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# Mid-pregnancy androgen levels are negatively associated with breastfeeding

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**Abstract**

*Objective.* Breastfeeding depends on endocrine changes during pregnancy. The association between gestational hormones and lactation has been sparsely investigated. Previously, androgens were used for lactation inhibition. We investigated a possible association between second trimester maternal androgen levels and breastfeeding. *Design.* Prospective observational study. *Setting.* University hospital setting. *Population.* Women from a random sample of pregnancies (n = 63) and from a group with an increased risk for giving birth to a small-for-gestational age newborn (n = 118) were included. All participants had singleton pregnancies and one or two previous births. *Methods.* Maternal androgen levels were measured in gestational week 25. The association with reported breastfeeding was explored by univariate and multivariate linear regression analyses. Analyses were adjusted for factors known to be associated with breastfeeding. *Main outcome measures.* Breastfeeding at six weeks, three months, and six months postpartum. *Results.* In the random group, breastfeeding at three and six months was negatively associated with maternal testosterone, androstendione, and free testosterone index levels. After correction for maternal age, education and smoking, breastfeeding at both three and six months was negatively associated with the free testosterone index. In the group of women with an increased risk for giving birth to a small-for-gestational age newborn, breastfeeding at six weeks and three months was associated negatively with maternal dehydroepiandrosterone and this association persisted after correction for maternal age, education, and smoking. *Conclusions.* Maternal androgen levels in mid-pregnancy are negatively associated with breastfeeding.

**Key words:** *Breastfeeding, lactation, androgens*

## Introduction

Breastfeeding is the single factor that most favorably influences infant death rates worldwide (1). According to WHO, women are recommended to exclusively breastfeed for six months, with subsequent introduction of solid and complementary food. Thereafter breastfeeding should be combined with complementary and solid food for another 6–18 months (1).

Breastfeeding protects the infant against infectious diseases and is held to reduce the incidence of diabetes mellitus (type 1 and type 2), obesity, asthma, and a delay in the onset of atopic eczema in both teenagers and adults (2). Breastfeeding may even be associated with increased intelligence in the offspring (2,3). However, in several reports, a recent large cluster, randomized trial questions many of these positive effects shown in a number of observational studies (4–7).

Breastfeeding is influenced by many factors. Increasing maternal age and education are positively associated with breastfeeding, while high socioeconomic status and marriage show weaker positive

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associations (8). On the other hand, maternal smoking and infant health problems are negatively associated with breastfeeding (8).

Major endocrine changes occur during pregnancy and puerperium. These changes are crucial for mammary gland development and lactation (9). However, the effects of androgens have been sparsely focused. Before dopamine agonists were introduced, androgens were used to suppress lactation (10–12). Pregnant women with polycystic ovary syndrome (PCOS) have elevated androgen levels (13). Further, PCOS mothers seem to breastfeed less than controls, and breastfeeding is negatively associated with dehydroepiandrosterone sulfate (DHEAS) among these women in late pregnancy (14).

We hypothesized that breastfeeding may be associated with maternal androgen levels. Previously we have shown that birthweight is negatively associated with maternal testosterone levels (15). Accordingly, we investigated our hypothesis in two cohorts of parous pregnant women. They were a random sample of pregnancies and a group of women with an increased risk of giving birth to a small-for-gestational age (SGA) newborn.

## Material and methods

The study was based on the Norwegian part of a study performed in 1986–1988 (16). Briefly, Caucasian women with singleton pregnancies and one or two previous childbirths were included. Among these women, two groups were characterized in detail throughout pregnancy, and their children followed quite comprehensively until five years of age. One group consisted of a 10% random sample of pregnant women (n = 561), while the second group included women with at least one definite risk factor for giving birth to an SGA child. The risk criteria were a history of a previous low birthweight newborn or perinatal death, cigarette smoking at conception, pre-pregnancy weight less than 50 kg, and chronic renal disorder or hypertension (n = 1,384).

In 2001–02, adolescent girls born in the late 1980s and still living at a reasonable distance from the university hospitals in Bergen and Trondheim were invited to a follow-up examination. Among a total of 523 girls who made up this potential study group, 146 were excluded because they were not born at term (before pregnancy week 37 or after week 42) or because exact information about gestational age was missing. Of the remaining 377 girls, some had died (n = 5), were reluctant to participate (n = 8), gave some other reasons for non-participation (n = 26), or did not respond (n = 76). Accordingly, 262 out of 372 girls attended (70.4 %).

Our two study cohorts consisted of the mothers of these teenage daughters. In 181 mothers, maternal serum from gestational week 25 was available concomitant with information about breastfeeding from at least two of three points of time (i.e. six weeks, three and/or six months postpartum). Of these 181 women, 63 stemmed from the original random sample of pregnant women and 118 from the increased risk group.

This study was approved by the Regional Committee for Medical Research Ethics and the Norwegian Data Inspectorate.

At study inclusion in gestational week 17, maternal age (years), completed education (less than 9 years, level = 1; 9 years, level = 2; 10–11 years, level = 3; 12 years, level = 4; more than 12 years, non-university level, level = 5; more than 12 years university level, level = 6), family status (single = 1, co-habitating = 2, married = 3), and pre-pregnancy body mass index (BMI, $kg/m^2$) were registered. In gestational week 25, serum samples were drawn and stored at –80°C for later endocrine analyses, done after an average storage of 20 years.

Smoking at conception was registered (no; value = 0, yes; value = 1), while gestational age (days) and offspring birthweight (grams) and length (cm) were registered at birth. Breastfeeding was registered at regular health controls when the infants were six weeks, three months, and six months old. Mothers were classified into supplementary feeding only (value = 0), breast + supplementary feeding (value = 1), and breastfeeding only (value = 2).

Dehydroepiandrosterone (DHEA), DHEAS, testosterone, and sex hormone binding globulin (SHBG) were measured by ELISA technique using kits from DRG (DRG Instruments GmbH, Marburg, Germany). Androstendione was measured by ELISA technique with kits from DPC (Diagnostic Products Corporation, Los Angeles, CA, USA). Single estimations were used for all analyses. Free testosterone index (FTI) was calculated as (total testosterone/SHBG) x 100.

All statistical analyses were conducted using the statistical software SPSS for Windows, Release 15.0.1, Copyright © SPSS Inc., 1989–2007 (Chicago, IL, USA). Values are given as means and standard deviations. Differences between groups were investigated with Mann-Whitney test for unrelated samples. A $p$-value $\leq 0.05$ was considered significant. We made no adjustments for multiple comparisons.

The associations between breastfeeding and other variables were investigated by univariate and multivariate linear regression analyses. Mean values with 95%

Table I. Characteristics of the study groups.

| | Random group (n = 63) | Increased risk group (n = 118) | |
|---|---|---|---|
| Variable | Mean ± SD | Mean ± SD | *p*-Value |
| Maternal age (years) | 29.1 ± 4.9 | 28.6 ± 4.0 | 0.41 |
| Maternal BMI ($kg/m^2$) | 21.8 ± 2.6 | 20.5 ± 2.6 | 0.001 |
| Gestational age (days) | 282 ± 7 | 283 ± 7 | 0.65 |
| Birth weight (grams) | 3,539 ± 507 | 3,501 ± 447 | 0.46 |
| DHEA (nmol/L)[1] | 32.6 ± 16.6 | 31.7 ± 17.0 | 0.65 |
| DHEAS (μmol/L)[1] | 3.15 ± 1.57 | 3.22 ± 1.69 | 0.97 |
| Androstendione (nmol/L)[1] | 8.22 ± 2.45 | 9.30 ± 3.76 | 0.13 |
| Testosterone (nmol/L)[1] | 2.15 ± 0.52 | 2.27 ± 0.79 | 0.51 |
| Free testosterone index[1] | 0.68 ± 0.26 | 0.74 ± 0.39 | 0.67 |

[1]Measured in gestational week 25.
Note: SD, standard deviation; BMI, body mass index; DHEA, dehydroepiandrosterone; DHEAS, dehydroepiandrosterone sulfate.

confidence intervals were used and a $p$-value ≤ 0.05 was considered significant.

## Results

Compared to the random group, women in the increased risk group had lower pre-pregnancy BMI (20.5 ± 2.6 vs. 21.8 ± 2.6 $kg/m^2$; $p = 0.001$) (Table I). Maternal DHEA, DHEAS, androstendione, testosterone, and free testosterone index in gestational week 25 were the same for both groups. We found no differences in maternal age, gestational age at delivery, and birthweight of the offspring between study groups. This was also the case for reported breastfeeding and supplementary feeding (Table II).

In univariate analyses of mothers in the random group, breastfeeding was negatively associated with smoking at six weeks. At three months, breastfeeding was positively associated with maternal age and education, and negatively with smoking, androstendione, testosterone, and FTI. Finally, breastfeeding was positively associated with maternal education and negatively associated with smoking, androstendione, testosterone, and FTI at six months of age (Table III).

In multivariate linear regression, breastfeeding at three months was associated negatively with smoking and FTI, while breastfeeding at six months did so with FTI (Table IV). In the multivariable analyses, breastfeeding at all ages of the newborn were unrelated to maternal age and education. Further, breastfeeding was unrelated to maternal BMI, gestational age at birth, birthweight, and gender of the child in univariate and multivariate as well as linear regression analyses (data not shown).

Univariate regression analyses of women in the increased risk group showed that breastfeeding was positively associated with maternal education at six weeks and three months, while it was negatively associated with DHEA. At six months, breastfeeding was unrelated to any of the variables under study.

In multivariate linear regression analyses, breastfeeding at six weeks and three months associated negatively with maternal pregnancy levels of DHEA. Again, breastfeeding throughout the first six months of life was unrelated to maternal BMI, gestational age at birth, birthweight, and gender of the child, in univariate and multivariate as well as linear regression analyses (data not shown).

In the random group of mothers, BMI was positively correlated to FTI, while smoking was so to androstenedione and testosterone (Table V). There were several significant correlations in the high risk group. Thus, maternal age correlated negatively to testosterone, BMI positively to DHEAS, and education negatively to DHEAS, DHEA, and androstenedione. Also, smoking was positively associated with DHEA and androstenedione. On the other hand, status as married correlated negatively with DHEAS and DHEA (data not shown), while birthweight was negatively associated with DHEA (Table V).

Table II. Breastfeeding according to study group.

| | Week 6 | | 3 months | | 6 months | |
|---|---|---|---|---|---|---|
| Type of infant feeding | Increased risk group[1] | Random group | Increased risk group | Random group | Increased risk group | Random group |
| Breastfeeding only[2] | 93 (79%) | 52 (83%) | 78 (66%) | 45 (71%) | 1 (1%) | 0 (0%) |
| Breast + supplementary feeding | 18 (15%) | 8 (13%) | 20 (17%) | 5 (8%) | 65 (55%) | 41 (65%) |
| Supplemenatry feeding only | 7 (6%) | 3 (5%) | 20 (17%) | 13 (21%) | 52 (44%) | 22 (35%) |

[1]Two-sided Pearson Chi-square tests were non-significant ($p > 0.20$) for difference between study groups at all three time points.
[2]Number (proportion).

Table III. Univariate linear regression analyses of breastfeeding in women according to study group.

| | | Breastfeeding[1] | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | 6 weeks | | | 3 months | | 6 months | |
| Variable | Group | *n* | B (95% CI) | *p*-Value | B (95% CI) | *p*-Value | B (95% CI) | *p*-Value |
| Maternal age (years) | Random | 63 | 0.02 (–0.01; 0.04) | 0.22 | 0.05 (0.01; 0.09) | 0.012 | 0.01 (–0.02; 0.03) | 0.46 |
| | Increased risk | 118 | –0.01 (–0.03; 0.02) | 0.59 | 0.03 (–0.01; 0.06) | 0.13 | 0.01 (–0.02; 0.03) | 0.69 |
| Maternal BMI ($kg/m^2$) | Random | 63 | –0.01 (–0.06; 0.04) | 0.64 | –0.05 (–0.13; 0.04) | 0.26 | –0.02 (–0.06; 0.03) | 0.50 |
| | Increased risk | 118 | 0.00 (–0.04; 0.04) | 0.86 | –0.01 (–0.07; 0.04) | 0.70 | –0.02 (–0.06; 0.02) | 0.29 |
| Maternal education (level 1–6)[2] | Random | 63 | 0.05 (–0.06; 0.17) | 0.36 | 0.18 (0.01; 0.36) | 0.045 | 0.15 (0.04; 0.25) | 0.006 |
| | Increased risk | 117 | 0.10 (0.01; 0.19) | 0.038 | 0.12 (0.00; 0.24) | 0.050 | 0.06 (–0.02; 0.14) | 0.14 |
| Smoking (yes/no) | Random | 60 | –0.36 (–0.66; –0.07) | 0.018 | –0.94 (–1.36; –0.51) | <0.0005 | –0.38 (–0.64; –0.11) | 0.006 |
| | Increased risk | 116 | –0.20 (–0.403; 0.01) | 0.065 | –0.22 (–0.51; 0.06) | 0.12 | –0.12 (–0.31; 0.08) | 0.23 |
| Gestational age (days) | Random | 63 | –0.00 (–0.02; 0.02) | 0.78 | –0.01 (–0.04; 0.02) | 0.33 | 0.00 (–0.02; 0.02) | 0.97 |
| | Increased risk | 118 | 0.00 (–0.02; 0.02) | 0.98 | –0.01 (–0.03; 0.01) | 0.22 | –0.01 (–0.02; 0.01) | 0.25 |
| Androstendione (nmol/L) | Random | 63 | –0.05 (–0.10; 0.00) | 0.071 | –0.14 (–0.22; –0.06) | 0.001 | –0.07 (–0.12; –0.02) | 0.005 |
| | Increased risk | 118 | –0.02 (–0.04; 0.01) | 0.28 | –0.02 (–0.06; 0.02) | 0.27 | –0.01 (–0.04; 0.01) | 0.27 |
| DHEA (nmol/L) | Random | 63 | 0.00 (–0.01; 0.01) | 0.42 | –0.00 (–0.02; 0.01) | 0.58 | 0.00 (–0.01; 0.01) | 0.82 |
| | Increased risk | 118 | –0.01 (–0.01; 0.00) | 0.007 | –0.01 (–0.02; 0.00) | 0.003 | –0.01 (–0.01; 0.00) | 0.078 |
| DHEAS (μmol/L) | Random | 63 | 0.03 (–0.06; 0.11) | 0.52 | –0.03 (–0.16; 0.11) | 0.69 | 0.00 (–0.08; 0.08) | 0.95 |
| | Increased risk | 118 | –0.02 (–0.08; 0.05) | 0.61 | –0.04 (–0.12; 0.05) | 0.40 | –0.04 (–0.10; 0.01) | 0.13 |
| Testosterone (nmol/L) | Random | 63 | –0.15 (–0.40; 0.10) | 0.23 | –0.62 (–0.98; –0.25) | 0.001 | –0.37 (–0.58; –0.15) | 0.001 |
| | Increased risk | 118 | 0.00 (–0.13; 0.14) | 0.96 | –0.04 (–0.22; 0.14) | 0.64 | –0.04 (–0.16; 0.08) | 0.50 |
| Free testosterone index | Random | 63 | 0.10 (–0.42; 0.62) | 0.70 | –0.97 (–1.74; –0.19) | 0.016 | –0.618 (–1.07; –0.17) | 0.008 |
| | Increased risk | 118 | 0.05 (–0.22; 0.31) | 0.73 | 0.21 (–0.15; 0.57) | 0.26 | 0.00 (–0.24; 0.25) | 0.97 |

[1]Supplementary feeding only (value = 0), breast + supplementary feeding (value = 1), and breastfeeding only (value = 2).
[2]Education <9 years (level = 1), 9 years (level = 2), 10–11 years (level = 3), 12 years (level = 4), >12 years, non-university level (level = 5), and >12 years, university level (level = 6).
Note: CI, confidence interval; BMI, body mass index; DHEA, dehydroepiandrosterone; DHEAS, dehydroepiandrosterone sulfate; B, regression coefficient.

## Discussion

To our knowledge, this is but the second study that has explored the possible association between maternal gestational androgen levels and breastfeeding, and the first that has employed a random sample of pregnant women. In accordance with our overall hypothesis, we found that breastfeeding among women in both our study groups was negatively associated with maternal androgen levels in the second trimester of pregnancy. In both groups of women, the effect was essentially unaffected by adjustment for factors known to influence breastfeeding such as maternal age, education, and smoking (8).

Theoretically, if there is a causal relation, at least three mechanisms may explain the observed association between maternal gestational androgen levels and ensuing breastfeeding. High androgen levels during pregnancy may inhibit transformation of the breast in to the lactating state. Postpartum androgen levels, which may associate with gestational levels, may inhibit lactation. Third, it is also possible that androgens have psychological effects making women less dedicated to breastfeeding. A combination of these mechanisms is a fourth possibility.

Previous Norwegian data have shown that breastfeeding was associated positively with maternal age, education, marital status, degree of urbanization, number of children, and birthweight, and negatively with smoking (17). Hence, we explored the association between variables known to affect breastfeeding by performing Pearson's correlation tests between androgens and these variables. We found that many of the variables known to correlate with breastfeeding also did so with androgens. Earlier, we have reported some of these associations, such as the negative ones between mid-pregnancy androgens and maternal age, and offspring birthweight (15,18). However, we also

Table IV. Multivariate linear regression analyses of breastfeeding in women according to study group.

| | | Breastfeeding[1] | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | 6 weeks | | 3 months | | 6 months | |
| Variable | Group | $n^2$ | B (95% CI) | *p*-Value | B (95% CI) | *p*-Value | B (95% CI) | *p*-Value |
| Maternal age (years) | Random | 60 | 0.01 (–0.02; 0.04) | 0.58 | 0.02 (–0.02; 0.06) | 0.34 | –0.01 (–0.03; 0.02) | 0.64 |
| | Increased risk | 116 | –0.02 (–0.05; 0.01) | 0.11 | 0.02 (–0.02; 0.05) | 0.40 | 0.00 (–0.03; 0.02) | 0.79 |
| Maternal education (level 1–6)[3] | Random | 60 | –0.01 (–0.14; 0.13) | 0.93 | 0.01 (–0.17; 0.20) | 0.89 | 0.10 (–0.02; 0.21) | 0.96 |
| | Increased risk | 116 | 0.07 (–0.02; 0.17) | 0.13 | 0.07 (–0.06; 0.20) | 0.29 | 0.04 (–0.05; 0.13) | 0.40 |
| Smoking (yes/no) | Random | 60 | –0.35 (–0.73; 0.03) | 0.072 | –0.74 (–1.26; –0.23) | 0.006 | –0.21 (–0.52, 0.10) | 0.18 |
| | Increased risk | 116 | –0.13 (0.35; 0.09) | 0.24 | –0.05 (–0.35; 0.25) | 0.74 | –0.06 (–0.27; 0.14) | 0.54 |
| DHEA (nmol/L) | Random | 60 | 0.00 (–0.01; 0.01) | 0.60 | 0.00 (–0.01; 0.01) | 0.93 | 0.00 (–0.01; 0.01) | 0.70 |
| | Increased risk | 116 | –0.01 (–0.01; 0.00) | 0.022 | –0.01 (–0.02; 0.00) | 0.018 | –0.00 (–0.01; 0.00) | 0.15 |
| FTI | Random | 60 | 0.17 (–0.42; 0.75) | 0.58 | –0.83 (–1.63; –0.04) | 0.040 | –0.65 (–1.13; –0.17) | 0.009 |
| | Increased risk | 116 | –0.01 (–0.27; 0.26) | 0.97 | 0.22 (–0.14; 0.58) | 0.23 | –0.01 (–0.26; 0.25) | 0.97 |

[1]Supplementary feeding only (value = 0), breast + supplementary feeding (value = 1), and breastfeeding only (value = 2).
[2]Data missing for three women in the random group and two women in the high risk group.
[3]Education <9 years (level = 1), 9 years (level = 2), 10–11 years (level = 3), 12 years (level = 4), >12 years, non-university level (level = 5), and >12 years, university level (level = 6).
Note: CI, confidence interval; DHEA, dehydroepiandrosterone; FTI, free testosterone index; B, regression coefficient.

found previously unreported associations between maternal androgens and BMI, education, and smoking. It is noteworthy that the association between breastfeeding and androgens persisted in multivariable analyses, while it disappeared for all other variables except smoking.

In women who abuse androgenic steroids, and in female to male transsexuals who were treated with androgens, atrophy of the breast was observed (19,20). It seems that one physiological role of testosterone is to limit the estrogenic stimulation of the breast (21,22). Before dopamine agonists became available for lactation inhibition, testosterone was used for this purpose (10–12). Further, we have recently reported that breastfeeding rates were lower among pregnant women with PCOS and associated negatively with DHEAS levels in the third trimester (14). In fact, it was this observation that prompted us to perform the present study.

The present observations are further supported by studies of women with hypertensive disorders in pregnancy who have difficulties with lactation (23). Since women with preeclampsia have elevated androgen levels both in the second and the third trimesters of pregnancy (24–26), we suggest that our observed association between maternal gestational androgen levels and breastfeeding is not just a spurious one, but may represent a possibly true relation. On the other hand, we found no elevated androgens levels in women at risk of giving birth to an SGA child. This may explain why breastfeeding rates were equal between our two study cohorts.

We would like to emphasize that the study was done in a country where breastfeeding rates are high (17). In the present study, only 5–6% of women did not breastfeed at six weeks after delivery and that 79–83 and 55–65% were still breastfeeding at three and six months, respectively. Since rather few women apparently refused to breastfeed their child initially, we hold that breastfeeding rates at six weeks were quite reliable. This, in turn, reflects the ability to breastfeed, while breastfeeding at three and six months, in addition to a failure to initiate breastfeeding, may reflect either the motivation to keep up with it or the physical ability to breastfeed. These assumptions are supported by the fact that lactating women in Norway are not prescribed combination oral contraceptives during this period.

We found that the androgens that were associated with breastfeeding differed between our two groups of pregnant women. Thus, in the random group the 'classical' ones, i.e. androstendione and testosterone, associated negatively with breastfeeding while in the increased risk group only DHEA did so. This does not, however, exclude an important androgen effect on the breast tissue since DHEA is taken up by peripheral cells and converted to androgens (27). We can offer no explanation for this discrepancy between our two groups, but would like to point out that all androgens exert their biological effects via a common pathway by binding to the androgen receptor.

The fact that almost all women exclusively breastfed at six weeks and only 1% did so at six months means that three months was the only age

Table V.Pearson correlations between androgen and other variables known to associate with breastfeeding.

| | | | DHEAS | | DHEA | | Androstenedione | | Testosterone | | FTI[1] | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Variable | Group | *n* | $r^2$ | *p*-Value | $r^2$ | *p*-Value | $r^2$ | *p*-Value | $r^2$ | *p*-Value | $r^2$ | *p*-Value |
| Maternal age | Random | 63 | –0.22 | 0.078 | –0.19 | 0.14 | –0.22 | 0.090 | –0.18 | 0.16 | –0.18 | 0.15 |
| | Increased risk | 118 | –0.17 | 0.059 | –0.15 | 0.098 | –0.22 | 0.015 | –0.22 | 0.017 | –0.14 | 0.14 |
| Maternal BMI | Random | 63 | –0.04 | 0.74 | 0.02 | 0.88 | 0.01 | 0.97 | 0.20 | 0.12 | 0.28 | 0.028 |
| | Increased risk | 118 | 0.33 | <0.0005 | 0.04 | 0.68 | 0.09 | 0.32 | 0.06 | 0.51 | 0.15 | 0.11 |
| Maternal education | Random | 63 | 0.05 | 0.70 | –0.03 | 0.82 | –0.11 | 0.38 | –0.23 | 0.077 | –0.12 | 0.37 |
| | Increased risk | 117 | –0.31 | 0.001 | –0.23 | 0.013 | –0.18 | 0.047 | –0.12 | 0.20 | –0.08 | 0.40 |
| Smoking | Random | 60 | –0.07 | 0.58 | –0.06 | 0.67 | 0.29 | 0.023 | 0.33 | 0.009 | 0.20 | 0.12 |
| | Increased risk | 116 | 0.17 | 0.064 | 0.21 | 0.022 | 0.19 | 0.044 | 0.03 | 0.78 | –0.13 | 0.16 |
| Gestational age | Random | 63 | –0.05 | 0.68 | 0.16 | 0.21 | 0.25 | 0.053 | 0.17 | 0.18 | 0.22 | 0.077 |
| | Increased risk | 118 | –0.09 | 0.32 | –0.10 | 0.29 | –0.08 | 0.37 | 0.00 | 0.98 | –0.06 | 0.55 |
| Birth weight | Random | 63 | –0.16 | 0.21 | –0.01 | 0.91 | 0.13 | 0.32 | 0.10 | 0.44 | 0.19 | 0.13 |
| | Increased risk | 118 | –0.15 | 0.11 | –0.24 | 0.010 | –0.10 | 0.27 | 0.04 | 0.69 | 0.09 | 0.34 |

[1]Free testosterone index = (total testosterone/SHBG) × 100.
[2]Pearson's correlation coefficient.
Note: BMI, body mass index; DHEA, dehydroepiandrosterone; DHEAS, dehydroepiandrosterone sulfate; FTI, free testosterone index; SHBG, sex hormone binding globulin.

with a reasonably diverse distribution between no, partial, and exclusive breastfeeding. Accordingly, it is noteworthy that the negative association between breastfeeding and androgens was strongest at three months postpartum, both in univariate and multivariate analyses.

That we found breastfeeding to be associated with androgens in both study groups adds to the strength of our study. Since nulliparous women both breastfeed less and have a higher incidence of preeclampsia, which in turn is associated with hyperandrogenism, the omission of first-time mothers strengthens our study by giving more homogenous study populations (25). It is a further strength that the association with androgens persists after adjustment for factors shown to associate with breastfeeding. On the other hand, there is an obvious weakness that breastfeeding was associated with different androgens in our two study groups. We could have avoided this if we had only presented data from the random sample of pregnant women. However, we erroneously anticipated that androgen levels were higher and breastfeeding rates lower in the group of women with an increased risk for an SGA birth. We hold that researchers should present all the relevant data even when it may turn out to weaken the hypothesis. Further, it can be argued that given the relatively limited sample sizes, too many variables were included in the multivariate analyses.

Recent data from a well-conducted cluster randomized trial of breastfeeding seem to question many of the positive health effects for the child shown in observational studies (4–7). This raises the possibility that the statistical association between breastfeeding and offspring health depends on a common factor. We hypothesize that maternal gestational androgen levels may be this factor. If confirmed in future studies, one implication may be that the association between breastfeeding and offspring health is not purely an effect of breastfeeding, but is influenced by the intrauterine endocrine milieu presented to the fetus (28).

In conclusion, we found that breastfeeding was negatively associated with second trimester androgen levels, both in a random sample of parous pregnant women and women with an increased risk of giving birth to an SGA child. This may suggest that maternal androgen levels during pregnancy interfere with the ability to breastfeed and highlights the need for further investigations into the effect of gestational hormones on lactation.

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