine scores than the African-American mothers, and their fetuses were more active. After birth, these infants had higher dopamine levels and lower cortisol levels than the African-American infants,

and the mothers had higher serotonin scores as well as higher anger scores than their African-American counterparts. The correlation of frontal cortex activity with depression and biochemistry was carried out in another study published in 2002 (Field, Diego, Hernandez-Reif, Schanberg, &Kuhn, 2002) in which 52 second-trimester women were given EEG's to determine which hemisphere showed the greater frontal activity. A discrimination analysis using maternal antenatal depression scores and biochemistry correctly assigned 74% of the women to the correct hemisphere: women with higher depression scores and lower dopamine levels had greater activity in the right frontal cortex. Again, the neonates of these mothers also had more activity in the right frontal cortex, as well as lower dopamine and serotonin levels and lower Brazelton scores. Another study published in the same year found that a small population of neonates of depressed mothers (10, vs 10 neonates of non-depressed mothers) demonstrated slowed habituation to their mother's face and voice, as well as no preference for the mother after habituation (Hernandez-Reif, Field, Diego, &Largie, 2002) A 2003 study categorized depressed mothers by styles of interaction with their 3-month-old neonates: withdrawn, intrusive, and good. A retrospective analysis was then done of their antenatal depression and anxiety scores, and their biochemistry. Interestingly, these groups did not differ in their antenatal biochemistries: all three groups had elevated cortisol, epinephrine, and norepinephrine. Both mothers and neonates showed greater right frontal cortex activity in EEG's after birth, yet the infants of the "good interacting" mothers had better Brazelton scores and less indeterminate sleep, despite the similarity of all three groups' scores, frontal laterality, and biochemistry. In this case, it appears that the physiological transmission of a non-optimal emotional trait (depression) can be mediated by the interactions between the mother and infant, providing support for plasticity on the behavioral as well as on the neurological levels. Another study published in 2003, attempting to disentangle the effects, often confounded in studies of stress, of anxiety, depression, and anger, measured a group of high-anxiety mothers (who also scored high in depression and anger), and had similar results: the biochemistry of the neonates was significantly predicted by the mother's antenatal biochemistry and EEG. The mothers had elevated norepinephrine and low dopamine antenatally; their neonates had lower dopamine and serotonin, as well as higher levels of activity and lower birth weights (Field et al., 2003). The role of cortisol and the neurotransmitters norepinephrine, epinephrine, dopamine, and serotonin was the focus of a study published in 2004, measuring 45 depressed and 47 non-depressed mothers of mixed ethnicity antenatally and postnatally, which found that all biochemical variables except serotonin decreased for all subjects postnatally. The findings continued to support the biochemical transduction of emotional patterns from mother to fetus and demonstrated the specificity of the relationships between specific biochemicals in the mother and prenate. Other researchers have shown that specific biochemical markers are predictive of other, nonemotional characteristics also: elevated maternal corticoreleasing hormone (CRH-a precursor of cortisol) not only predicts elevated cortisol in the neonate, it also predicts prematurity (Wadhwa, Porto, Garite, Chicz-DeMet, &Sandman, 1998) (for results indicating CRH does not predict preterm delivery, see Berkowitz et al., 1996); elevated maternal norepinephrine predicts lower birth weight due to its constrictive effect on the mother's uterine arteries (Giannakoulopoulos, Teixeira, Fisk, &Glover, 1999). In this study, maternal antenatal cortisol and norepinephrine were both significant predictors of cortisol levels in the neonate; there were no significant predictors of the neonate's norepinephrine; neonatal epinephrine was significantly predicted by antenatal maternal epinephrine; elevated maternal antenatal norepinephrine was a significant predictor for depressed dopamine in the neonate, and there was no significant predictor for neonatal serotonin. These results were confirmed in a study published in 2004 in which both mothers with depressive symptoms (N = 70, mixed ethnicity) and their neonates had higher cortisol levels and lower dopamine and serotonin levels (Field, Diego, Dieter et al., 2004). In addition, they were more likely to give birth prematurely and the babies were more likely to be low birthweight. In contrast to prior studies, the design was prospective and longitudinal, with repeated assessments of the mother during the prenatal period, and more comprehensive studies of mother and neonate after birth. The neonates were measured within 24 hours after birth and again showed more lengthy habituation, lower autonomie stability, lower vagal

tone, and greater motor activity. In this study, behaviors were also found to be significantly predicted by maternal depression, including activity (significantly predicted by antenatal maternal cortisol), and habituation (significantly predicted by antenatal maternal norepinephrine). This series of studies by Field and colleagues has compensated for fairly small groups of subjects by replicating measures from study to study, consistently showing that biochemistries of depressed or anxious mothers are transmitted to the prenate. Unfortunately, some reports do not specify explicitly whether or not mothers on drugs, psychotropic as well as recreational, were excluded. The groups, despite their small size, have consistently been of mixed race and ethnicity. Because of the concerns noted above related to doing research on pregnant women, the researchers elected to avoid invasive procedures such as obtaining blood samples, and the biochemical assays were done on urine samples, which may not always be accurate indicators of plasma hormones. In extrapolating backwards from neonate to prenate, it is important that neonatal testing be done immediately after birth, although it also must be recognized that the birth experience itself might have an effect. In the more recent studies, Field has been a pioneer in bringing together physiological evidence (e.g. vagal tone); behavioral evidence (e.g. Brazelton scores), and biochemical evidence (e.g. neurotransmitter levels). This convergence of disciplines constitutes the next step in studies of prenatal programming and transduction of emotional predispositions from mother to prenate. In an unrelated study, evidence is presented indicating that the indeterminate sleep patterns noted in Field's studies may be the result of interference with optimal neuronal development in the suprachiasmatic nucleus (SCN) during the prenatal period. The SCN controls circadian rhythms and disturbance of its development leads to changes in rhythmicity that are similar to those commonly associated with depression (Kennaway, 2002). This disturbance of rhythms might be another aspect of transduction of depression from mother to prenate. The mechanisms of this aspect of prenatal programming have not been specified-it would be interesting to know if they are also a consequence of the abnormal maternal biochemistries identified by Field et al. Anti Depressants: Selective Serotonin Reuptake Inhibitors SSRIs, the most commonly used antidepressants, are increasingly being prescribed for other disorders than major depression, such as dysthymia, anxiety disorders, violent and aggressive behavior, chronic pain, obsessive-compulsive disorder, attention deficit/hyperactivity disorder, and nicotine dependence (Julien, 2002, pp. 413-455). Given the impact of maternal depression on the prenate, it is comforting to know that maternal depression can be treated with psychotropic medications. However, the question is then raised of what effect these medications might have on the prenate. What are the costs of prenatal depression to both mother and prenate versus the costs to the prenate of the impact of antidepressants? The data are not clear. Consistent with the practice of conducting preclinical trials of medication only on males, psychotropics are rarely tested on pregnant or breast-feeding women before release, even though 10-16% of pregnant women are reported to experience depression (Goldstein, 1995). A study of 267 women who took SSRIs during pregnancy found no increased risk for major malformations (teratogenesis), miscarriage, stillbirth, or prematurity (Kulin et al., 1998). However, other studies have found slight risk for prematurity and low birthweight as well as possible neonatal disturbances representing increased serotonin activity as a result of withdrawal from the SSRI (Jacqz-Aigrain &Koren, 2005) or, at the other extreme, as a result of serotonin toxicity (Koren, Matsui, Einarson, Knoppert, &Steiner, 2005). At least one study states the opinion that maternal depression is a greater risk to the prenate/neonate than the use of SSRIs (Koren et al., 2005). Unfortunately, few of the studies focus on long-term effects or on the possibility of more subtle neuropsychological deficits, which might be expected from abnormally high serotonin levels during development of the nervous system. One study did find disruptions in a wide range of neonatal neurobehavioral characteristics (startles, behavioral states, sleep patterns) (Zeskind &Stephens, 2004), and another study found lowered reactivity to pain and increased vagal tone (briefer sympathetic arousal in heart rate after heel stick) (Oberlander et al., 2002). Another limitation of these studies is that they for the most part do not consider drug combinations (Oberlander et al., 2004), whereas dual-diagnosis clients (and other users of psychotropic medications) often are prescribed three or four psychotropics concurrently. In most studies, if the drug doesn't

cause immediate damage, such as teratogenesis, prematurity, death of the prenate, or severe postnatal symptoms in the neonate, it is declared safe without consideration of possible longer-term effects (Einarson &Einarson, 2005). PTSD PTSD is the most recent mental health issue to be examined for possible transduction. At present, only one study has been done, by Rachel Yehuda et al. (2005), which indicates a new direction for work on transduction. In prior studies, Yehuda demonstrated that the biochemistry of PTSD is different from the biochemistry of classic stress in that levels of cortisol are not elevated, but are lower than normal, and also fluctuate more widely (Yehuda, 2001). As in the case of stress and depression, a specific biochemical profile may be associated with a clinical disorder, and, if so, this profile could be examined in neonates of mothers with PTSD-associated biochemistry. In a preceding study, which examined risks for anxiety, depressive, and post-traumatic stress disorders in adult children of Holocaust survivors who had PTSD, it had been found that they were also at risk for PTSD, with significantly lower 24-hour mean urinary cortisol excretion, and that general parental exposure to trauma, rather than PTSD specifically, was significantly associated with lifetime depressive disorder in their offspring (R. Yehuda, Halligan, &Bierer, 2001). The current study examined women who had been pregnant while in or close to the World Trade Center at the time of the building collapse on 9/11/2001. The results are based on a group of 38 women out of a cohort of 187 who responded to publicity recruiting subjects for a longitudinal prospective epidemiological study on the effects of fetal growth and other effects. In a separate report, the neonates of the original 187 mothers were found to have lower birthweights than the babies of mothers who had not been directly involved. A subgroup of 38 women was divided into 2 groups, those who scored positively on the Post-Traumatic Stress Disorder Checklist (PCL), and those who did not (group N's not reported; groups otherwise matched) and reported severity of depression was assessed with the Beck Depression Index. When the mothers came for their infants' 9-month checkup, they were asked to collect saliva samples (a less reliable method than urinary samples, which are in turn less reliable than blood samples) for themselves and their babies. The results showed that mothers with PTSD had lower cortisol than non-PTSD mothers, that the infants of mothers with PTSD had significantly lower cortisol than the infants of non-PTSD mothers, and that there was a significant effect for mothers who had been in their third trimester, but not for mothers who had been in the first two trimesters of pregnancy. The fact that there was a difference by trimester points to the likelihood that the difference in cortisol was not a result of intrinsic genetic difference. In contrast to Field's results, Yehuda et al found no correlation with severity of depression. The fact that the infants were 9-12 months old, whereas the neonates in Field's studies were observed in the first day after birth, weakens the link to prenatal programming and maternal transduction. Furthermore, Yehuda et al do not mention infant gender, although a study of children aged 8-18 found that urinary levels of cortisol and epinephrine in boys on first contact with a Level 1 trauma center predicted close to 7-10% of the total population's variance in PTSD symptoms six weeks later (Delahanty, Nugent, Christopher, &Walsh, 2005). Nonetheless, this study opens up a new direction for continuing to examine the possibility of maternal-fetal transduction of PTSD. Such studies might follow the model of the depression studies in collecting data immediately after birth, in using observations that were behavioral and physiological as well as biochemical, and in using urinary as opposed to salivary samples. If further research does indeed bear out the transduction of PTSD as well as chronic stress and depression, it will provide yet another reason to reshape the "big-bang", single-event view of trauma still embodied in the DSM IV-TR definition of PTSD (Bowman, 1999). As Yehuda says, "Cumulative lifetime stress, particularly a history of exposure to trauma, is a very important risk factor for PTSD," (Yehuda, 1999). Hyperactivity Although Yehuda unfortunately focused only on one physiological variable in the possible transduction of maternal PTSD to the prenate, without examining behavior at all, there is one prenate behavioral characteristic that appears across diagnostic categories and may signify prenatal response to a wide range of stressors. Increased fetal activity has been seen as an immediate response in near-term prenates of women with high stress/anxiety (Van den Bergh et al., 2005). In addition, Field et al. found that maternal antenatal cortisol predicted neonatal movement and a greater time in indeterminate sleep (sleep behavior that does not

conform to specific codable behavior patterns) (Field, Diego, Hernandez-Reif, Schanberg, Kuhn et al., 2002), after finding in an earlier study that maternal antenatal cortisol predicted abnormal reflexes in the neonate, whereas neonatal excitability and withdrawal was predicted by maternal antenatal depression scores (Lundy et al., 1999). The finding that maternal antenatal cortisol levels predict neonatal levels of motor activity was replicated in a study published in 2005 (Field, Diego, Hernandez-Reif, Gil, &Vera, 2005). Another study found Brazelton Neonatal Behavior Assessment Scale scores indicating lower motor organization, lower autonomie stability, and higher withdrawal, as well as showing more state changes and less time in quiet and active alert states (Field et al., 2003). In terms of fetal, as opposed to neonatal, behavior, a Swedish study observed near-term prenates (37-40 weeks gestational) using ultrasound and cardiotocography. Mothers were divided into low and high anxiety groups. In this study the only significant difference found was in fetal heart rate variability while the prenate was in the state corresponding to pattern D (state 4F/active awake) (Sjostrom, Valentin, Thelin, &Marsal, 2002), which replicates findings by Van den Bergh et al (Van den Bergh, Vandenberghe, Daniels, Casaer, &Marcoen, 1989). Anecdotal reports from mothers having sonography and amniocentesis suggest that increased activity on the part of the prenate is a common response to these procedures. It might be that increased fetal activity represents the prenate's own response to stress (cf. verification that by 20 weeks gestational, the prenate is producing its own cortisol in response to invasive procedures independently of the mother, Gitau et al., 2001). A case can be made that increased activity represents the prenate's independent response to stress, and it would be interesting to further investigate the correlation of stress, motor activity, fetal heart rate variability, and fetal and maternal cortisol. It is tempting to speculate that the origins of ADHD may lie in the prenatal response to elevated antenatal maternal cortisol, in terms of both motor activity, less organized behavioral states, and longer periods of active awake behavior. One researcher has argued that the origins of ADHD lie in the developing brain (Zametkin &Liotta, 1998), but that discussion lies outside the scope of this survey. CONCLUSION In reviewing studies of transduction of maternal emotional traits to the prenate, we have found well-documented evidence of changes in biochemical markers, either in utero or immediately after birth, for stress and depression, with a preliminary indication that transduction of antenatal maternal PTSD may also occur. These findings are both good news and bad news. The good news is that identification of at-risk prenates through assessments of maternal emotional traits and biochemistry creates the possibility of intervention for those prenates who are at high risk for becoming young people with a dual diagnosis of mental illness and addiction. Practitioners of pre- and perinatal psychology such as Ray Castellino and Wendy McCarty, as well as forthcoming graduates of the pre- and perinatal program at Santa Barbara Graduate Institute are currently expanding practices developed to work with peri- and neonatal difficulties to include work with prenates and their parents. The more widespread these practices become, the more hope there is of reducing the number of individuals who suffer from the twin difficulties of mental illness and addiction by creating a firm foundation to withstand later insults and traumas. The bad news is that these ideas are only beginning to spread beyond a relatively small group of practitioners into mainstream practice, with the result that many, if not most, of the prenates of depressed, chronically stressed, or PTSD-affected mothers, will not benefit from early intervention and will still remain at risk for responding to additional trauma, in the broader sense, with mental illness and addiction. References REFERENCES Aerts, L. &Van Assche, F.A. (2003). Intra-uterine transmission of disease. Placenta, 24(10), 905-911. Albrecht, E. &Pepe, G. (1990). Placental steroid hormone biosynthesis in primate pregnancy. Endocr Rev, 11(1), 124-150. Berkowitz, G.S., Lapinski, R.H., Lockwood, C.J., Florio, P., Blackmore-Prince, C., &Petraglia, F. (1996). Corticotropin-releasing factor and its binding protein: maternal serum levels in term and preterm deliveries. Am J Obstet Gynecol, 174(5), 1477-1483. 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