0013-7227/01/$03.00/0
Printed in U.S.A.

The Journal of Clinical Endocrinology & Metabolism 86(10):4798–4804

Effects of Suckling on Hypothalamic-Pituitary-Adrenal Axis Responses to Psychosocial Stress in Postpartum Lactating Women

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In several studies lactation has been shown to be associated with a hypothalamic-pituitary-adrenal axis hyporesponsiveness to physical and psychological stressors. As it is not known whether the marked blunting of endocrine stress reactivity in women can be ascribed to suckling as a short-term effect or to lactation in general, the acute effects of suckling on the hypothalamic-pituitary-adrenal axis and the sympathetic-adrenal-medullary system responses to mental stress were investigated in lactating women.

Forty-three lactating women were randomly assigned either to breast-feed or to hold their infants for a 15-min period with the onset 30 min before they were exposed to a brief psychosocial stressor (Trier Social Stress Test). Both breast-feeding and holding the infant yielded significant decreases in ACTH, total plasma cortisol, and salivary free cortisol (all $P < 0.01$). There were no significant differences in baseline hormone levels between the groups 1 min before the stress test. In response to stress exposure, ACTH, total plasma cortisol, salivary free cortisol, norepinephrine, and epinephrine were significantly increased in all lactating women (all $P < 0.001$). However, total cortisol and free cortisol responses to stress were attenuated in breast-feeding women ($P = 0.001$ and $P = 0.067$, respectively), who also showed significantly decreasing PRL levels during the stress test ($P = 0.005$). In addition, there was no change in plasma oxytocin or vasopressin in response to the stressor. Breast-feeding as well as holding led to decreased anxiety ($P < 0.05$), whereas, in contrast, stress exposure worsened mood, calmness, and anxiety in the total group (all $P < 0.001$).

From these data we conclude that lactation in women, in contrast to that in rats, does not result in a general restraint of the hypothalamic-pituitary-adrenal axis response to a psychosocial stressor. Rather, suckling is suggested to exert a short-term suppression of the cortisol response to mental stress. (*J Clin Endocrinol Metab* 86: 4798–4804, 2001)

EVIDENCE FROM ANIMAL and human studies has suggested that suckling is associated with significant alterations in the reactivity of the hypothalamic-pituitary-adrenal (HPA) axis during the postpartum period (1, 2). Observations in the lactating rat have shown an endocrine hyporesponsiveness to physical and psychological stressors, including attenuated secretion of ACTH (3–7), corticosterone (3–5, 8–11), catecholamines (12), oxytocin (9, 13–15), and PRL (12, 16).

In humans, the effect of lactation on stress-responsive neurohormonal systems has been explored, to date, only for treadmill exercise. Altemus and associates (17) found significantly attenuated plasma ACTH, cortisol, and glucose responses to physical stress in lactating compared with nonlactating women. However, the effects of suckling on endocrine as well as behavioral responses to a psychosocial stress paradigm in humans have not yet been investigated. More important, it is not known whether the marked blunting of endocrine stress responsiveness can be ascribed to suckling as a short-term effect or to lactation in general.

In lactating women, suckling increases both oxytocin and PRL release and decreases plasma levels of ACTH and cortisol, suggesting an inhibitory influence of lactogenic peptides on the HPA axis (8, 18–20). In addition, systemic administration of exogenous oxytocin decreased basal as well as pharmacologically stimulated ACTH and cortisol plasma levels in healthy humans (21–30). Notably, suckling results not only in peripheral (31, 32), but also in intracerebral release of oxytocin (33–35). An inhibitory effect of brain oxytocin on basal and stress-induced HPA axis activity has recently been described in rats (36).

The present study was undertaken to determine the effects of suckling on the HPA axis and the sympathetic-adrenal-medullary system responses to a brief psychosocial stress exposure compared with those in lactating women who were only holding their infants before the stressor. By this means, both groups ensure a similar basal endocrine pattern related to lactation to investigate the acute effect of suckling on stress responsiveness. For mental stress challenging, we employed the Trier Social Stress Test (37), which was repeatedly found to be a potent psychosocial stressor (38–41).

Subjects and Methods

Subjects

Forty-three healthy lactating women (mean ± SEM age, 30.8 ± 0.7 yr) participated in the study. Subjects were 7.9 ± 0.2 wk postpartum

Abbreviations: AUC, Area under the curve; BF, breast-feeding; HD, holding infants without feeding; HPA, hypothalamic-pituitary-adrenal; VAS, visual analog scales.

(mean ± SEM) with 30.2% 6–7 wk, 37.2% 8 wk, and 32.6% 9–11 wk postpartum. Subjects were recruited by local advertisements for paid participation in a study of lactation and stress. Participants underwent a medical examination and a diagnostic interview before entering the study and they were considered eligible if they were free of chronic diseases, mental disorders, medication, smoking, and drug or alcohol abuse. All women had uncomplicated pregnancy, and they all delivered full-term, healthy infants by the vaginal route. They were exclusively breast-feeding every 3–4 h on demand by the infant. No subjects had resumed menses before the experimental session. Subjects were instructed to consume their normal food and drink and to abstain from caffeine, alcohol, and running or other strenuous activity during the 24 h before the experiment. The study protocol was approved by the ethics committee of the University of Trier, and all volunteers gave written informed consent for participation in the study.

Experimental protocol

Two weeks before the experiment subjects were instructed in writing either to breast-feed their infants 40 min before the appointment (100 min before the stress procedure) or in a private room in the laboratory 30 min before the stress test. The women were randomly assigned to the breast-feeding or holding group. Further, they were asked to bring somebody with them to care for the infant for about 100 min during the experiment. All subjects with their infants and the accompanying persons reported to the laboratory at 1400 h. On arrival, all participants had a catheter inserted in a brachial vein 45 min before the psychosocial stress procedure. In the following 15-min resting period, subjects completed questionnaires on trait anxiety and depression as well as mood and state anxiety (see below). After this, the first blood and saliva samples were collected at −30 min. According to the aforementioned randomly assigned groups, participants then were instructed either to breast-feed their infants (BF) or to hold their infants without feeding them (HD) during the following 15-min period. Because the mothers were breast-feeding their infants at intervals of 3–4 h and the experimental session lasted 150 min, both the women who fed their infants 40 min before arrival at the laboratory as well as the woman who were instructed so as to feed their infants during the experimental session were scheduled so as to avoid interfering with the mother-infant feeding interval. Breast-feeding 30 min before the start of the stress procedure ensured similar peripheral baseline levels for oxytocin and PRL in both groups immediately before stress. Thereafter, the mothers handed over their infants to the accompanying person who took care of the infant the following 100 min. A second blood and saliva sample was then obtained at −10 min. After additional completion of questionnaires measuring mood and state anxiety, subjects were given instructions about the stress test between −5 and −1 min, and a third blood and saliva sample was collected immediately before the stressor at −1 min. Thereafter, subjects were exposed to the Trier Social Stress Test (37), which primarily consists of an unprepared speech and mental arithmetic performed in front of an audience. Previous studies indicated that this stress protocol reliably induces a significant activation of the HPA axis, with 2- to 3-fold increases in free cortisol as well as cardiovascular and subjective responses indicative of moderate stress in healthy individuals (38–41). Due to the well known phenomenon that repeated exposure to stressful stimulation has been reported to result in rapid habituation of HPA axis responses, a cross-over study design with the same subjects being tested in each experimental situation was not possible. Including introduction to the test, subjects were confronted with the psychosocially stressful situation for a total of 15 min. Then, further blood and saliva samples were collected at 1 min after termination of the stressor, and subjects completed the questionnaires on mood and state anxiety again, followed by visual analog scales (VAS) for measuring the stressfulness of stress exposure. Additional blood samples were obtained 30 and 60 min after the stress test. Further saliva samples were collected 10, 20, 30, 45, and 60 min after cessation of stress.

Blood and saliva sampling

At the beginning of the experiment, blood samples were obtained for single measurements of basal 17β-E2 and progesterone levels. Additional blood samples were collected before and after the breast-feeding or holding period, as well as directly before starting the stress test for determining ACTH, cortisol, norepinephrine, epinephrine, oxytocin, and PRL. The remaining three blood samples were collected after cessation of stress as described above. Vasopressin was measured 1 min before and 1 min after the stressor. All blood samples were collected on ice in EDTA-coated tubes and centrifuged at 4 C at 5000 rpm for 5 min. Plasma samples were stored at −80 C until assay.

Saliva was collected using Salivette (Sarstedt, Rommelsdorf, Germany) collection devices immediately before and after the breast-feeding or holding period as well as directly before the stress. Six additional saliva samples were obtained after stress induction as reported above. After chewing for about 60 sec, devices were stored at −20 C until required for biochemical analysis. Before assaying for free cortisol, samples were thawed and spun at 3000 rpm for 5 min to obtain 0.5–1.0 ml clear saliva with low viscosity.

Biochemical analyses

ACTH and total plasma cortisol concentrations were determined with a commercial two-site chemiluminescence assay (Nichols Institute Diagnostics, Bad Nauheim, Germany). PRL, 17β-E2, and progesterone concentrations were measured employing a commercial ELISA kit (IBL, Hamburg, Germany). Norepinephrine and epinephrine concentrations were assayed by HPLC with electrochemical detection, as previously described (42). Oxytocin and vasopressin concentrations were measured in extracted plasma samples by highly sensitive and selective RIAs (for a detailed description, see Ref. 43). The free cortisol concentration in saliva was analyzed using a time-resolved immunoassay with fluorescence detection, as described previously (44). The limits of detection were 0.5 pg/ml for ACTH, 0.5 nmol/liter for saliva cortisol, 0.8 μg/dl for plasma cortisol, 10 pg/ml for norepinephrine, 10 pg/ml for epinephrine, 0.5 pg/ml for oxytocin, 0.5 pg/ml for vasopressin, 2 ng/ml for PRL, 16 pg/ml for E2, and 0.18 ng/ml for progesterone. Inter- and intraassay coefficients of variance were below 12% and 10%, respectively, for all analytes.

Psychological assessment

A mood questionnaire was employed that is especially suited for repeated measures within several minutes or hours (45). Twelve items are rated on a five-point scale, ranking from 1 = not at all to 5 = very strongly. Factor analyses revealed three scales, termed elevated *vs.* depressed mood, wakefulness *vs.* sleepiness, and calmness *vs.* restlessness. Depression and anxiety were assessed by the German versions of the Self-Rating Depression Scale (46) and the State-Trait Anxiety Inventory (47). Mood and state anxiety were assessed at three times: before the breast-feeding or holding period, between that period and the stressor, and after the psychosocial stress test. Fifteen VASs were employed for subjective ratings of perceived stressfulness of the test. VAS ratings were transformed into values between 0 = not at all to 100 = absolutely.

Statistical analyses

Two-way (group and time) ANOVAs for repeated measures were performed to reveal time effects, between-group differences, and the interaction of time and group for each variable of interest. All reported results were corrected by the Greenhouse-Geisser procedure where appropriate (reflected by the degrees of freedom with decimal values). Single time point data were compared using two-tailed *t* test. To determine where significant differences from baseline hormone levels in groups were occurring, *t* tests with Bonferroni corrections were applied. Pearson's correlations were computed for the areas under the individual response curves (AUC). AUCs were calculated using the trapezoid formula, aggregating the three blood and six saliva hormone levels, respectively, after the stress induction relative to the individual baseline concentration before stress. For all analyses, the level of significance was set at 0.05. All results shown are the mean ± SEM.

Results

Physical and psychometric characteristics of the subjects are presented in Table 1. As described in *Subjects and Methods*, women were randomly assigned to either the BF or HD

group according to the 15-min feeding or holding period 30 min before stress exposure. BF and HD did not differ significantly in age and weeks postpartum. There were no differences among groups in basal E2 and progesterone levels. Trait anxiety and depression scores were similar for both groups and were within the general population normal range.

Endocrine responses to breast-feeding vs. holding the infant

Hormone levels of BF and HD before and after breast-feeding or holding the infants are shown in Table 2. There were no differences in basal hormone levels before the breast-feeding or holding period between both groups of lactating women (all P = NS). Only PRL levels were higher in the holding group [$t(33.1) = 2.91$; $P = 0.006$] based on the shorter time interval since their last breast-feeding period (60 min in HD *vs.* 100 min in BF before the experiment). There were significant decreases in ACTH [$F(1,41) = 21.56$; $P < 0.001$], salivary free cortisol [$F(1,41) = 8.36$; $P = 0.006$], and total plasma cortisol [$F(1,41) = 31.95$; $P < 0.001$] in the combined group of BF and HD. As expected, breast-feeding compared with holding caused significant elevations of PRL concentrations [time effect: $F(1,41) = 6.67$; $P = 0.013$; group by time effect: $F(1,41) = 12.20$; $P = 0.001$]. No significant changes could be observed in norepinephrine, epinephrine, and oxytocin levels in the total group. All hormone levels of BF and HD were similar 10 min after the feeding or holding period (all P = NS).

Endocrine responses to psychosocial stress

There was a significant ACTH stress response in the total group during the psychosocial stress test [$F(1.4,58.3) = 25.45$; $P < 0.001$] with a peak 1 min after cessation of the stressor. Similarly, salivary free cortisol and total plasma cortisol levels were significantly increased during the stress [free cortisol: $F(2.4,96.7) = 13.65$; $P < 0.001$; total cortisol: $F(3,123) = 12.61$; $P < 0.001$]. As depicted in Fig. 1B, cortisol levels were highest 20 min after stress exposure with the expected time lag compared with the peak for ACTH. Correlations between ACTH and cortisol stress responses (AUC) were significant, with r = 0.51 (ACTH *vs.* free cortisol, $P < 0.001$) and r = 0.44 (ACTH *vs.* total cortisol, $P = 0.003$).

There were also significant norepinephrine and epinephrine responses during the stress test in both groups of women [$F(2.5,104.1) = 22.86$; $P < 0.001$ and $F(1.3,34.9) = 7.07$; $P = 0.007$, respectively]. Norepinephrine and epinephrine concentrations peaked 1 min after cessation of the stressor, with, as for ACTH and cortisol, decreasing hormone levels thereafter. PRL levels were significantly decreased after the stressor in the combined group of BF and HD [$F(1.4,55.6) = 18.44$; $P < 0.001$]. No significant changes in oxytocin responses to stress were observed. Analyses of pre-post stress measurements of vasopressin levels also revealed no significant changes.

As shown in Fig. 1, no significant differences in baseline ACTH, salivary free cortisol, total plasma cortisol, norepinephrine, epinephrine, oxytocin, and PRL levels were observed between BF and HD 1 min before stress (all P = NS). Although PRL baselines may visually appear to differ between groups, *post-hoc* analyses indicated no significant difference. However, there was a main effect of subject group on cortisol [free cortisol: $F(1,41) = 4.15$; $P = 0.048$; total cortisol: $F(1,41) = 7.75$; $P = 0.008$], with blunted cortisol levels in BF compared with HD. The mean absolute increases in salivary cortisol and plasma cortisol were 6.0 nmol/liter and 3.0 μg/dl, respectively, in HD and 2.4 nmol/liter and 0.7 μg/dl, respectively, in BF. HD showed significant higher free cortisol levels at 10, 20, 30, and 45 min as well as higher plasma cortisol levels 1 and 30 min after cessation of stress compared with baseline (all $P < 0.001$), whereas in BF no cortisol increases after stress compared with baseline concentrations occurred (all P = NS). Breast-feeding women demonstrated a significant suppression of total plasma cortisol responses [group by time effect: $F(3,123) = 5.09$; $P = 0.002$] and a trend toward suppressed salivary free cortisol levels [group by time effect: $F(2.4,96.7) = 2.65$; $P = 0.067$] compared with the holding group. Differences in ACTH levels between both groups did not reach statistical significance [$F(3,123) = 1.87$; $P = 0.138$].

TABLE 1. General characteristics of women who were breastfeeding (BF) or holding (HD) their infants

	BF (n = 20)	HD (n = 23)
Age (yr)	31.4 ± 0.9	30.2 ± 1.0
Wk postpartum	8.3 ± 0.3	7.5 ± 0.3
E2 (pg/ml)	81.6 ± 11.4	76.0 ± 5.0
Progesterone (ng/ml)	0.39 ± 0.28	0.09 ± 0.02
Trait anxiety	34.1 ± 2.1	35.7 ± 1.7
Depression	33.3 ± 1.2	35.1 ± 1.6

Values are the mean ± SEM.

TABLE 2. Hormone levels in breastfeeding (BF) and holding (HD) women before (PRE) and after (POST) the feeding or holding period

	BF		HD		P
	PRE	POST	PRE	POST	
ACTH (pg/ml)	30.3 ± 3.4	25.9 ± 3.2	25.7 ± 1.8	23.2 ± 1.7	<0.001[a]
Saliva cortisol (nmol/l)	8.2 ± 0.5	7.1 ± 0.5	7.9 ± 0.9	7.2 ± 0.7	0.006[a]
Plasma cortisol (μg/dl)	7.9 ± 0.4	6.2 ± 0.4	7.1 ± 0.5	6.3 ± 0.4	<0.001[a]
Norepinephrine (pg/ml)	240.9 ± 23.4	255.9 ± 32.1	246.3 ± 15.3	244.5 ± 18.4	NS
Epinephrine (pg/ml)	20.7 ± 5.4	18.7 ± 3.4	21.8 ± 2.5	24.5 ± 4.0	NS
Oxytocin (pg/ml)	6.6 ± 1.1	10.3 ± 1.7	9.6 ± 2.1	9.7 ± 2.1	NS
PRL (ng/ml)	15.7 ± 2.3	36.1 ± 7.6	29.8 ± 4.3	26.8 ± 4.3	0.013[a] 0.001[b]

Values are the mean ± SEM.
[a] Time effect.
[b] Group by time effect.

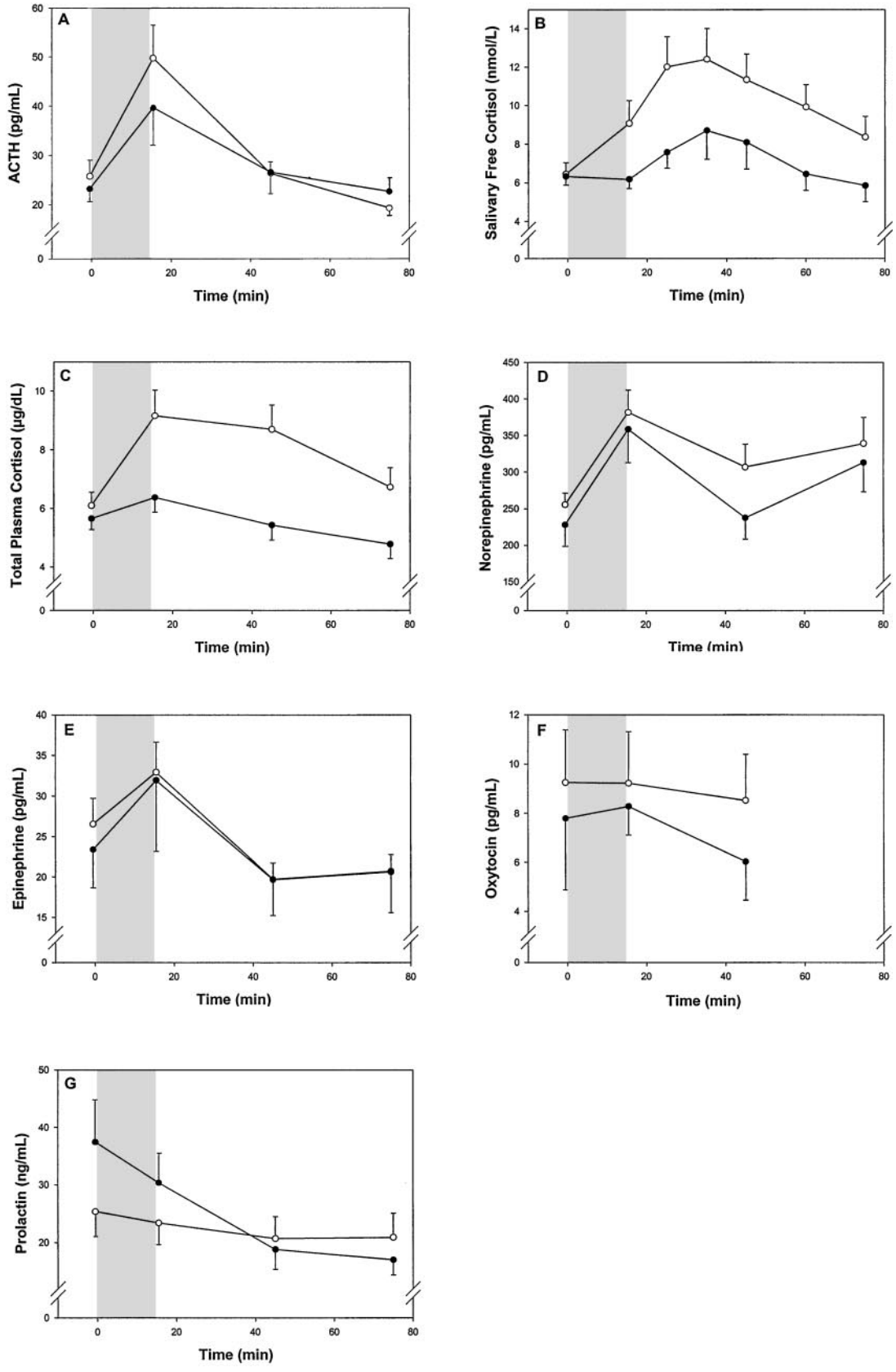

FIG. 1. Mean ACTH (A), salivary free cortisol (B), total plasma cortisol (C), norepinephrine (D), epinephrine (E), oxytocin (F), and PRL (G) levels (±SEM) before and after exposure to the Trier Social Stress Test in lactating women after breast-feeding (●) and holding (○) their infants. The *shaded areas* indicate the period of stress exposure.

PRL levels steadily decreased after cessation of psychosocial stress in the breast-feeding women, with an interaction between subject group and PRL concentration after stress [$F(1.4,55.6) = 7.31$; $P = 0.005$]. There were no differences between BF and HD in patterns of norepinephrine, epinephrine, and oxytocin changes during the stress test (all P = NS).

Psychometric parameters

Mood and anxiety were assessed before the breast-feeding and holding periods, respectively, as well as before and after the following psychosocial stress test. There was a main effect of subject group on mood [$F(1,41) = 4.87$; $P = 0.033$], with elevated mood in breast-feeding women. In the scale calmness *vs.* restlessness, feeding increased calmness and composure, whereas holding the infant resulted in elevated tension and feelings of unrest [group by time effect: $F(1,41) = 6.41$; $P = 0.015$]. Scores in the scale wakefulness *vs.* sleepiness remained unchanged.

Stress exposure worsened mood in both the BF and HD groups [$F(1,41) = 15.42$; $P < 0.001$]. Similarly, the scale calmness *vs.* restlessness revealed a significant increase in tension and feelings of unrest in response to stress in the total group [$F(1,41) = 11.61$; $P = 0.001$]. Again, no changes in the scale wakefulness *vs.* sleepiness were observed.

Both breast-feeding as well as holding the infant led to significantly decreased state anxiety responses of BF and HD [$F(1,41) = 4.21$; $P = 0.047$], with a stronger decrease following breast-feeding (BF: before, 36.3 ± 1.8; after, 33.5 ± 1.5; HD: before, 37.1 ± 1.5; after, 36.9 ± 1.5) that did not reach statistical significance [group by time effect: $F(1,41) = 2.90$; $P = 0.096$].

In contrast, state anxiety significantly increased in response to the psychosocial stress test in the combined group [$F(1,41) = 21.08$; $P < 0.001$] with no significant difference among groups. No group differences in VAS ratings of perceived stressfulness were observed.

Discussion

This is the first study to investigate the longer-term (holding group) as well as the short-term (breast-feeding group) effects of suckling on endocrine and behavioral responses to psychosocial stress in lactating women. The Trier Social Stress Test activated the HPA axis as well as the sympathetic-adrenal-medullary system and worsened mood, calmness, and anxiety in the total group. The present results show that salivary free cortisol and total plasma cortisol responses to the stress protocol were markedly blunted in lactating women who were breast-feeding 30 min before stress exposure compared with those who were only holding their infants 30 min before stress (time interval to the last breast-feeding period before stress induction, 100 min). However, the reduction in ACTH stress responses in breast-feeding women did not reach statistical significance.

In lactating rats as well as in lactating women, neuroendocrine responses to stress are known to be reduced, and lactogenic peptides released not only into the blood but also within the brain during suckling may modulate the activity of the HPA axis (4–14, 16, 17, 36). Besides the potential inhibitory effects of oxytocin at the adrenal level described by Legros and associates (27) in healthy men, inhibition of the basal and stress-induced activity of the HPA axis by intracerebral oxytocin has been demonstrated in both male (48) and female rats (36, 49). Oxytocin release in the hypothalamic paraventricular nucleus and in extrahypothalamic brain regions (*e.g.* septum) has not only been shown during suckling in both lactating rats (34, 35) and lactating sheep (50), but also in response to an emotional stressor in male rats (51). Because oxytocin was at baseline in both groups at the start of the stress test, and no changes in oxytocin concentrations after stress were observed, plasma oxytocin levels do not seem to mediate the suppression of cortisol stress responses at the adrenal level in the present study. The inhibitory effect of oxytocin on HPA axis responsiveness points to a more central change and could, in fact, be localized in the paraventricular nucleus and the septum, as shown in rats (48).

Furthermore, PRL, significantly released into the blood during suckling, is known to inhibit HPA axis reactivity (8). Interestingly, PRL passes the blood-brain barrier via selective uptake by choroid plexus receptors and exerts central nervous effects (52). Within this context, it is noteworthy to mention that PRL receptors within various hypothalamic and limbic brain regions are up-regulated during lactation (53), although intracerebral release of PRL, *e.g.* in response to suckling, has not been demonstrated.

In the present study a strong decline in PRL levels occurred after cessation of the stressor in the breast-feeding compared with the holding group. Interestingly, a gradual decrease in plasma PRL levels after stress has also been observed in the lactating rat (12, 16) and in lactating women (17), whereas PRL levels increased in nonlactating animals and humans. In light of these findings, it may be speculated that the prolonged decrease observed in the present study might not only be due to the cessation of suckling, but could also represent a specific response to the mental stressor. Only recently have animal studies revealed support for a prolonged decline in PRL levels after psychological stress in lactating rats (7, 54). Further animal and human studies are needed to determine the role of PRL on stress responsiveness during lactation. Thus, it is possible that the attenuated HPA axis response to the psychosocial stress protocol found in lactating women who were breast-feeding their infants before stress exposure is due to the inhibitory effect of both oxytocin and PRL released into the blood and into distinct brain regions.

Moreover, evidence from animal research has suggested that the increased pulsatile secretion of corticosterone reflecting the effect of suckling during lactation (55, 56) might lead to increased negative feedback on the HPA axis (4, 5). Adaptations of HPA axis activity in the peripartum period occur at all brain levels, including reduction in the activity of hypothalamic CRF neurons (57), reduced excitatory inputs to these neurons (58, 59), and reduced CRF binding at the adenohypophysis (3) to name a few. These adaptations are reversible upon weaning (7).

There were no differences between groups in levels of ACTH and cortisol before the breast-feeding or holding period 30 min before the stress procedure and no differences in baseline levels 1 min before the onset of the stressor. It is important to note that not only breast-feeding women but

also women who were only holding their infant showed significant decreases in ACTH, free cortisol, and total plasma cortisol before stress during the suckling and holding periods, respectively. This might be interpreted as due to a decrease in HPA axis hormones after the implantation of the venous catheter 15 min before the breast-feeding or holding period. A further interpretation might be a suckling-related classical conditioning of the HPA axis response in lactating women under the close presence of the infant. In addition, both breast-feeding as well as holding the infant led to significantly decreased anxiety, whereas mood and calmness improved only in the breast-feeding group. However, stress exposure worsened mood and led to an increase in tension, restlessness, and anxiety to a similar extent in both groups of lactating women. These findings agree with data from animal research in which lactating rats showed normal behavioral reactivity to a psychological stress with no statistically significant activation of the HPA axis (4, 6, 7).

The present findings are in accord with a previous report by Altemus *et al.* (17) showing decreasing PRL concentrations and increasing ACTH, cortisol, norepinephrine, and epinephrine concentrations in women who were breast-feeding their infants 60 min before the start of a physical stressor (treadmill exercise). Also, the researchers found no changes in oxytocin levels. Whereas Altemus and associates (17) investigated the general effect of lactation on endocrine stress responsiveness comparing lactating and nonlactating women, the present study was undertaken to determine the acute effects of suckling (short-term *vs.* longer-term effects) on endocrine and behavioral responses to psychosocial stress during lactation. In this respect, the present study might extend the findings of Altemus and associates (17) to the sensitive time period for suckling-induced endocrine stress hyporesponsiveness in humans.

In conclusion, the salient result from the present study is that suckling is suggested to exert a short-term suppression of the HPA axis response to mental stress. Thus, lactation in women does not result in a general restraint of the HPA axis response to psychosocial stress. Whereas in animal research it is generally believed that HPA axis hyporesponsiveness can be observed during the whole period of lactation (7), the present data strongly suggest that the blunted adrenocortical stress response in women seems to be counterbalanced if the acute psychosocial stressor occurs 2 h after suckling. The neurobiological mechanisms mediating reduced stress responsivity in lactating women remain to be determined. From an evolutionary-biological point of view, short-term attenuation of cortisol stress responses appear to have several protective functions (17), such as isolating the mother from distracting stimuli of the environment, facilitating the immune system of lactating women, protecting the infant against stress-related high cortisol concentrations in the milk, and preventing inhibition of lactation caused by stress. Future work is needed to determine the short- and long-term effects of breast-feeding in humans in relation to different kinds of stressors.

Acknowledgments

We are grateful to Jutta Bennecke, Mathias Boeck, Ulrike Bucerius, Katrin Burmeister, Helen Esser, Jens Langer, Patrick Mayer, Eva Poll, Miriam Seng, and Silke Welter for excellent research assistance, and to our volunteers for participating in this study. We thank Urs Nater and Marc Simon for preparing the manuscript.

Received March 7, 2001. Accepted June 15, 2001.

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