POSTPARTUM DEPRESSION IN THE ABSENCE OF LACTATION:

AN EVOLUTIONARY PERSPECTIVE

A Thesis

by

KELLY J. PEYTON

Submitted to the Office of Graduate Studies of Texas A&M University in partial fulfillment of the requirements for the degree of

MASTER OF ARTS

May 1996

Major Subject: Anthropology
POSTPARTUM DEPRESSION IN THE ABSENCE OF LACTATION:

AN EVOLUTIONARY PERSPECTIVE

A Thesis

by

KELLY J. PEYTON

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

MASTER OF ARTS

Approved as to style and content by:

Katherine A. Dettwyler
(Chair of Committee)

Lee Cronk
(Member)

Jeffry Simpson
(Member)

Vaughn M. Bryant, Jr.
(Head of Department)

May 1996

Major Subject: Anthropology
ABSTRACT

Postpartum Depression in the Absence of Lactation:
An Evolutionary Perspective. (May 1996)

Kelly J. Peyton, B.A., Pennsylvania State University
Chair of Advisory Committee: Dr. Katherine A. Dettwyler

It is currently estimated that up to one third of new mothers in
industrialized nations suffer from moderate postpartum depression in the
weeks and months following childbirth. However, cross-cultural data reveal
that this condition is not nearly so prevalent outside of the West. While the
traditional structuring of the postpartum period may contribute to maternal
well-being, an important covarying factor is feeding practice. Diverse
breastfeeding patterns can create meaningful differences between the
hormonal profiles of women in Western and non-Western countries.
Specifically, the activity of oxytocin, a neurohormone released during
parturition and lactation, is reduced in the postpartum period in mothers who
do not breastfeed. Because recent studies demonstrate that oxytocin is a
potent antidepressant, decreases in oxytocin activity may significantly
contribute to an increase in postpartum depression in the absence of
lactation. This thesis explores the ultimate and proximate functions of
oxytocin in mediating maternal affect in the postpartum period. Several studies linking breastfeeding to positive affective tone are reviewed. It is concluded that, by redefining postpartum depression to fit ecologically valid diagnostic criteria, future studies will demonstrate that women who do not breastfeed are more likely to develop postpartum depression.
for Lindsay and Ellen

and all of the women I love

who may someday be mothers
ACKNOWLEDGEMENTS

I would like to take this opportunity to thank the members of my committee, Kathy Dettwyler, Lee Cronk, and Jeffry Simpson, for their input, encouragement, and time. In addition, I would like to thank Karen Taylor for helping me at so many crucial points. I would also like to thank the people I interviewed in Lestesti, Romania for sharing their lives with me and for having the patience to answer all of my questions. Lastly, I owe many, many thanks to my good friends and family for their constant support and occasional pushes forward.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>CHAPTER</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>INTRODUCTION .................................................</td>
</tr>
<tr>
<td>II</td>
<td>THE PSYCHOBIOLOGY OF MAMMALIAN MOTHERHOOD ...</td>
</tr>
<tr>
<td></td>
<td>Ultimate Function--Generating Predictions ..........</td>
</tr>
<tr>
<td></td>
<td>Proximate Mechanisms--Testing Predictions ..........</td>
</tr>
<tr>
<td>III</td>
<td>THE ROLE OF OXYTOCIN IN MATERNAL BEHAVIOR AND AFFECT</td>
</tr>
<tr>
<td></td>
<td>Oxytocin--Background ................................</td>
</tr>
<tr>
<td></td>
<td>Oxytocin in Maternal Behavior ......................</td>
</tr>
<tr>
<td></td>
<td>Oxytocin in Maternal Affect .........................</td>
</tr>
<tr>
<td></td>
<td>The Adaptive Value of Maternal Depression ..........</td>
</tr>
<tr>
<td>IV</td>
<td>IMPLICATIONS FOR POSTPARTUM DEPRESSION ..........</td>
</tr>
<tr>
<td></td>
<td>Definitions and Distinctions .......................</td>
</tr>
<tr>
<td></td>
<td>A Biocultural Approach ................................</td>
</tr>
<tr>
<td></td>
<td>Absence of Lactation and Postpartum Depression ....</td>
</tr>
<tr>
<td>V</td>
<td>CONCLUSION ..................................................</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>.........................................................</td>
</tr>
<tr>
<td>APPENDIX: ROMANIAN POSTPARTUM RITUALS..........</td>
<td>86</td>
</tr>
<tr>
<td>VITA</td>
<td>...............................................................</td>
</tr>
</tbody>
</table>
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>FIGURE</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>The antidepressant effects of oxytocin</td>
<td>36</td>
</tr>
<tr>
<td>2</td>
<td>Stimulated oxytocin in partial and exclusive breastfeeders</td>
<td>53</td>
</tr>
<tr>
<td>3</td>
<td>A biocultural model of postpartum depression</td>
<td>55</td>
</tr>
<tr>
<td>4</td>
<td>Predicted levels of postpartum depression</td>
<td>57</td>
</tr>
<tr>
<td>5</td>
<td>Proposed neurohormonal role of oxytocin in maternal affect</td>
<td>73</td>
</tr>
</tbody>
</table>
# LIST OF TABLES

<table>
<thead>
<tr>
<th>TABLE</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>The Three Levels of Postpartum Depression</td>
<td>44</td>
</tr>
<tr>
<td>2</td>
<td>Previous Questionnaires Used to Diagnose PPD</td>
<td>68</td>
</tr>
<tr>
<td>3</td>
<td>Traditional Structuring of the Postpartum Period</td>
<td>91</td>
</tr>
</tbody>
</table>
CHAPTER I
INTRODUCTION

The growing interdisciplinary field of evolutionary medicine seeks to explain physical ailments as being at least partly derived from a significant mismatch between our ancestral and modern environments (Nesse & Williams, 1994). Applying this evolutionary perspective to the study of behavior and cognition has led to the emergence of evolutionary psychology (Barkow, Cosmides, & Tooby, 1992)—a subfield of psychology that views the human mind as a product of evolutionary processes and searches for explanations for modern psychological disorders in the same disparity between past and present environments. Behaviors and emotions that have served successfully reproducing hominids well for millions of years can die hard, leaving modern humans torn between what is now sometimes expected and what was in the past consistently demanded (Cronk, 1992). Examining how relatively novel behavioral demands may impact mental health is particularly appropriate in attempting to uncover the etiology of a psychological disorder that correlates with modernity either temporally or

This thesis follows the format used by Psychological Bulletin.
geographically. The contemporary and Western psychological disorder known as postpartum depression (PPD) has both of these qualities.

It is currently estimated that 10 to 30% of new mothers in industrialized nations suffer from moderate postpartum depression (Boyle, 1993; Weissman & Olfson, 1995), contrasting with very low rates outside of Western countries (Stern & Kruckman, 1983). These cross-cultural data have been used to support the view that only social factors account for differences in the prevalence of postpartum depression. Specifically, Stern and Kruckman (1983) conclude that the structure of the postpartum period in traditional societies offers the kind of social support that is conducive to the psychological well-being of a new mother. In addition, they downplay the contribution of biological factors in the development of PPD because of the assumption that relevant biological events are experienced uniformly around the world.

Stern and Kruckman's dismissal of psychobiological explanations neglects how differences in behavior can be traced to differences in physiology. Two important behaviors that covary with a traditionally structured postpartum period and have a marked effect on the hormonal status of a new mother are breastfeeding and levels of environmental stress. The trend for more breastfeeding and lower amounts of specific stressors in the traditional treatment of mothers in the postpartum period may create
significant differences between the modal hormonal profiles of Western and non-Western mothers. These hormonal differences, in turn, may affect the emotional experience of early motherhood. This thesis will explore how diverse patterns of breastfeeding and levels of stress can specifically alter the activity of oxytocin, a neurohormone synthesized in the hypothalamus and secreted from the posterior pituitary, and how these physiological differences may alter the risk of developing PPD.

The major objective of this thesis is to examine the evidence for mechanisms linking PPD to the absence of lactation both by predictions based on ultimate considerations and by a testable proximate mechanism. In Chapter II, predictions about maternal behavior in humans are derived from a more general discussion of the psychobiology of mammalian motherhood. This discussion of the archetypical mammalian mother depicts a suite of "normal" maternal behaviors, influenced by the hormones of lactation, from which some human cultures have diverged more than others.

In Chapter III, a proximate mechanism by which absence of lactation may be associated with depression is proposed. Again, the physiological link between depression and little or no breastfeeding is the reduced activity of oxytocin. Oxytocin levels increase during breastfeeding and are associated with the milk-ejection reflex (Newton, 1978). Recent research has also shown that oxytocin is capable of preventing or reversing symptoms of
depression (Arletti & Bertolini, 1987; Bakharev, Tikhomirov, & Lozhkina, 1986). By not breastfeeding, modern women may forfeit the antidepressant effects of oxytocin and so introduce a relatively new and Western risk factor for developing PPD.

Chapter III also includes a discussion of the adaptive value of maternal emotions and, in particular, the possible adaptive value of depression in the postpartum period. I suggest that the absence of lactation mimics the physiological conditions under which depression can be adaptive and, thus, may elicit this response when it is clearly not adaptive.

In Chapter IV, I provide a biocultural model for the etiology of postpartum depression and offer ecologically valid amendments to the criteria by which PPD is currently diagnosed. This holistic model incorporates relevant social factors, biological factors, and the numerous and meaningful interactions between them. These interactions are valuable beyond their ability to shatter the false dichotomy of social or biological causal factors, for they also have implications for preventative measures to take in optimizing maternal well-being during the postpartum period.

Chapter IV concludes by making recommendations for redefining "normal" behavior and affect in the postpartum period. Supported by the initial discussion of the psychobiology of mammalian motherhood and the portrayal of natural maternal behaviors and emotions, I critically examine
the criteria by which a diagnosis of postpartum depression is currently made. Specifically, in an attempt to redefine normal adaptation following childbirth, I scrutinize the assumption, common in the psychiatric literature, that returning to a pre-pregnancy state represents postpartum normality. In parallel, I question the validity of a diagnosis based on symptoms of "hostility" and "social disruption" when maternal aggression and seclusion are both predicted by evolutionary theory. I suggest that by creating a cluster of ecologically valid symptoms by which "abnormal" maternal functioning and mood can be identified, future research will find a link between postpartum depression and the absence of lactation.

The appendix contains a summary of ethnographic research derived from interviews conducted with townspeople in Leresti, Romania in the summer of 1994. The information gathered on the traditional treatment of women in the immediate postpartum period highlights the difficulty in dissecting social from biological factors, as interviewees consistently reported a strict social code of behavior along with an explicit belief that breastfeeding is extremely important for the health of an infant and an implicit assumption that a new mother will breastfeed.

By approaching the psychiatric literature from a decidedly anthropological perspective (both in the evolutionary and cross-cultural sense), I hope to create a more complete portrayal of the etiology of
postpartum depression than either a strictly social or biological interpretation can offer. However, it is beyond the scope of this thesis to delineate and explore all of the factors and interactions that contribute to the development of depression in the postpartum period.

This thesis was conceived from a particular perspective and under certain ideological assumptions. My perspective as a student of psychobiology and physical anthropology leads me to explore both proximate and ultimate functions of behavior. I do assume causal links between both brain and behavior, and physiology and mood, that some would find unwarranted by correlational data. In addition, I assume that certain levels of homology exist between humans and other mammals and that animal models can be used to describe human conditions. I also assume that humans have evolved and that differential reproductive success has left our species with certain behavioral tendencies that have been adaptive for millions of years.

Removed from the environment that has slowly shaped our behavioral tendencies, humans can and sometimes do devise cultural norms that contradict our natural heritage. Perhaps no behavioral deviation has more intricate consequences than the relatively modern, Western trend to substitute the bottle for the breast and artificial formula for breast milk. While the nutritional and medical advantages of breastfeeding are widely accepted, the psychological benefits gained by either mother or child are not well
understood. This thesis explores the possibility that not lactating may pose a risk factor for the development of postpartum depression in Western women.
CHAPTER II

THE PSYCHOBIOLOGY OF MAMMALIAN MOTHERHOOD

Mammalian mothers have a long and costly relationship with their offspring. Evolutionary theory predicts that this investment will not be made indiscriminately. Rather, it predicts that maternal investment will follow patterns based on life-history variables and ecological constraints or opportunities that maximize a mother's reproductive success. The maternal behaviors of modern mammals and the motivation for these behaviors, having survived one hundred million years in mammalian natural history, are predicted to reflect these ultimate considerations.

If ultimate functions are used to predict patterns of behavior, proximate mechanisms can be the corollary physiological evidence by which evolutionary theories are either refuted or supported. The complementary relationship between ultimate and proximate functions can also flow in the opposite direction, in which case prolific amounts of just-so evolutionary scenarios can be created to account for physiological mechanisms. However, Popperian falsification (or the support of a theory derived from failing to falsify it) is only possible when observable phenomena come after pre-stated predictions (Popper, 1959).

With this in mind, and in an attempt to bring together systematically
the literatures of evolutionary biology and psychobiology, mechanisms of specific maternal behaviors will be explored after a discussion of general evolutionary predictions about the mammalian mother.

**Ultimate Function--Generating Predictions**

Mammals are mothered. In contrast to varied patterns of relatively high paternal investment in many fishes and shared parental investment in many birds, mammals generally show a pattern of primary maternal investment (Krebs & Davies, 1987). To some extent, this follows from the physiological ability of the female to lactate from her mammae, the organs after which this class of animals is named. In addition, decreased paternity certainty due to internal fertilization and the greater opportunity for males to desert (Trivers, 1972) leads to the prediction that most mammalian fathers will invest little more than is necessary to reproduce. For these reasons, the mammalian mother is usually the primary care-taker, and offspring survival is dependent on her investment.

**Mother-offspring conflict.** Mammalian mothers and offspring are related by one half of their genetic material. Thus, in a cost-benefit analysis, what is good for the offspring is not always best for the mother. This is the

Over-investment in any one offspring can limit investments in other existing
or future offspring and can reduce a female's life-long reproductive success.
Evolutionary theory predicts that the mammalian mother should only invest
under specific conditions that optimize her own cumulative genetic
self-interest.

However, successful mammalian offspring have managed to elicit a
sufficient amount of this precious investment. Therefore, on the other side of
this mother-offspring relationship, evolutionary theory also predicts that the
offspring--only related to its siblings by one half or one fourth (when siblings
have different fathers)--should try to deceive its mother into over-investment
under many conditions. "Altruistic" behaviors are only predicted to occur
when the benefit to another full sibling is more than twice the cost to the
acting one.

This sibling rivalry should result in even more discriminating maternal
investment. It is predicted that the successful mother will counteract
deceptive eliciting stimuli by basing decisions to invest on more subtle cues.
So evolves the never-ending arms race of maternal discrimination and
offspring cues (Krebs & Dawkins, 1984). While this conflict between mother
and offspring interests begins during pregnancy (e.g., Haig, 1993; Peacock,
1991), parturition changes the nature of these signals from directly
biochemical to behavioral. The focus here is on maternal behavior in the postnatal period.

As applies to maternal behavior, the spiral of deception and discrimination predicts that investment should only be made in response to specific and complex physiological and environmental conditions. Postnatal maternal investment in mammals is particularly dear because of the great energetic costs of lactation (Leon, 1987) and the relatively long-term investment made by many of the more k-selected species, including primates (McKenna, 1981; Nicolson, 1987). In addition, different amounts of investment should be made in response to different maternal, environmental, and offspring conditions.

**Maternal conditions.** As was previously mentioned, reproductive success accumulates over a female's lifetime. Because maternal age and parity are significant life history variables, they are also likely predictors of maternal investment. Specifically, young and/or low parity mothers have more to lose than older and/or high parity mothers by investing too heavily in current offspring because their potential to invest in future offspring is greater. Other factors held equal, one would generally expect less investment from younger, low parity mothers and greater investment from older, high parity mothers.
Evidence for this in mammals is multi-fold. Nicolson (1987) finds that older olive baboon mothers spend more time in contact or close proximity to their infants and are less likely to interrupt nursing than younger mothers. Capitanio, Weissberg, and Reite, in their review of maternal behavior, note "conspicuous differences in the maternal behavior of primiparous versus multiparous animals" (1985:81). Specifically, multiparous rhesus monkeys are relatively less restrictive and anxious and even show maternal care of unfamiliar infants. While this latter phenomenon does not seem adaptive, it does reflect the decreasing discrimination of older mothers in making investments.

In addition, the maternal behavior of multiparous mothers seems to fluctuate less in response to biochemical manipulation. In an experiment using cage-reared rats, morphine injections (which usually disrupt maternal behavior in rats) are less effective in disturbing maternal behavior in multiparous mothers than in age-matched primiparous mothers (Kinsley & Bridges, 1988).

Maternal age differences in human infanticide may also reflect an increasing tendency to invest towards the end of a female's reproductive career. Daly and Wilson (1984) find that, in Canadian women, infanticidal mothers are significantly younger than most new mothers. If infanticide is viewed as the absolute refusal to perform postnatal maternal behaviors, older
mothers are more likely to invest because they are less likely to commit infanticide.

Thus, the effects of parity and maternal age seem to introduce significant variables in the differential display of maternal behaviors. The tendency for older, higher parity females to be calmer mothers and less discriminating investors most likely results from both biological changes associated with childbirth (a biological preparedness to learn) and greater experience with the stresses of motherhood (the stimuli for learning). The role of hormonal priming in the onset of maternal behaviors is discussed in the next major section of this chapter. For now, it is only important to note the observed association between age, parity, and maternal investment.

**Environmental conditions.** Cost-benefit analyses lead to the prediction that limited resources should have an effect on maternal behavior. Specifically, a reduction in energetic inputs for the mother should correlate with her reluctance to invest or continue to invest in a poor bet. Limited access to food, for example, should make a mother more selective in her investments. Changes in postnatal maternal behavior are one means of accomplishing this adaptive discrimination.

Leon (1987) hypothesizes that rates of !Kung infanticide, particularly high when one child is born too soon after another, reflect a combination of
limited nutrition and the high energetic cost that carrying a second infant would impose. Under conditions of nutritional stress, breastfeeding the newborn would mean risking the life the older child, in whom the mother has already invested several years of lactation and care. Rather than invest poorly in both, the mother forfeits the newborn.

Another strategy for dealing with nutritional stress is to treat female offspring preferentially (Trivers & Willard, 1973). The Trivers-Willard model is based on the observation that males of a polygynous species have a much wider range of reproductive success than females. While a son receiving low investment may not reproduce at all, a daughter is likely to have roughly the same number of offspring even under some amount of nutritional stress. Therefore, when resources are limited, the surer bet is on a daughter. Only when resources are plentiful is it wise to fully invest in a son. Cronk (1991) reviews evidence for this effect found in eight human societies, including contemporary North America.

*When, how much, and for whom.* Assuming that maternal and environmental conditions are conducive to maternal investment, these energetically expensive behaviors should only be performed at the proper time, within certain limits, and for specific offspring.

One probable waste of energy would be to begin post-natal maternal
behaviors before giving birth. While alloparenting can have benefits in the form of kin selection, improved social relations, and the chance to practice maternal behaviors without risking one's own reproductive success (e.g., McKenna, 1987), the full suite of maternal behaviors (including lactation) would almost certainly be lost on an infant that is not one's own. Therefore, it would be adaptive for some kind of physiological "switch" that initiates maternal behaviors to be turned on when a female first gives birth, to prevent premature over-investment. In addition, it should not be surprising if this behavioral change depends on physiological feedback loops inextricably linked to the natural succession of gestation, parturition, and lactation in mammals. Absence of lactation, for example, may provide a signal to the body that the infant has died; therefore, maternal investment should be curtailed. This is discussed at greater length in the next chapter.

Another waste of energy would be to invest too much in one offspring. A mother should be able to determine when it is best for her to wean her offspring and to resist ploys made by her offspring to continue to nurse beyond that point. Nicolson, in a review of primate mother-offspring relationships notes that "...for all wild primate species in which the transition from nutritional dependence to independence has been described, conflict is evident" (1987:330-331). This weaning conflict results from the infant's desire to nurse longer than is optimal for the mother. In addition, the rate of
maternal rejection should depend on individual offspring development.

Trivers (1985) finds that the positive correlation between infant activity and maternal rejection in captive rhesus monkeys is an adaptive response to "hurry along" those who no longer need as much from the mother.

Mechanisms that control kin recognition (specifically the identification of one's own offspring) and promote feelings of attachment to this large investment are also adaptive (e.g., Petrovich & Gewirtz, 1985). The prediction that a mother should protect her sizeable investment is based on similar evolutionary concerns. In the case of an immediate physical emergency for either the mother or infant, one would expect the shut-down of other adaptive behaviors and the instantaneous change to offensive or defensive "fight or flight" behaviors.

In sum, evolutionary theory makes specific predictions about the mammalian mother. She should not act maternally until her first offspring is born. She should only mother in response to specific cues. She should form an attachment to her own offspring. Lastly, she should protect her investment fiercely.
Proximate Mechanisms--Testing Predictions

Evolutionary theory predicts that under optimal maternal and environmental conditions mammalian mothers should react to specific and perhaps complex stimuli after a parturitional switch in behavioral tendencies. The natural sequence of gestation, parturition, and lactation would normally be synchronized with the onset and maintenance of these behaviors.

The parturitional switch. In most mammals, some maternal behaviors begin before offspring are born (e.g., nest-building). However, the first external mother-to-infant interactions are typically immediate positive responses to infant stimuli (e.g., anogenital licking of the newly born). It is likely that pregnancy and parturition provide the hormonal priming for these behaviors.

Rosenblatt (1989) finds that the vigorous uterine contractions that occur three or four hours before parturition are closely related to the onset of positive response to nest odors and attraction to pup calls in rats. He also suggests that the vaginal-cervical stimulation that occurs during parturition may reinforce these preferences because nonpregnant, estrogen-treated female rats given such stimulation show a rapid response to pups that is not shown in similarly estrogen-treated females without this stimulation. In
addition, the fostering of alien lambs can be invoked in normally rejecting mother ewes when vaginal-cervical stimulation is applied for several minutes (Keverne, Levy, Poindron, & Lindsay, 1983). Thus, the physical act of parturition itself seems to be crucial for some maternal behaviors.

One method of studying changes in maternal responsiveness is to observe the amount of time necessary for a pup to invoke nurturing behaviors in the tested female. This amount of time is termed "latency." After puberty, virgin female rats not treated with hormones show a latency of five to seven days. This latency declines precipitously during the last few days of the first pregnancy to the parturitional latency of only one hour. Latency remains extremely low until the end of lactation. After this, it only increases to half a day throughout the rest of a parous female rat's life (Bridges, 1990). Therefore, there seems to be a parturitional behavioral switch with respect to latency.

What is the mechanism involved? Studies performed by Terkel and Rosenblatt (1968, 1972) suggest a blood-borne parturitional factor. In their study, virgin rats received blood transfusions from one of four groups: other virgins, parturitional females, and females either 24 hours before or after parturition. Latency was only reduced in the virgins transfused with parturitional blood. This indicates that the change that takes place on the day when a female rat first gives birth creates a permanent neurological
change and that the subsequent reduction in latency (throughout her life) is
not induced by continuous hormonal activity, but by the developmental switch
at parturition. Otherwise, transfusions from post-parturitional females would
also reduce latency.

However, other studies indicate that particular stimuli during this
hormonally distinct moment are necessary to maintain specific maternal
responses. These include proximity to the newborn (Fleming, Corter, Franks,
Surbey, Schneider, & Steiner, 1993) and suckling (Gandelman, 1980). This
is not incompatible with the idea that parturition prepares new mothers to
respond to infant stimuli. Rather, it implies an interaction between biological
preparedness and critical environmental cues.

The idea that responses to certain stimuli can only develop during
certain sensitive periods is familiar to ethologists and psychologists alike.
However, while the psychological development of pre-reproductive
individuals is discussed extensively in the literature (especially imprinting in
newborns), the psychological development of adults is rarely mentioned.
Klaus & Kennell (1983) note that their proposal that there is a sensitive
period for bonding in the human mother is reluctantly received. However,
studies of maternal behavior in other mammals suggest that adult
development can and does occur.

The recognition that the immediate postpartum period may be a
sensitive time in the development of maternal behaviors in the human female
(e.g., Kennell, Trause, & Klaus, 1975) has transformed some hospital policies
regarding maternal access to premature neonates. For example, Eckes
(1974) describes an "open door" policy in which mothers of premature or
high-risk term babies are permitted to freely enter the neonatal intensive care
unit to care for their babies. The wider acceptance of the phenomenon of a
sensitive period for maternal bonding would have implications for birthing
practices in general. Specifically, mother-infant separation would be
consistently eliminated or minimized and early skin-to-skin contact would be
encouraged.

What are the critical stimuli for turning on the parturitional switch?
Studies from human and nonhuman mammals suggest the importance of
mother-infant proximity, olfactory or pheromonal communications, skin-to-
skin contact, and suckling.

Mere proximity to an infant during the postnatal period has been
correlated with the establishment of maternal behaviors. Rosenblatt (1989)
reviews the literature on rats, sheep, and goats demonstrating that the
separation of mother from infant just after birth results in a rapid decline in
maternal responsiveness. In these species, olfactory stimuli from the
newborn are suspected to play an important role in the establishment of
appropriate maternal behaviors. Kinsley and Bridges (1990) suggest that
endogenous opiates released during lactation may regulate these olfactory preferences.

In humans, while new mothers prefer infant smells more than nonmothers, mothers who rate infant odorants the most positively are those who experience a shorter period of separation from their infants after birth (Fleming et al., 1993). Studies also suggest that increased skin-to-skin contact between human mothers and babies just a few hours after birth can have long-term effects (Klaus & Kennell, 1976). Mothers who are given increased contact during this time show greater soothing behavior and less physical distance in a routine physical exam one month later, and greater soothing, cuddling, and nurturing behaviors in an interview one year later! Thus, early contact seems critical in the formation of maternal behaviors.

The critical stimulus of suckling has been observed in the development of maternal aggression in mice and maternal responsiveness in humans. Experiments show that this stimulus is only effective in establishing aggression in mice if received during the first two to four days after parturition (Gandelman, 1980). Alternatively, exogenous hormone treatment mimicking pregnancy (estrogen and progesterone) can prime virgin mice for the development of maternal aggression (Gandelman, 1980), an adaptive behavior for the mammalian mother.

In humans, suckling within 30 minutes after birth positively affects the
early mother-infant relationship. With skin-to-skin contact held constant for mother-infant pairs, mothers who received early areola and nipple stimulation had more contact with their babies when not breastfeeding and talked to them more during breastfeeding four days after birth (Widstrom, Wahlberg, Matthiesen, Eeroth, Uvnas-Moberg, Werner, & Winberg, 1990). This suggests that while early skin-to-skin contact may aid in mother-infant bonding, infant suckling, in particular, is a powerful stimulus in this process.

This discussion of the parturitional switch supports evolutionary predictions in two ways. First, the initiation of this mechanism prevents the expression of full maternal behaviors before one has offspring. Second, maternal behaviors are only fully developed when infant stimuli immediately follow--indicating a live and healthy child.

**Maternal aggression.** One of the major predictions from evolutionary theory is that a mammal's offspring should be fiercely protected. One would expect that altricial young should be kept safely with the mother whenever possible and that suspicious intruders should be driven out effectively. Maternal aggression has been observed in many mammalian species including mice, rats, cats, squirrels, baboons, rabbits, moose cows and sheep (Svare, 1981). Personally, I have observed both of our family dogs uncharacteristically bite unfamiliar humans who came too close to the
whelping box. I have also seen our most docile horse charge at a dog that came too close to her baby.

Maternal aggression is different from intermale aggression. In mice, ritualized intermale aggression can usually be stopped by certain submissive gestures; in contrast, maternal aggression is more intense and shorter-fused, and cannot be appeased by submissive gestures. In addition, the object of maternal attacks can be a male or a female of the same species or of a different species, as long as the animal is unfamiliar to the mother (Gandelman, 1980).

In mice, mothers do not attack infants who are not their own until they grow a coat of fur. The adult-like appearance of a fur coat makes them fair game for an attack. Likewise, many primate infants have certain coat or skin colorations that differ strikingly from adult patterns (Higley & Suomi, 1986). This "infant coloration" may signal to adults (including other mothers) to make special allowances for them. Lorenz remarks that infant stimuli are so effective in arresting aggression that many submissive adult gestures are based on infantile behavior patterns (1963).

As was previously discussed, a strong relationship exists between lactation and maternal aggression (Svare, 1981). In mice, this aggression falls strictly into the period of time between birth and weaning. Gandelman (1980) and Svare (1990) both stress that the necessary components to
induce maternal aggression are the hormonal priming of pregnancy and the stimulation of the nipple (leading to lactation). Oxytocin, a neurohormone released during lactation, may be the chemical messenger by which this behavior is evoked (in at least some species), as injections of it facilitate maternal aggression in hamsters (Ferris, Foote, Meltser, Plenby, Smith, & Insel, 1992). Again, in the normal and natural sequence of gestation, parturition, and lactation, maternal aggression does develop, and it is an adaptive reaction to unfamiliar intruders.

**Conclusions about the mammalian mother.** This chapter makes it clear that certain changes in a female's behavior at the time of birth and during lactation are not only normal, but adaptive. Among these normal changes is a parturitional switch in responsiveness that is strengthened by healthy infant signals. Specific behaviors that normally develop in new mammalian mothers include increased caution and protectiveness (expressed as aggression towards intruders) and bonding to the offspring. Both of these behaviors appear to be at least partly dependent on the stimulus of suckling—a cue that has indicated infant health throughout the natural history of mammals and, therefore, has elicited the precious investment of the mammalian mother.
CHAPTER III
THE ROLE OF OXYTOCIN IN MATERNAL BEHAVIOR AND AFFECT

In the preceding chapter, I refer to a "hormonal preparation" for the establishment of maternal behaviors and a suspected "blood-borne factor." In the discussion of the acute switch to maternal responsiveness, this factor is associated with the vaginal-cervical stimulation of parturition. A reduction in latency is observed until the end of lactation. In addition, maternal aggression is strongly linked to suckling and lactation. Oxytocin is associated with both the contractions of childbirth and the milk-ejection reflex of lactation. It is, therefore, a likely hormonal influence on the onset of maternal behaviors and, as this chapter will demonstrate, on maternal emotions.

Oxytocin--Background

The hormones of childbirth. Because of the pronounced changes in behavior that accompany childbirth, hormones whose levels change dramatically at this time are suspected to play an influential role. Of particular interest are the ovarian hormone, estrogen, and the pituitary
hormones, prolactin and oxytocin (Rosenblatt, 1989).

In a review article on the biology of maternal behavior, Capitanio and colleagues stress the importance of oxytocin (Capitanio, Weissberg, & Reite, 1985). Estrogen also seems to be very important in the onset of maternal behaviors, as injections of estradiol cause shortened latency. However, the pathway by which estrogen has this effect may very well be through its ability to increase oxytocin synthesis and, more importantly, to cause a proliferation of oxytocin receptors (Insel & Shapiro, 1992).

The up-regulation of oxytocin receptors alone is significant. Insel and Shapiro remark that "an increase in oxytocin function can result from an induction of receptors even in the absence of a detectable increase in oxytocin release" (1992:124). The elevation of estrogen at the end of pregnancy causes a proliferation of oxytocin receptors in the brain, which can be similarly induced by the administration of exogenous estrogen (Insel, 1986). It is likely that the stimulatory effect of estrogen on maternal responsiveness depends on its ability to up-regulate oxytocin receptors.

Prolactin has also been implicated in the onset of maternal behaviors. However, the effects of oxytocin can be mistaken for those of prolactin because the two hormones are released in synchronous pulses during lactation. Prolactin is associated with milk production and oxytocin with the milk-ejection reflex. The intimate relationship between the two hormones
stems from the ability of the pulsatile release of oxytocin (during suckling) to stimulate prolactin production and release (Yokoyama, Ueda, Irahara, & Aono, 1994).

The confusion caused by the temporal association of these two hormones can be resolved by blocking prolactin or oxytocin separately. In animal studies using pregnant rats, blocking prolactin has no influence on the onset of maternal behaviors (Numan, Rosenblatt, & Komisaruk, 1977). Because of this, Capitanio and colleagues conclude that "prolactin is only minimally, if at all, involved in this process" (1985:54). However, blocking oxytocin activity by either lesioning areas of the brain where oxytocin is synthesized (Insel & Harbaugh, 1989) or administering oxytocin antagonists (Pedersen, Caldwell, Johnson, Fort, & Prange, 1985; van Leengoed, Kerker, & Swanson, 1987) does inhibit the initiation of maternal behaviors. Indeed, current research clearly points to the primary role of oxytocin in mediating maternal behaviors (e.g., Angier, 1991; McCarthy, Kow, & Pfaff, 1992).

The importance of oxytocin for mammals. Oxytocin is the quintessential mammalian molecule. Its release leads to a harmony of physical functions and complex behaviors that is necessary for the coordination of such events as live birth, lactation, and the nurturing of altricial young. As mammals evolved, so did oxytocin (Sherwood & Parker,
The antecedents of this neuropeptide (present in nonmammalian animals) are associated with increases in sexual behaviors (Moore, 1992). In mammals, however, the behavioral corollaries become more generally affiliative (Insel, 1992), forming a spectrum of prosocial functions that spans the range from mating to care-taking.

The coordination of the physical and behavioral events necessary for successful mammalian reproduction is achieved by the dual function of oxytocin. Oxytocin has two systems of release, named after the parts of the body into which it is released: peripheral and central. Peripheral release (that is, release into the periphery) is associated with orgasm, the uterine contractions of labor, and the milk-ejection reflex. The traditional view of the effects of oxytocin assumes that only receptors in the mammary myoepithelium and uterus respond to the systemic release of this hormone from the posterior pituitary. However, recent anatomical studies have revealed an extensive network of oxytocin fibers distributed throughout the mammalian brain and spinal cord, leading from oxytocin-synthesizing nuclei to both the posterior pituitary and to other nerve bundles (Sofroniew, 1983). In its central release (into the central nervous system), oxytocin functions as a neurotransmitter rather than a hormone.

The realization that the brain is a target organ of oxytocin has led to an expanded view of the effects of this neuropeptide. Its central function
consists of a pantheon of social behaviors including maternal behavior, sexual behavior, yawning, memory and learning, tolerance and dependence mechanisms, feeding, and grooming (Argiolas & Gessa, 1991). Insel and Shapiro hypothesize that the parallel processing of oxytocin's peripheral and central pathways in the mammalian mother, allowing for the crucial coordination of lactation and care-giving, is achieved by a neural "bridge" (1992:130), leading from a center of maternal behavior (the bed nucleus of the stria terminalis) to a center of peripheral release (the supraoptic nucleus). This may explain the synchrony of oxytocin's central and peripheral release in the mammalian mother.

**Oxytocin in Maternal Behavior**

Oxytocin is involved in a host of maternal behaviors ranging from nest-building and infant-retrieval (Pederson & Prange, 1979), to pup licking and grouping (Insel & Shapiro, 1992), to the olfactory imprinting involved in offspring recognition (Gribben, 1988). Injections of oxytocin inhibit infanticide, a normal behavior in virgin mice and rats (McCarthy et al., 1992), and induce the full complex of maternal responses when given to estrogen-primed female mice (Insel & Shapiro, 1992). In addition, it has been shown that oxytocin treatment facilitates the adaptive and normal establishment of
maternal aggression in hamsters (Ferris et al., 1992).

Crucial support for the involvement of oxytocin in maternal behavior is that oxytocin antagonism blocks the initiation (but not the maintenance) of maternal behaviors. While blocking oxytocin function by destroying oxytocin synthesizing cells just before birth results in a significant reduction in maternal behavior, the same destruction does not disrupt maternal behavior when administered on day four postpartum (Insel & Harbaugh, 1989). Presumably, it is the effect of early surges of oxytocin, during both parturition and the first days of lactation, that is necessary for the initiation of maternal behaviors.

There is additional support for the assumption that the critical stimulus for the initiation of maternal behavior is not only the oxytocin surge of parturition, but also the pulses released early during lactation. The oxytocin antagonist, d(CH2)5-8-ornithine-vasotocin, administered into the cerebral ventricles of primiparous rats half an hour after the birth of their first pup does delay the onset of maternal behaviors (van Leengoed et al., 1987). Therefore, the surge at parturition itself is not enough to initiate a permanent reduction in latency. However, after the antagonist wears off and the mothers suckle their pups overnight, normal maternal behavior begins.

It is interesting that perinatal surges in oxytocin are associated with permanent changes in female behavior. This provides a physiological
mechanism for the "parturitional switch" discussed earlier.

**The inhibition of oxytocin by stress.** A striking characteristic of the activity of oxytocin is that it can be inhibited by stress. This has been well documented in the inhibition of the milk-ejection reflex (Newton, 1978). While the let-down of milk can occur in response to a conditioned stimulus that the mother associates with her baby, it can also be inhibited by embarrassment or distraction (Newton & Newton, 1967). The mild stressor of novelty can specifically reduce peripheral levels of oxytocin. At the time of birth, introducing a female rat to a novel cage can inhibit oxytocin levels so much that suboptimal uterine contractions prolong parturition (McCarthy et al., 1992).

Chronic stress (especially continuous exposure to novel environments) can lead to consistently low levels of oxytocin. This can make the initiation or maintenance of lactation very difficult and prevent the establishment of lactational amenorrhea (McNeilly, Tay, & Glasier, 1994). McNeilly and colleagues note that "oxytocin is essential for milk let-down and is susceptible to inhibition of release by stress. The successful initiation of lactation which would lead to the potential of utilizing breastfeeding as contraceptive may require more attention to be paid to the establishment of the non-stress release of oxytocin" (1994:152).
The ability of environmental stress and emotional upset to inhibit oxytocin makes evolutionary sense. The activities that are arrested because of oxytocin inhibition are ones which introduce temporary physical vulnerability: sexual intercourse, labor, and nursing (Newton, 1978). If a threatening environment more appropriately calls for fight or flight behaviors, oxytocin-related activities should be postponed. Reproduction takes a back seat to survival itself.

The modern environments of humans, especially those in which postpartum women are expected to resume pre-pregnancy social activities within a few days or weeks following childbirth, introduce chronic levels of stress by constant exposure to novelty. This may not only affect lactation, but also important central functions of oxytocin.

**Oxytocin in Maternal Affect**

Oxytocin's coordination of peripheral and central functions in the mammalian mother may accomplish more than a synchrony of physical functions and appropriate behaviors; oxytocin may also induce emotions and central psychological states that serve as internal motivators for these adaptive behaviors. Panksepp (1992:243) writes, "neuropeptides may serve as key neuromodulators within the genetically provided emotional operating
systems of the brain that coordinate behavioral, physiological, and psychological responses to major life-challenging circumstances." A prediction derived from this is that brain oxytocin may evoke feelings of warmth and comfort. Applying this general principle to the specific case of the mammalian mother, Panksepp (1992:244) predicts that central oxytocin "would contribute to a sense of ease and relaxation." Insel and Shapiro (1992:137) also remark on the "intuitive appeal to the suggestion that oxytocin influences the motivational or affective properties of maternal behavior...[by making] pup contact rewarding."

While natural selection operates on behaviors and not the motivations for behaviors, affective states may still result from the harmony of brain and behavior. That is to say, whether or not pleasure is experienced, mammalian mothers who both breastfeed and nurture will be selected for. However, an epiphenomenon of the brain activity that coordinates maternal physiology and behavior may be the emotions of motherhood. The internal rewards derived from the emotional impact of oxytocin may increase the probability of repeating certain behaviors (as learning theory would predict). However, it is the behavior itself, whether influenced by pleasure or not, on which evolutionary forces act.

With this in mind, what are the internal states experienced during oxytocin release? Newton (1978) observes that oxytocin-related
reproductive acts are associated with certain measurable mood changes. Oxytocin has been linked to many affective states including increased feelings of pleasure and warmth and reduced anxiety, neophobia and depression.

In the breastfeeding mother, feelings of psychological pleasure are often experienced during nursing. The "warm fuzzies" are described as a tranquil and peaceful feeling of happiness and love (Riordan & Rapp, 1980). While in general, oxytocin may be viewed as "one of nature's ways of ushering in joy" (Angier, 1991:C1), in maternal affect it seems to cause feelings of contentment.

Insel (1992) suggests that oxytocin generally facilitates social bonding. It is involved in such affiliative behaviors as allogrooming, sitting together, and attachment. He hypothesizes that oxytocin's prosocial effects are a result of its ability to make social contact rewarding. An alternative and complementary theory is that oxytocin release decreases feelings of neophobia (Insel & Shapiro, 1992). This is supported by the finding that injections of oxytocin in rats increase exploration in a novel environment (Carter, Williams, Witt, & Insel, 1992). However, in a natural situation (without the exogenous introduction of oxytocin), novelty inhibits oxytocin release. It would seem that familiarity is necessary for the endogenous release of oxytocin and that the observed decreases in neophobia are a
result of tricking the system by mimicking the physiology under which it is safe to "open up."

The affective tone caused by oxytocin can also be seen as a reduction in anxiety. In animal studies, oxytocin causes a decrease in distress vocalizations (Panksepp, 1992), and an increase in yawning (Argiolas & Gessa, 1991), behaviors that indicate a generally comfortable central state. For this reason, it has been suggested that drugs that increase oxytocin activity may be useful in treating panic attacks, social phobias, and depression (Panksepp, 1992).

**Oxytocin as an anti-depressant.** An important affective role of oxytocin is its action as an antidepressant (Meisenberg, 1982). Figure 1 summarizes the studies that demonstrate the antidepressant effects of oxytocin.

In a study on mice using the learned-helplessness model of depression, oxytocin is as powerful an antidepressant as the classical tricyclic antidepressant, imipramine. Furthermore, chronic injections over a period of ten days (perhaps more closely resembling oxytocin release during lactation) produces an even more intense effect than similarly administered imipramine (Arletti & Bertolini, 1987). These findings have also been noted by other researchers (Bakharev et al., 1986) using mice and rats.

It is interesting that the behaviorist model for depression (learned
**Behavior:**

Mice less depressed if chronically injected with oxytocin than with imipramine (Arletti & Bertolini, 1987)
Mice and rats less depressed if injected with oxytocin than with placebo. (Bakharev, Tikhomirov, & Lozhkina, 1986)

**Experience of Depression:**

Schizophrenic patients receiving intranasal oxytocin report feeling less apathy, anxiety, and depression, and a general sense of well-being (Bakharev et al., 1986)

**Indirect line of evidence in humans:**

Abnormal levels of oxytocin found in the CSF of manic-depressive, schizophrenic, anorectic, and obsessive-compulsive patients (Demitrack & Gold, 1988; Leckman et al., 1994)

*Figure 1.* The antidepressant effects of oxytocin.
helplessness) relies on a measure that could also be called "perseverance." (Animals are reported to be less depressed if they continue to try to escape an aversive stimulus after many failed attempts.) Recalling the explanation that natural selection acts on behaviors (not emotions), oxytocin may cause more perseverance rather than less depression. While perseverance may be the behavioral corollary of positive affective tone, there are logical gaps in the assumption that animals who continuously strive to escape pain actually feel less depressed.

To assess whether the experience of depression is reduced by oxytocin, it is necessary to ask human subjects. Bakharev and colleagues (1986) find that oxytocin administered intranasally to schizophrenic subjects has a positive effect on affective tone. Subjects report a decrease in feelings of apathy, anxiety, and depression, and an increase in a general sense of well-being. Indirect evidence that oxytocin may act as an antidepressant in humans is that estrogen can be used to treat depression (McEwan & Parsons, 1982; Oppenheim, 1983); again, the pathway by which estrogen has this effect may be by causing a proliferation of oxytocin receptors, thereby creating a significant increase in oxytocin function.

Another indirect line of evidence that oxytocin may have an antidepressant effect in humans is that many psychological illnesses are associated with oxytocin dysfunction. Abnormal levels of oxytocin have been
The Adaptive Value of Maternal Depression

In Chapter II, maternal behaviors were found to follow certain patterns based on evolutionary predictions. If emotions are seen as the experiential corollaries of behavior, then they should parallel the patterns predicted for behavior. Here, I am assuming that physiological mechanisms in the brain synchronize behavior with emotional experience. In particular, since behaviors reflect a subconscious goal to increase reproductive success, emotions—the experiential corollary of these behaviors—should also reflect this goal. This is Nesse's (1990) provocative statement.

Nesse explains, "Happiness is aroused by information that is often correlated with increasing reproductive success—being admired, being loved, making love, having children, watching them succeed, and having grandchildren. Sadness is aroused by situations associated with decreasing fitness—sickness, loss of resources, social rejection, loss of a friend or lover, or the death of a child. Happiness and sadness seem to be tracking some environmental variable, but what is it? Can it be fitness itself?" (1990:273).

He goes on to explain that things that feel good (and are good for reproductive success) are repeated in order to achieve that state of happiness again. Thus, instrumental learning occurs with an internal reinforcement—feeling good. However, he also states that people avoid
actions that make them feel sad. While this claim makes intuitive sense, it is somewhat at odds with current models of learning. Aversion learning only occurs when one behavior leads to an aversive stimulus while an alternative behavior does not. Seligman and Maier's (1967) famous experiments in which dogs received electric shock without an opportunity to avoid the pain established the learned-helplessness effect. Without an alternative behavior, the dogs soon stopped trying to escape the shock at all. This inactivity in response to chronic, uncontrollable pain is the behaviorist model of depression (see also Seligman, 1968).

In Nesse's list of situations that make one sad (especially the death of a child), sad feelings do not necessarily lead to avoidance behavior—they can also lead to depression. (No alternative behavior will bring back this particular child. The investment of gestation and postnatal care is lost.) Is depression ever adaptive? Is complete apathy and lack of action ever good for one's reproductive success? The answer may truthfully be no—depression may simply be the malfunctioning of a complicated system under rare conditions. However, the answer may be yes—under certain circumstances, temporary inaction may be the best action. What might these conditions be? Inaction may be adaptive when large amounts of energy would otherwise be wasted—when an altricial mammalian newborn dies.

As was discussed earlier, maternal behaviors, especially in mammals,
are very "expensive." Lactation alone raises energy requirements between 20 and 50 percent in primates (Buss & Voss, 1971). Long-term effects of lactation in females with a calcium-poor diet include a reduction in stored calcium for future pregnancies (Leon, 1987), although this does not apply to females with a calcium-rich diet. When an infant has died, the myriad of maternal behaviors that have been turned on at parturition should be turned off again, lest precious resources be wasted. While on the experiential level depression is painful, it is possible that the physiological mechanisms that produce it are acting in the individual's best interest genetically. Evolutionary theory predicts that the harmony of physical and behavioral events in mammalian motherhood should only be displayed when an infant is alive and capable of survival. When an infant dies, it is adaptive for these maternal functions and behaviors to cease. Changes in maternal affect may be a by-product of this adaptive shift. In sum, the behavioral correlates of depression may actually be adaptive in certain circumstances, and the experience of depression may be dependent on the same neural networks.

If Nesse is right about the adaptive value of emotions, oxytocin may be the missing physiological link. It is released during reproductive or social behaviors and it feels good (or at least it prevents feeling bad). Its role in humans has not yet been clearly delineated, but it is quite possibly the messenger of reproductive success that delivers happiness.
CHAPTER IV

IMPLICATIONS FOR POSTPARTUM DEPRESSION

In the previous discussion about oxytocin and the adaptive value of depression, the evolutionary significance of the physiological cues and responses is clear. When an infant dies, suckling and lactation stop, and oxytocin levels are reduced. The antidepressant effects of oxytocin halt, leading to temporary inactivity (which prevents energetic loss) and a return to menstrual cycling. It is in the reproductive female's best genetic interest to cease maternal behaviors and return to fecundity.

It is possible that in the absence of breastfeeding, Western women experience a similar endocrinological state. When suckling—the critical stimulus that has always accompanied a live, healthy offspring—is absent, the physiology under which behavioral inactivity is adaptive (when an infant dies) may be mimicked. This change in brain activity may be experienced as depression. While nonbreastfeeding women clearly know that they must take care of their infants, they may experience a conflicting internal signal. Because the infant is not suckling, the mother may receive subconscious information that it does not need to be nurtured. Thus, because the evolved complex of maternal functions, behaviors, and emotions depends somewhat on the critical stimulus of suckling, postpartum depression may be
experienced in the absence of lactation even when the behavioral correlates of depression are not appropriate.

An additional strain for the nonbreastfeeding mother is that not only is she dealing with the normal stresses of motherhood without the buffer of oxytocin, she is dealing with the behavior of infants that are not receiving breast milk, yet another novel phenomenon in the natural history of mammals. This means she may be subjected to an infant that cries more, has more colic, experiences more gastric upset, and displays more unsettled behavior.

The mismatch between what the physiology of nonlactation has always "meant" to a female mammal's body and what it "means" for many Western women today may pose a modern problem for maternal well-being.

Definitions and Distinctions

Before exploring the evidence for this hypothesis, it is necessary to define postpartum depression and to distinguish it from the two other major psychological disorders that occur in the postpartum period. The three levels of postpartum depression recognized in the psychological literature are outlined in Table 1. Postpartum depression (PPD) is the intermediate disorder in terms of prevalence and severity. It is both more prevalent and
Table 1

*The Three Levels of Postpartum Depression*

<table>
<thead>
<tr>
<th>Blues:</th>
<th>Depression:</th>
<th>Psychosis:</th>
</tr>
</thead>
<tbody>
<tr>
<td>50-80% U.S.</td>
<td>10-30% U.S.</td>
<td>0.1-0.2% U.S.</td>
</tr>
<tr>
<td>onset 3 or 4 days postpartum</td>
<td>onset 4 to 6 weeks postpartum</td>
<td>onset a few months postpartum</td>
</tr>
<tr>
<td>one or two days of:</td>
<td>months of:</td>
<td>months of:</td>
</tr>
<tr>
<td>crying</td>
<td>crying</td>
<td>depressive symptoms (same</td>
</tr>
<tr>
<td>irritability</td>
<td>irritability</td>
<td>as depression</td>
</tr>
<tr>
<td>insomnia</td>
<td>disturbed sleep</td>
<td>column)</td>
</tr>
<tr>
<td>tension</td>
<td>dysphoria</td>
<td>PLUS,</td>
</tr>
<tr>
<td>anxiety</td>
<td>anxiety</td>
<td>confusion</td>
</tr>
<tr>
<td></td>
<td>changes in appetite</td>
<td>hallucinations,</td>
</tr>
<tr>
<td></td>
<td>self-doubt</td>
<td>delusions</td>
</tr>
<tr>
<td></td>
<td>mood swings</td>
<td></td>
</tr>
</tbody>
</table>
less severe than postpartum psychosis, and less common and more serious than "baby blues."

**Postpartum psychosis.** The most severe of the three defined postpartum disorders, postpartum psychosis, occurs in only one or two out of every thousand postpartum women. Onset typically occurs in the first two to four weeks postpartum (Cutrona, 1982) and symptoms include delusions, auditory hallucinations, obsessive thoughts, severe mood swings, and memory loss (Boyle, 1993).

Based on the familial and personal psychiatric histories of the women who develop postpartum psychosis and its general resemblance to other psychoses, it is believed that postpartum psychosis is actually schizophrenia or bipolar affective disorder with onset during the postpartum period. The psychological and physical stresses of childbirth are viewed as precipitants for a disease to which these women are already predisposed (Cutrona, 1982; Dean & Kendell, 1981; Gitlin & Pasnau, 1989).

It is interesting to note that bromocriptine, a drug that was widely prescribed in the past to inhibit lactation, has been linked to the onset of mania in postpartum psychosis (Cantebury, Haskins, Kahn, Saathoff, & Yazel, 1987; Lake, Reid, Martin, & Chernow, 1987).
**Baby blues.** The baby blues occurs in one to two thirds of postpartum women (Pitt, 1973), making it statistically normal. Symptoms include tearfulness, confusion, anxiety, and mild dysphoria. Onset is typically the third or fourth day after birth, and the duration of symptoms is usually only one or two days (Boyle, 1993; Cutrona, 1982).

Because of the concurrence of the baby blues and dramatic hormonal changes following birth, it is considered a model for psychobiological research (Campbell, 1992; Pitt, 1973). Cutrona (1982) notes that symptoms of the baby blues strongly resemble those of premenstrual tension. This implies that estrogen withdrawal (the hormonal common denominator of these two conditions) may be partly responsible for mood changes in the immediate postpartum. This interpretation is supported by the finding that women who get the baby blues experience a greater drop in free estriol levels after birth than women who do not get the baby blues (O'Hara, Schlechte, Lewis, & Wright, 1991). In this psychobiological model, it is not the absolute level of estrogen, but the withdrawal from the pregnancy-elevated level, that is linked to symptoms of depression. Presumably, when estrogen levels are very high, a desensitization of estrogen receptors occurs in response to increased activity. When estrogen drops dramatically after parturition, there is a period of adjustment to the new, lower levels of circulating estrogen. After receptors adapt to this level, symptoms subside.
Since profound drops in estrogen are a hallmark of parturition, it is not surprising that the rate of this mild postpartum mood disorder is so high, if it can be called a disorder at all.

**Postpartum depression.** Ten to thirty percent of Western women experience the intermediate disorder, postpartum depression (PPD) (Boyle, 1993), which is sometimes called "moderate postpartum depression" to avoid confusion. Cutrona notes that low estimates are based on hospitalization rates and that high estimates are based on relatively loose diagnostic criteria (1982). Onset occurs in the weeks to months following childbirth and duration is at least a few weeks. The syndrome is characterized by symptoms of depression and anxiety that interfere with normal functioning. Specific symptoms used to define PPD vary from study to study, but include prolonged dysphoric mood, fatigue, apathy, hostility, social dysfunction, and disturbances in appetite and sleep (Cutrona, 1982; Dalton, 1971).

The quality that sets PPD apart from major depression occurring outside the postpartum period is an increased lability of symptoms (Dean & Kendell, 1981). In fact, because the prevalent symptoms tend to change over time, PPD posed something of a threat to the system of classification used for mental illnesses early in this century. For this reason, repeated attempts have been made to remove the term "postpartum" from psychiatric
literature altogether (Hamilton, Neel, Harberger, & Parry, 1992). Ironically, it is this quality that validates the construct. The unique pattern of shifting clusters of symptoms makes PPD different from depression occurring outside the postpartum period.

Attempts to uncover demographic factors that predict vulnerability for PPD have not been consistently successful. Low maternal age and parity have been correlated with PPD in some studies (Frost, Stevens, & Lum, 1990; Nott, Franklin, Armitage, & Gelder, 1976; Paykel, Emms, Fletcher, & Rassaby, 1980), but not in others (Cox, Connor, & Kendell, 1982; Gotlib, Whiffen, Mount, Milne, & Cordy, 1989). A more consistent relationship has been noted between PPD and a lack of social support (Cox et al., 1982; Cutrona, 1982; Frost et al., 1990). Most demographic studies have not considered breastfeeding as a potential factor (noted by Auerbach & Jacobi, 1990). (Those that have will be reviewed at the end of this chapter.) The mixed results of demographic studies may reflect not only the heterogeneity of women who are diagnosed with PPD, but also the various clusters of symptoms by which PPD has been identified.
A Biocultural Approach

Physiological explanations for PPD. The list of physiological factors suspected to have a role in the development of PPD is similar to that for the onset of maternal behaviors, with a few notable additions and one important omission. Researchers have explored contributions of the gonadal steroids, estrogen and progesterone, and the pituitary hormone, prolactin (Filer, 1992; Nott et al., 1976). In addition, PPD has been explained as being partly due to a post-natal withdrawal from pregnancy-elevated levels of thyroid activity (Hamilton, 1992), cortisol (Bonnin, 1992), and endorphins (Smith & Thomson, 1991). Perhaps because of the relatively recent finding that there are oxytocin receptors in the brain (Sofroniew, 1983), reviews of possible biological factors involved in PPD have completely neglected this neuropeptide (e.g., Cutrona, 1982; Filer, 1992; Smith & Singh, 1992).

The finding that oxytocin can be a powerful antidepressant in nonhuman animals and humans (Arletti & Bertolini, 1987; Bakharev et al., 1986), paired with the knowledge that it is released in significant surges during the postpartum period when breastfeeding occurs, makes oxytocin an intriguing candidate in the search for physiological factors for PPD. Dalton (1971) finds that many women who later develop PPD experienced prolonged labor. Oxytocin dysfunction (perhaps due to environmental stress
inhibiting oxytocin release) could both prolong labor and contribute to depression in these women. Indeed, the consistent finding that stress correlates with PPD (Cutrona, 1982; Paykel et al., 1980) supports the idea that oxytocin may function as an antidepressant in the postpartum period. The fact that oxytocin is inhibited by stress leads to the prediction that if oxytocin is important in preventing depression, its inhibition by stress would be associated with higher levels of PPD. In addition, the oxytocin-inhibiting effect of the specific stressor of novelty suggests that postpartum women may benefit from a period of seclusion after birth.

The theory that oxytocin acts as an antidepressant in the postpartum period leads to the prediction that societies in which women are encouraged to breastfeed and are subjected to less novelty stress in the immediate postpartum will have a lower rate of PPD.

Cultural influences. Stern and Kruckman (1983), in a review of cross-cultural literature, found that the high rates of PPD suffered by Western women are not experienced outside the West. They conclude that the only real difference between the societies in which PPD is prevalent and those in which it is rare is the social structuring of the postpartum period. Specifically, they state that women profit from the experience of a distinct postpartum period, ritualized protective measures, social seclusion, mandated rest,
practical assistance in the house, and the social recognition of their new status as mothers. While this finding complements the physiological explanations that have been described here (especially the predicted advantage of a period of seclusion), Stern and Kruckman believe that their cross-cultural data rule out biological explanations. Kruckman writes, "Postpartum depression cannot claim a hormonal etiology or we would see its expression globally" (1992:140).

The assumption that postpartum women share the same endocrinological state around the world overlooks the significant effects of feeding method and levels of stress on oxytocin function. Researchers on the possible relationship between breastfeeding and PPD note, "Whereas all women experience massive and rapid changes of hormonal levels immediately following delivery of the placenta... the pattern of feeding is the principal factor influencing the woman's hormonal state later in the postpartum period" (Alder & Bancroft, 1988:389). Johnston and Amico (1986) have found that there are even differences between the hormonal states of partial and exclusive breastfeeders. In their study of oxytocin release in four exclusive breastfeeders, four partial breastfeeders, and three bottle-feeders from the U.S., they found that plasma oxytocin levels stimulated by suckling in partial breastfeeders remained the same over a period of twenty-four weeks. However, the stimulated plasma oxytocin of
exclusive breastfeeders increased significantly over this period of time. In addition, levels of stimulated oxytocin were significantly higher in exclusive breastfeeders than in partial breastfeeders starting at five weeks postpartum. Prolactin levels were also different. While both kinds of breastfeeders experienced a gradual decline in prolactin levels over the twenty-four weeks, initial and twenty-fourth week levels of prolactin were both higher in exclusive breastfeeders (see Figure 2).

While stress is not as easily measured as breastfeeding, it has been consistently quantified and positively correlated with PPD. Cortisol taken from saliva can serve as a physiological indicator of stress. Cortisol levels have been negatively correlated with postpartum mood; specifically, a peak in cortisol coincides with the fourth day postpartum blues. An interesting interaction between stress and breastfeeding early in the postpartum period is that cortisol correlates with mood in bottle-feeders, but not in breastfeeders (Bonnin, 1992). It would seem that some of the physiological effects of breastfeeding can supersede or act as a buffer for those of stress.

The ability of stress to inhibit oxytocin provides a pathway by which stress can affect hormonal state. Since the inhibition of oxytocin by stress can "lead to major problems in either initiating or maintaining lactation" (McNeilly et al., 1994:146), stress influences breastfeeding and all of its hormonal correlates.
Figure 2. Stimulated oxytocin in partial and exclusive breastfeeding.

(Data are from Johnston & Amico, 1986.)
Synthesis: A biocultural model. What emerges is an etiology of PPD that links cultural beliefs and practices with physiological mechanisms that have evolved to deal with the energetic costs of motherhood. Cultural factors have an influence on the behaviors and perceptions that can alter hormonal state. To say that either hormones or social factors are exclusively responsible for the development of PPD is to neglect the meaningful and multiple interactions between such variables as cultural practices and levels of stress, social seclusion and oxytocin levels, and cultural beliefs and rates of breastfeeding. In my own research on postpartum rituals in Romania, I find that the social structuring described by Stern and Kruckman (1983) is linked by implicit expectations and explicit beliefs to the practice of breastfeeding (see Appendix).

The interactions among the social and biological factors explored in this thesis are schematized in Figure 3. This list of factors is selected to demonstrate the interaction between culture and physiology, and is not exhaustive of the significant risk factors for the development of PPD. It is of particular interest that culture can affect breastfeeding rates via so many pathways (directly because of beliefs and expectations about breastfeeding itself, and indirectly through pathways of postpartum rituals and stress.) Another interesting cascade is that while less social support and more stress both contribute directly to postpartum depression, part of their effect may be
Figure 3. A biocultural model of postpartum depression.
through their negative influence on oxytocin activity.

Following the arrows leading from breastfeeding and social support to postpartum depression, it is easy to determine the best and worst case scenarios for maternal well-being. This model predicts that if breastfeeding and social support are both present, there is the least likelihood of developing PPD. If neither is present, there is the greatest risk. While the relative contributions of social support and breastfeeding have not been determined, one would expect an intermediate risk of developing PPD in situations of breastfeeding without social support or social support without breastfeeding (see Figure 4).

Absence of Lactation and Postpartum Depression

I have implied that the cross-cultural data support my hypothesis that decreased breastfeeding may be a principal factor in the increased PPD present in Western women. (Levels of breastfeeding and stress, together, change the average level of oxytocin function. Oxytocin activity, in turn, has a direct effect on depression.) I do not deny the psychological value of postpartum rituals, but I argue that at least part of their advantage stems form indirect action, via their capacity to encourage breastfeeding and provide a low-stress environment for the development of the milk-ejection reflex.
Figure 4. Predicted levels of postpartum depression.
What Western women suffer from may be the risk factor of a stress-induced and culturally accepted (or even encouraged) reduction in breastfeeding.

While little research has directly addressed the relationship between breastfeeding and postpartum depression, most of it has been plagued by poor definitions of both PPD and breastfeeding, lumping together women who exclusively breastfeed on demand with those who supplement and feed by the clock (discussed by Auerbach & Jacobi, 1990). Perhaps it should not be surprising that studies have demonstrated positive, negative, and no correlations between breastfeeding and postpartum depression (Alder & Bancroft, 1988; Alder & Cox, 1983; Cox, Connor & Kendall, 1982; Dalton, 1971; Kumar & Robson, 1978).

Past research refuting hypothesis. Only one study has found a significant positive correlation between breastfeeding and postpartum depression. Alder and Bancroft (1988) found that slightly more breastfeeders than artificial feeders are depressed at three months, while this pattern disappears by six months (Alder & Bancroft, 1988). The cases of PPD in this study were diagnosed with Goldberg's Standardized Psychiatric Interview (SPI) and Goldberg's General Health Questionnaire (GHQ). Goldberg's SPI is a psychological test created specifically to identify "changes in the mental state" (Goldberg, Cooper, Eastwood, Keward, & Shepherd, 1970:23).
Goldberg's GHQ is a longer questionnaire consisting of 28 items designed to measure four subscales based on somatic symptoms, anxiety and insomnia, social dysfunction, and severe depression. At three months, the breastfeeding women do not differ on the subscale of severe depression, they differ only on the social dysfunction subscale.

Recalling the earlier discussions of a parturitional switch, a tendency for mothers to be protective of their young, and the benefits of a period of seclusion, it appears that postpartum changes in social functioning are actually normal and adaptive. In fact, PPD researchers have noted the particular inappropriateness of using the Goldberg GHQ to diagnose this kind of depression (Watson & Evans, 1986). The ecological validity of a diagnosis of PPD based on criteria such as "defensiveness" and a "lack of spontaneity" (Goldberg et al., 1970:20) must be questioned when maternal protectiveness and caution are both predicted by evolutionary theory. In fact, the absence of these adaptive behaviors on the part of bottle-feeding mothers may actually pose a threat to the health of the mother and/or child.

Two other studies that claim to find a positive relationship between breastfeeding and PPD are based on statistically insignificant data. First, it has been found that women who become depressed are more likely to breastfeed at two weeks postpartum than women who do not become depressed (Dalton, 1971). This conclusion is based on the fact that 35% of
the 175 normal British women were breastfeeding at two weeks postpartum, while 57% of the 14 depressed subjects were. A second study found that women who exclusively breastfeed for at least twelve weeks have a higher incidence of depression than those who partially breastfeed (Alder & Cox, 1983). Again, neither of these differences were significant.

Two more studies relying on Goldberg's GHQ or SPI for diagnosis failed to show any relationship between breastfeeding and PPD (Cox, Connor, & Kendell, 1982; Kumar & Robson, 1978). These inconsistent findings, again, are partly due to poor definitions of PPD, which are plagued by diagnostic criteria that describe some normal maternal adaptations. The second major problem with these studies is that breastfeeding groups are defined arbitrarily, and frequently in ways that ensure within group variability.

This intragroup heterogeneity is primarily a result of attempting to include all subjects in two categories, at the expense of ignoring meaningful differences in feeding practices. For example, Dalton (1971) has only two kinds of breastfeeding, those who are successfully breastfeeding at two weeks and those who are not. She does not mention whether these are partial or exclusive breastfeeding, whether they feed by the clock or on demand, or even if the milk-ejection reflex is ever established.

The same criticism can be applied to Alder and Bancroft’s (1988) study, in which the binary classification is based on whether mothers fully
breastfed for more than twelve weeks or less. In this case, the arbitrary definition is even more suspect because the measures of depression were taken one to two years postpartum, after all but one mother stopped breastfeeding. The authors actually correlated a measure of breastfeeding (based on an arbitrary cut-off) with mood assessed at least one year later in these women. It is remarkable that the same pool of subjects used in Alder and Cox's (1983) study showed no relationship between breastfeeding and depression when studied up to five months postpartum by Cox, Connor, and Kendell (1982)!

The same problem of comparing affect and breastfeeding at non-overlapping time points is present in Alder and Bancroft's (1988) study. Here, the cut-off is six weeks of full breastfeeding. However, measures of mood (based on Goldberg's GHQ) were taken at three and six months. At three months, more than one half of the "fully breastfeeding" group were not fully breastfeeding. At six months, none of them were. Researchers cannot discover the relationship between breastfeeding and postpartum affect when these two variables are measured at different times.

In sum, previous research concluding that breastfeeding has a negative or neutral influence on maternal affect suffers from three major problems: an invalid measure of PPD, arbitrary and incomplete definitions of breastfeeding, and correlations of events that are not concurrent. The
proposed independent variable (breastfeeding), dependent variable (affect), and their correlation are all flawed. Combined, these problems shed great doubt that any conclusions drawn from these studies.

Past research supporting hypothesis. Evidence linking a reduction in breastfeeding to depression is both direct and indirect. Researchers relying on measures of depression other than Goldberg's GHQ or SPI (e.g., the Beck Depression Inventory) have found a negative correlation between depression and breastfeeding. In addition, it may not be a trivial detail that the studies that found a positive or no correlation between breastfeeding and PPD used only British women as subjects, while the studies that found a negative correlation interviewed women from the U.S., Finland, Canada, and Chile.

One study found that a significantly higher proportion of mothers who are categorized as depressed at three months begin artificial feeding by that time (Fleming, Flett, Ruble, & Shaul, 1988). These women not only breastfeed less, but also displayed less affectionate behavior with their infants at the interview. Similarly, it has been observed that depressed mothers have more difficulty breastfeeding (Tamminen, 1988). It is difficult to determine causation in this situation. An original reduction in breastfeeding may contribute hormonally to the depression (via reduced oxytocin activity.) Alternatively, the depression may lead to less breastfeeding (as Tamminen
assumes). Either way, reduced breastfeeding is associated with depression. Another study designed to correlate factors of pregnancy, labor, and breastfeeding with PPD found that only major stress situations (like emergency caesareans) and a decrease in breastfeeding duration are significant factors in the development of depression in the postpartum period (Alvarado, Perucca, Rojas, Monardes, Olea, Neves, & Vera, 1993).

Indirect evidence comes from differences in transient mood states in breastfeeding and bottle-feeding women. In one study, partial breastfeeders were studied. These women experienced more depression, stress, fatigue, guilt, and anxiety when bottle-feeding than when breastfeeding (Modahl & Newton, 1979). In another study, researchers found that new mothers who breastfeed at five weeks postpartum are much less emotionally distressed than their bottle-feeding counterparts (Rickels, Garcia, Lipman, Derogatis, & Fisher, 1976). In this study, lactation-suppressed mothers were not more distressed in the immediate postpartum. Significant differences only developed by week five postpartum—a typical time for the onset of PPD.

One last bit of indirect evidence is that the hormonal state of depressed breastfeeders looks like that of bottle-feeders. Specifically, it has been found that there are significant differences between the prolactin levels of depressed and normal breastfeeders and that "in effect, the concentration of prolactin in depressed breastfeeders [is] at levels more appropriate for
bottle-feeders" (Harris, Johns, Fung, Thomas, Walker, Read, & Riad-Fahmy, 1989:666). A supporting finding is that prolactin secretion increases when patients recover from depression outside of the postpartum period (Fava, Lisansky, Buckman, Kellner, Pathak, Fava, & Peake, 1988). While neither of these studies looked at oxytocin, it is feasible that oxytocin activity would also be abnormally low in depressed breastfeeders and that oxytocin levels would increase in patients recovering from depression. As was discussed earlier, the two hormones are intimately related. In fact, elevated prolactin in the blood increases the activity of centrally released oxytocin (Crowley, Parker, Armstrong, Spinolo, & Grosvenor, 1992). The hormonal resemblance of depressed breastfeeders and bottle-feeders may indicate intragroup variability in the amount or success of breastfeeding. Since these authors do not distinguish between partial and exclusive breastfeeding, it is feasible that many of the depressed breastfeeders (who produced significantly less prolactin) were partial breastfeeders.

**Directions for future research.** Testing the hypothesis that the absence of breastfeeding is linked to PPD via suppressed oxytocin requires several amendments to current trends in PPD research. First of all, oxytocin should be considered as a hormonal factor in physiological examinations of etiology. Breastfeeding groups should reflect truly distinct feeding practices.
Measurements of affect and breastfeeding success should be taken at the same time. Lastly, an ecologically valid questionnaire should be created.

The first and most obvious change is that PPD studies should include a measurement of oxytocin. A positive correlation between oxytocin activity and depression would falsify this hypothesis in one clean sweep, while a negative correlation would support it. Animal studies could directly examine the relationship between depression, lactation, and oxytocin. This thesis predicts that a valid animal model of depression in the postpartum period can be created by physically blocking lactation after parturition. Subjecting these animals to classical behavioral tests of depression would be very interesting. If the nonlactating animals become helpless faster than the lactating animals, the theory is supported. The central oxytocin activity in these animals could be directly assessed by either radioimmunoassay of oxytocin in CSF or by using in vitro radioligand receptor assays to quantify receptor regulation in the brain.

Human studies would probably not include measurements of central oxytocin (unless patients were scheduled to receive a spinal tap for some other reason) and would be limited to indirect indicators of central oxytocin activity: peripheral levels measured from the plasma and inferred from successful lactation. Peripheral levels are established by radioimmunoassay of blood plasma. Another less invasive way to assess peripheral oxytocin
activity is to interview subjects about their success in breastfeeding. Because it has been observed that exclusive breastfeeders have a higher peripheral level of oxytocin (Johnston & Amico, 1986), levels of breastfeeding should correlate with peripheral oxytocin. However, peripheral oxytocin does not provide a flawless indication of central oxytocin levels. Levels of central and peripheral oxytocin do not correlate perfectly, possibly due to the shortened half-life of this peptide in plasma (noted in Capitanio et al., 1985; and Insel, 1992). It is likely that human studies would be restricted to correlations of measures of breastfeeding (indirectly related to central oxytocin activity) and symptoms of depression.

The second major suggestion for future research on the relationship between lactation and PPD is that breastfeeding groups be redefined to reflect more homogeneous practices and hormonal levels. The term "breastfeeding" should refer to women who exclusively breastfeed, preferably on demand (rather than by the clock), including feeding on demand at night. The term "nonbreastfeeding" should be reserved for women who do not breastfeed at all rather than those who partially breastfeed. By eliminating partial breastfeeders from either group, it will be easier to make a clear distinction between the hormonal influences of breastfeeding and not breastfeeding.

With breastfeeding groups strictly defined in this way, the final major
change called for by an evolutionary perspective is a reexamination of normal changes in the postpartum period. There is an assumption in the psychological literature often implied and occasionally stated that normality in the postpartum period is the same as normality before pregnancy begins. This is evident in such statements as "breastfeeders showed greater impairment of sexuality" and "women who feed artificially are likely to resume normal ovarian cyclicity..." (Alder & Bancroft, 1988). In cultures in which postpartum sexual taboos are observed and lactational amenorrhea contributes to ideal birth spacing, these statements seem ludicrous. In fact, in cultures with traditional postpartum rituals, behavioral change is not only accepted, it is enforced by social code. Also, in evolutionary terms the resumption of "normal" ovarian cyclicity implies infant death. In other cultures and in our natural history, changes in postpartum behavior and hormonal status have always been both normal and adaptive.

In the West, where we are reluctant to accept the idea of adult development, changes in behavioral patterns are seen as problems. Thus, a diagnosis of PPD can based on the observation that a change in mental state has occurred. (For a comparison of diagnostic criteria used to diagnose PPD, see Table 2.)

Recalling the chapter on the psychobiology of mammalian motherhood, changes in behavior and possibly affect are "normally"
Table 2

Previous Questionnaires Used to Diagnose PPD

<table>
<thead>
<tr>
<th>Goldberg's SPI: (Goldberg et al., 1970)</th>
<th>Beck Depression Inventory: (Beck et al., 1961)</th>
<th>Hopkins Symptom Inventory: (Rickels et al., 1976)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Self-report:</strong></td>
<td><strong>Mood indicators:</strong></td>
<td><strong>Five dimensions:</strong></td>
</tr>
<tr>
<td>somatic symptoms *</td>
<td>pessimism **</td>
<td>Somatization:</td>
</tr>
<tr>
<td>fatigue</td>
<td>sense of</td>
<td>aches</td>
</tr>
<tr>
<td>sleep</td>
<td>failure</td>
<td>weakness</td>
</tr>
<tr>
<td>disturbances</td>
<td>lack of</td>
<td>pains *</td>
</tr>
<tr>
<td>irritability *</td>
<td>satisfaction **</td>
<td>Obsessive-Compulsive</td>
</tr>
<tr>
<td>lack of concentration</td>
<td>guilt</td>
<td>having to repeat actions</td>
</tr>
<tr>
<td>depression **</td>
<td>sense of punishment</td>
<td>worried about</td>
</tr>
<tr>
<td>anxiety **</td>
<td>self-hate</td>
<td>sloppiness</td>
</tr>
<tr>
<td>phobias</td>
<td>self accusations</td>
<td>Interpersonal</td>
</tr>
<tr>
<td>obsessions/compulsions</td>
<td>self punitive</td>
<td>Sensitivity</td>
</tr>
<tr>
<td>depersonalization</td>
<td>wishes</td>
<td>easily annoyed or irritated *</td>
</tr>
<tr>
<td>observed behaviors:</td>
<td>crying spells **</td>
<td>temper outbursts *</td>
</tr>
<tr>
<td>slow, lacking</td>
<td>irritability *</td>
<td>impulses to hurt others *</td>
</tr>
<tr>
<td>spontaneity *</td>
<td>social withdrawal *</td>
<td>Depression</td>
</tr>
<tr>
<td>suspicious, defensive *</td>
<td>indecisiveness</td>
<td>loss of sexual interest *</td>
</tr>
<tr>
<td>histrionic</td>
<td>body image</td>
<td>poor appetite</td>
</tr>
<tr>
<td>depressed **</td>
<td>work inhibition</td>
<td>feeling blue **</td>
</tr>
<tr>
<td>anxious, tense **</td>
<td>sleep</td>
<td>feeling hopeless **</td>
</tr>
<tr>
<td>elated</td>
<td>disturbances</td>
<td>difficulty sleeping</td>
</tr>
<tr>
<td>flat affect **</td>
<td>fatigue</td>
<td>Anxiety</td>
</tr>
<tr>
<td>excessive</td>
<td>loss of appetite</td>
<td>feeling tense</td>
</tr>
<tr>
<td>concern with bodily functions *</td>
<td>weight loss</td>
<td>having to avoid certain things *</td>
</tr>
<tr>
<td>depressive</td>
<td>somatic pre-occupation *</td>
<td></td>
</tr>
<tr>
<td>thought content **</td>
<td>loss of libido</td>
<td></td>
</tr>
<tr>
<td>confusion, delusions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>hallucinations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>intellectual impairment</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Key:**
- * particularly inappropriate criterion
- ** particularly appropriate criterion
synchronized with the natural sequence of gestation, parturition, and lactation. Comparative psychology demonstrates that nurturing, seclusion, protectiveness, and even aggression are normal. The recognition that a parturitional switch is both predicted by evolutionary theory and observed in animal studies sheds doubt on the implicit assumption that postpartum normality should resemble pre-pregnancy patterns of behavior. A valid questionnaire for the diagnosis of PPD should keep this in mind. Symptoms of "hostility" and "social dysfunction" should not be included if they are actually measuring the maternal aggression and seclusion that have been adaptive responses in our natural history. Rather, the questionnaire should focus on symptoms such as dysphoria, anxiety, and apathy.

The ideal study to test the hypothesis that successful breastfeeding can have a positive influence on maternal affect would be prospective, enrolling subjects during pregnancy. At several time points during the postpartum period, and particularly at two months postpartum (two to four weeks past the typical onset of PPD), subjects would be given both a breastfeeding and depression questionnaire.

Based on their answers to the questions on breastfeeding practices, subjects would be divided into three groups: exclusive breastfeeders, partial breastfeeders, and nonbreastfeeders. Ideally, the exclusive breastfeeders
would meet the additional criterion of feeding on demand (rather than by the clock). Feeding on demand would indicate establishment of the milk-ejection reflex and would imply healthy oxytocin functioning. The focus comparison would be between members of the first and third group, matched for socio-economic status and social support. The PPD questionnaire would be specifically designed to measure symptoms of anxiety and depression. In addition, it would be interesting to include measures of normal maternal changes, such as preferred seclusion and hostility towards strangers, to test the assumption that the breastfeeding group would rate higher on these questions.

I believe that when breastfeeding and PPD are both redefined in this way, a negative correlation will be found between breastfeeding and depression. This expectation is based on the prediction that a lack of suckling triggers certain adaptive behavioral and emotional responses in a new mother, and the observations that oxytocin is an antidepressant and that oxytocin levels are lower in the absence of breastfeeding.
CHAPTER V

CONCLUSION

The model presented here links the decrease in breastfeeding in the West with an increase in the prevalence of postpartum depression. There are both ultimate reasons predicting and proximate mechanisms demonstrating how reductions in breastfeeding may lead to the development of PPD.

The ultimate argument is based on the following observations and evolutionary predictions. In the development of some maternal behaviors, pregnancy and parturition appear to switch on a tendency to act in nurturing and protective ways. The release of oxytocin (presumably, at parturition and during lactation) is specifically responsible for the development of maternal aggression and probably for the formation of crucial bonds between mother and infant. The full suite of maternal behaviors is energetically expensive and, if reproductive success is maximized, is only displayed under the circumstance of a live and healthy birth. Since infant suckling has always been the sine qua non of infant viability, it is likely a critical stimulus that physiologically indicates to the mother that it is wise to continue investing. In our natural history, when suckling in the immediate postpartum ceased, a new mother did well to arrest maternal behaviors, return to fecundity, and try
again, perhaps at the expense of **experiencing** a period of emotional depression. In the modern practice of feeding by the bottle, it is possible that the same physiological message that the infant has died can influence similar changes in behavior and/or affect.

The proximate mechanism by which failure to lactate can be associated with PPD is through the effects of the neuropeptide, oxytocin. Oxytocin is involved in the onset of maternal behaviors and has recently been found to act as a powerful antidepressant. During its release, mothers report feelings of warmth, relaxation, and contentment. Oxytocin can be suppressed by stress and decreased nursing. The hypothesis presented here is that the combination of stress and decreased nursing in Western societies leads to a decrease in oxytocin activity, a decrease in its ability to inhibit the behavioral inactivity that would be adaptive in the case of infant death, and, concurrently, a decrease in its antidepressant actions. This is why one would expect an association between the absence of lactation and depression on a proximate level (see Figure 5).

The model presented here benefits from mutually compatible theories about human behavior in anthropology and psychology. An evolutionary perspective leads to ecologically valid definitions of normal postpartum behavior and predicts that when the critical stimuli that elicit this response are absent, there may be severe consequences. By combining the
Figure 5. Proposed neurohormonal role of oxytocin in maternal affect.
contributions of biocultural anthropology with those of biological psychology, a more holistic view of human behavior emerges, one that recognizes that humans have the ability to create social mandates that influence their own physiology. Approaching psychological research from this biocultural perspective may lead to both a more complete comprehension of the etiology of mental illnesses and to effective treatments or preventative measures derived from this understanding.
REFERENCES


APPENDIX

ROMANIAN POSTPARTUM RITUALS

Stern and Kruckman's 1983 criticism of the biology-heavy literature on postpartum depression (PPD) concludes that a particular pattern of postpartum rituals is associated with lower rates of PPD outside of the West. They list six characteristics of the traditional structuring of the postpartum period in non-Western countries. These are (1) a distinct postpartum period; (2) measures meant to protect the new mother; (3) social seclusion; (4) mandated rest; (5) practical assistance in the house; and (6) the recognition of a new social status for the mother. They mention that it "is reasonable to assume a possible relationship between the decline of breastfeeding in the U.S. ... and [increases in] postpartum depression" (1983:1030), but do not list the encouragement of breastfeeding as a major characteristic of the postpartum period. This may be a serious omission.

In my own research on the traditional treatment of women in the postpartum period in Romania, I find that the encouragement of breastfeeding is linked to adhering to these rituals. My account is based on interviews taken in the summer of 1994 in the Carpathian Mountain village of Leresti, Romania. A synthesis of taped conversations with local elders, priests, and doctors, this ethnographic snapshot reveals a postpartum
structure closely resembling the typical one described by Stern and Kruckman. However, it also highlights the difficulty in dissecting social from biological factors, as interviewees consistently reported both the belief that breastfeeding is critical for the health of an infant and the assumption that a new mother will breastfeed.

The traditional postpartum period in Romania consists of two overlapping periods: the first three and forty days after birth. During the first three days, the new mother stays in bed and does no housework. She is helped by either female relatives or an older woman specifically paid for these services, the “moasa”. The end of this initial period of vulnerability is marked by the infant’s first bath. The water for this bath is brought from the house by the “moasa” to the priest who blesses it; the “moasa” then brings it back for the bath. This bath is viewed as a protective measure for the baby.

Coinciding with this first bath is the creation of an “ursitoare”—a bundle of fabric containing the umbilical cord and various foodstuffs and objects such as cornmeal, sugar, salt, wine, bread, money, pencils, and scissors. This actual bundle is handed over to the “moasa” for payment of her services, but a recreated bundle is put into the baby’s crib one year later on the occasion of the first hair cut. At this future date, the first object the baby chooses from the “ursitoare” predicts his or her eventual profession. (For example, choosing a spool of thread may indicate that she will become a
seamstress.) Therefore, the original "ursitoare" is carefully assembled because of its future significance.

For the first forty days postpartum, the new mother is restrained to her own home and yard. The conscious reason for this is somewhat paradoxical. The mother is considered unclean in a spiritual sense because she has been tainted by her unbaptized baby. However, because the baby is not baptized, it is extremely vulnerable to unfriendly forces (e.g., the envious thoughts of jealous neighbors). Therefore, the mother must defend her baby (the source of her own pollution) for these forty days. The vulnerability is subtly dependent on the time of day and the location of the mother within the house. More central locations and daylight hours offer more protection. The result is that postpartum women avoid going into the yard at night. One interviewee even reported that during the hour of midnight to one o'clock, she and her baby stayed locked up in her bedroom.

The bath at day three helps to protect the baby, but the mother herself goes unprotected until the end of the forty day period. At that time, the priest comes to her house to bless her so that she may leave the house and, more importantly, so that she may enter the church to attend her baby's baptism. If she has not been blessed by the fortieth day, she may not be present at this joyous event.

For the forty day period, the new mother does less work than usual,
her normal load replaced by physically less demanding tasks such as weaving. Neighbors and relatives bring food and help out a little. A list of all visitors is given to the priest, who blesses them before they come in contact with the baby. It is believed that contact with spiritually unclean people will result in rashes and bumps on the baby's face. Great care is taken to avoid this. Among the visitors, very few are males. The husband of the new mother also has restricted contact with his wife—there is a postpartum sexual taboo during the forty day period.

The only special food eaten during this time is a mixture of "tuica" (plum brandy), oil, and sugar. This, along with some massage and abdominal binding, is supposed to restore the woman's figure to its pre-pregnancy shape. The only other change in diet is the avoidance of sour and spicy foods—these are believed to have a negative effect on the quality of breast milk.

When asked about breastfeeding, interviewees were consistently bemused. Asking if the new mother is encouraged to breastfeed was like asking if the baby is encouraged to breathe. However, they politely responded to this confused Westerner's questions; yes, of course breastfeeding is encouraged. One of the local priests even divulged that because of the tense relations often experienced between a new mother and her mother-in-law, the mother-in-law typically expresses her desire that the
new mother breastfeed by asking her son (the new mother’s husband) to encourage her. Thus, in this case, social pressure to breastfeed is exerted indirectly. While the two local doctors approximated that breastfeeding duration is presently between four and seven months, the older women interviewed reported that only a few generations ago, women normally breastfed for between twelve and fifteen months. Coincidentally, or not, the new generation is characterized by both less breastfeeding and a decline in following the traditions described here.

By taking an in-depth look at one culture’s structuring of the postpartum period, Stern and Kruckman’s model is reconfirmed (see Table 3). All six elements were found in this treatment of postpartum women: a distinct period of forty days, protective measures (although more for the baby than for the mother), restricted social contact, mandated rest (at least for three days), assistance from relatives and the "moasa", and social recognition at the time of the purification of the mother before the baby's baptism. However, the implicit encouragement of breastfeeding also correlates with these rituals. In their capacity to reduce stress and allow for the non-stress release of oxytocin, the importance of postpartum rituals may be partly physiological—allowing for the development of the milk-ejection reflex and successful breastfeeding. Seclusion and rest, in particular, may be critical in creating the kind of non-stress environment necessary for oxytocin
Table 3

*Traditional Structuring of the Postpartum Period*

<table>
<thead>
<tr>
<th><strong>Stern &amp; Kruckman's (1983) Characteristics of the Postpartum Period:</strong></th>
<th><strong>Romanian Postpartum Rituals (taken from 1994 interviews in Leresti, Romania)</strong></th>
</tr>
</thead>
</table>
| 1. A distinct period | 1. first 3 days in bed  
first 40 days in house and yard |
| 2. Protective measures | 2. bath for baby at 3 days  
avoid "unclean" people  
purification for mother and  
baptism of baby at 40 days |
| 3. Social seclusion | 3. only blessed visitors  
avoid spiritually unclean people |
| 4. Mandated rest | 4. bed rest first 3 days  
restricted work 40 days |
| 5. Practical assistance in the house | 5. "moasa" helps with bathing  
relatives help with food and work |
| 6. Recognition of new social status for mother | 6. cleansing ritual and  
attendance of baby's baptism |

**More Breastfeeding**  
--less novelty stress allows for oxytocin activity necessary for milk-ejection reflex?

**Breastfeeding**  
--when traditions were more strictly followed, breastfeeding duration was longer:  
50 years ago, 12-15 mo.  
currently, 4-7 mo.
release. The recognition that physiological functions are partly dependent on social norms and expectations does not diminish the importance of postpartum rituals. It does, however, validate a biocultural approach to PPD research.
VITA

Kelly J. Peyton  
100 Landrum Village  
Montgomery, Texas 77356

EDUCATION

1994-1996  Texas A&M University  
College Station, Texas  
M.A., Anthropology  
4.00 g.p.a., Regents Fellowship

1991-1992  Emory University  
Atlanta, Georgia  
Psychobiology  
3.81 g.p.a., Woodruff Fellowship

1987-1991  Pennsylvania State University  
State College, Pennsylvania  
B.A., Medical Anthropology  
3.89 g.p.a., summa cum laude

WORK EXPERIENCE

1995-present;  Research assistant to Dr. A. I. Schafer  
1993  V.A. Medical Center (Houston), Medical Hematology

1991-1992  Research assistant to Dr. F. deWaal,  
Yerkes Regional Primate Research Facility  
Emory University, Psychology

1991  Research assistant to Dr. G. E. McClearn,  
Penn State, Health and Human Development

PUBLICATIONS