



## Brief Report

## The physiology of women's power motive: Implicit power motivation is positively associated with estradiol levels in women

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## ABSTRACT

This study examined the relationship between implicit power motivation (*n* Power) and salivary estradiol in women. Forty participants completed the Picture Story Exercise, a measure of *n* Power, and salivary estradiol levels from two saliva samples were determined with radioimmunoassay. We found that *n* Power was positively associated with estradiol levels. The positive correlation between *n* Power and estradiol was stronger in single women and women not taking oral contraceptives than in the overall sample of women. These findings replicate those of Stanton and Schultheiss (2007), giving further credence to the argument that women's dominance striving is positively associated with their endogenous estradiol levels and that both social and biological factors influence the nature of that association.

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### 1. Introduction

The association between endocrinology and dominance motivation has been of interest to behavioral scientists since Berthold (1849) discovered that the removal and subsequent replacement of a rooster's testes led to a reduction in and resurgence of dominance behavior, respectively. This discovery occurred decades before testosterone had been chemically isolated, but it was clear that something being released from the testes was responsible for dominance motivation in males. Since then, the field of behavioral endocrinology has predominantly focused on testosterone and its relationship to dominance motivation in men (or other male mammals, Archer, 2006; Mazur & Booth, 1998). In the field of personality psychology, researchers have found that testosterone is positively associated with men's levels of implicit power motivation (*n* Power), which is a personality measure of dominance motivation in humans (Schultheiss, 2007). However, the biological basis of women's (and other female mammals') dominance motivation has often been overlooked (Cashdan, 2003; Stanton & Schultheiss, 2009). In the few studies that have examined biological correlates of dominance motivation in women, researchers have typically applied a male model to women by seeking to find consistent, positive associations between testosterone and dominance motivation. While some researchers have found that testosterone and dominance striving are positively associated in women

(Mehta, Jones, & Josephs, 2008; Mehta, Wuehrmann, & Josephs, 2009), several others have failed to find a consistent association (Booth & Dabbs, 1995; Cashdan, 1995; Gladue, 1991; cf. Archer, 2006; Mazur & Booth, 1998). With particular relevance to the current report, *n* Power is not related to levels of testosterone in women (Stanton & Schultheiss, 2007). Thus, research on the relationship between testosterone and dominance in women is inconsistent, and it is plausible that physiological factors other than testosterone underpin women's dominance motivation.

Studies of female mammals have shown that dominance motivation and behavior are positively related to levels of the steroid hormone estradiol, which is the most behaviorally potent form of estrogen and is produced and released principally by the ovaries (Bouissou, 1990; Faruzzi, Solomon, Demas, & Huhman, 2005; Michael & Zumppe, 1993; Zumppe & Michael, 1989). Yet, there was little consideration of such a relationship in humans until recently. Stanton and Schultheiss (2007) provided novel data that showed a positive relationship between women's *n* Power and their levels of estradiol. This positive association between estradiol and dominance motivation provided a human parallel to the animal literature (Michael & Zumppe, 1993). Moreover, the finding of Stanton and Schultheiss (2007) provided a cross-sex parallel to the positive relationship between men's testosterone and *n* Power, suggesting that estradiol might play a similar role in women's dominance motivation to that of testosterone in men.

However, the findings of Stanton and Schultheiss (2007) necessitate replication. Thus, in the present study, we aimed to replicate the positive associations obtained between salivary estradiol and *n* Power. Beyond the overall association between *n* Power and estra-

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diol, Stanton and Schultheiss (2007) also showed that single women showed a stronger correlation between *n* Power and estradiol than women in a close relationship. In addition, women who were not taking oral contraceptives had a stronger correlation between *n* Power and estradiol than did those women taking oral contraceptives. In the present study, we aimed to replicate these findings as well. However, Stanton and Schultheiss (2007) found that women in relationships were significantly more likely to be taking oral contraceptives, which was a confound that made it impossible to assert exactly which of these two factors was driving the relationships between *n* Power and estradiol. In the present study, we also examined the association between relationship status and contraceptive use. If contraceptive use and relationship status were unrelated, we could make a stronger case for independent effects of relationship status and oral contraceptive use on the relationship between *n* Power and estradiol.

## 2. Methods

### 2.1. Participants

Participants were a sample of 44 female undergraduate students (Age:  $M = 18.58$ ,  $SD = 0.81$ ) drawn from a larger sample of 102 total participants (58 men, see Edelman, Stanton, Henderson, and Sanders (submitted for publication), for additional details on the complete sample). Four participants' data were omitted from the analyses, because they reported having oral infections or oral lacerations, which can lead to blood contamination in saliva and subsequent elevations in steroid hormone levels (Schultheiss & Stanton, 2009), leaving 40 total participants. Twenty women reported being in a romantic relationship and 14 women reported taking oral contraceptives. On average, women reported being 17 days past the onset of their last menstruation. Participants were recruited randomly with no requirement regarding menstrual cycle stage, and subsequently represented all phases of the menstrual cycle. Participants were asked to refrain from eating, drinking, and brushing their teeth for one hour prior to the beginning of the experimental session. All procedures were approved by the University of Michigan Institutional Review Board. Participants received course credit for their participation.

### 2.2. Procedure

After informed consent was obtained, participants provided the first saliva sample that was later used to assess estradiol levels (Time 1). Participants then completed the Picture Story Exercise (PSE) to assess implicit power motivation (Schultheiss & Pang, 2007), followed by a demographic questionnaire that included information about relationship status, oral contraceptive use, and any other medical conditions that might affect hormone levels (Schultheiss & Stanton, 2009). Participants also completed a computerized attention task and a series of additional personality questionnaires, but the findings from these measures are not relevant to the current report. Lastly, approximately 1 h after the onset of the experiment, participants provided a second saliva sample (Time 2).

### 2.3. Saliva sampling

For each of the two saliva samples that participants provided, participants used a stick of sugar-free chewing gum to collect up to 7.5 mL saliva in a sterile polypropylene vial and then discarded the chewing gum (Schultheiss & Stanton, 2009). Participants sealed the vials immediately after each collection. The experimenter placed the vials in frozen storage immediately after the experimental session was complete. Samples were freed from muco-

polysaccharides and other residuals by three freeze thaw cycles followed by centrifugation.

### 2.4. Salivary estradiol measurement and characteristics

Salivary estradiol levels were assessed with solid-phase Coat-A-Count I-125 radioimmunoassays for estradiol (TKE2) from Diagnostic Products Corporation, Los Angeles. The assay manufacturer documents that its assay does not cross-react with estrogens in oral contraceptives. To determine salivary estradiol concentrations, we prepared water-based 1:80 dilutions of all standards (with a resulting range of 0.625–20 pg/mL) and controls (see Schultheiss, Dargel, and Rohde (2003a), for validation data; Schultheiss & Stanton, 2009). Eight hundred microliters of the saliva samples, standards, and controls were pipetted into antibody-coated tubes and allowed to incubate overnight. Next, 1-mL radio-labeled tracer was added to each tube and allowed to incubate overnight. Finally, tubes were aspirated and counted for 3 min. Analytical recovery was 104% on control samples of known concentration (0.48 pg/mL) (Bio-Rad Lyphochecks from Bio-Rad Laboratories, Hercules, CA). Analytical sensitivity (BO-3 SD) was at 0.05 pg/mL. Pooled saliva samples from female non-participants had an average concentration of 2.6 pg/mL, and the inter-assay coefficient of variation for this measurement was 12%. Participants' saliva samples were counted in duplicate and average intra-assay coefficients of variation for the two measurements were 9.77% and 7.91% at Time 1 and Time 2, respectively.

Taking two saliva measurements allowed us to test the stability of estradiol over time as well as the stability of the associations between *n* Power and estradiol. This is important because estradiol follows a diurnal pattern in which estradiol levels are highest in the morning and decrease progressively over the course of the day (Bao et al., 2003). Of note, estradiol also varies over the course of women's menstrual cycles: levels peak in the days around ovulation, but are relatively stable across the other phases of the cycle (Lu, Bentley, Gann, Hodges, & Chatterton, 1999; Riad-Fahmy, Read, Walker, Walker, & Griffiths, 1987).

### 2.5. Implicit power motivation

Implicit power motivation was assessed with the PSE using instructions specified by Schultheiss and Pang (2007). Participants were allotted 5 min per picture to write creative stories in response to pictures. Eight pictures were chosen for the PSE stories: *women in laboratory*, *ship captain*, *couple by river*, *trapeze artists*, *nightclub scene*, *boxer*, *girlfriends in café with male approaching*, and *bicycle race* (see Schultheiss & Pang, 2007, for a detailed description of the PSE stimuli and PSE methodology). These eight PSE stimuli were chosen due to their extensive use and validation in past research (Pang & Schultheiss, 2005; Schultheiss & Pang, 2007). PSE stories were coded for *n* Power by an expert coder, in accordance with Winter's (1994) Manual for Scoring Motive Imagery in Running Text. The themes in participants' PSE stories that were coded for implicit power motivation include strong, forceful actions that have impact over others, controlling others, influencing or persuading others, offering unsolicited help or advice, impressing others, fame, prestige, reputation, and actions that elicit a strong emotional response in others. On average, participants' eight PSE stories were a total of  $1007 \pm 48$  words long and contained a total of  $5.13 \pm 0.37$  power images. PSE total word count was correlated with the total number of *n* Power images in participants' stories ( $r = 0.28$ ,  $p = 0.08$ ). We corrected for this by using *n* Power images per 1000 words ( $M = 5.25$ ,  $SD = 2.44$ ) as our metric of *n* Power for all analyses (Schultheiss & Pang, 2007). The resulting *n* Power images per 1000 words metric did not correlate with total word count.

## 2.6. Design and data analysis

For the following analyses, salivary estradiol was the dependent variable and implicit power motivation was the independent variable. SYSTAT 12.0 statistical software was used for all analyses. Descriptive statistics are shown as mean ( $\pm$ SEM) unless otherwise noted.

## 3. Results

### 3.1. Estradiol concentrations and measurement stability

Participants' mean estradiol concentrations were  $2.15 \pm 0.13$  pg/mL at Time 1 and  $1.87 \pm 0.12$  pg/mL at Time 2. Estradiol levels were significantly lower at Time 2 as compared to Time 1 ( $t(38) = 4.44$ ,  $p < 0.001$ ). However, despite this expected diurnal decline in estradiol levels, we found a highly significant positive correlation between the two estradiol measurements ( $r = 0.91$ ,  $p < 0.001$ ), which suggests robust ordinal stability of estradiol levels over time. Thus, for all further analyses, we used the average of estradiol levels at Times 1 and 2 ( $M = 2.02$  pg/mL,  $SD = 0.77$ , Stanton & Schultheiss, 2007).

In the present sample, normally-cycling women ( $M = 2.18 \pm 0.16$  pg/mL) had significantly higher levels of estradiol than women taking oral contraceptives ( $M = 1.66 \pm 0.15$  pg/mL) ( $t(38) = -2.01$ ,  $p = 0.05$ ), but normally-cycling women ( $M = 5.83$ ,  $SD = 3.17$ ) did not differ in the levels of *n* Power from women taking oral contraceptives ( $M = 5.07$ ,  $SD = 2.19$ ) ( $t(36) = -0.84$ ,  $p = 0.41$ ). Women in relationships ( $M = 1.97 \pm 0.20$  pg/mL) and single women ( $M = 2.06 \pm 0.16$  pg/mL) did not have significantly different levels of estradiol ( $t(38) = -0.38$ ,  $p = 0.71$ ), nor did single women ( $M = 5.62$ ,  $SD = 2.90$ ) have significantly different levels of *n* Power than did coupled women ( $M = 4.93$ ,  $SD = 1.95$ ) ( $t(36) = 0.85$ ,  $p = 0.40$ ). In this sample, women in a close relationship were not more likely to be using oral contraceptives at the time of the study ( $\chi^2(1, N = 40) = 0.08$ ,  $p = 0.78$ ).

### 3.2. Relationships between *n* Power and salivary estradiol

As predicted, *n* Power scores were significantly positively correlated with salivary estradiol levels ( $r = 0.35$ ,  $p = 0.04$ , see Fig. 1).

Following our hypotheses, we then tested the effects of oral contraceptive use and relationship status on the nature of the rela-

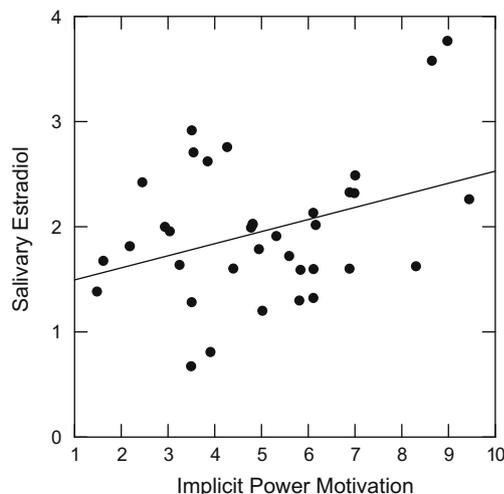


Fig. 1. Correlation between implicit power motivation (images/1000 words) and salivary estradiol (in pg/mL).

tionship between *n* Power and estradiol. Analysis of hormonal contraceptive use showed that the positive relationship between *n* Power and estradiol was significant in the 26 normally-cycling women ( $r = 0.44$ ,  $p = 0.03$ ), but not in the 14 women who were taking hormonal contraceptives ( $r = -0.15$ ,  $p = 0.68$ , see Fig. 2, Panel A). We used Fisher's *r*-to-*Z* transformation on the two contraceptive-use correlations to discern whether or not the slopes were significantly different, and we found that they were significantly different ( $Z = 1.70$ ,  $p = 0.04$ , one-tailed). When grouping the women into those who were in close relationships and those who were not, we found a significant, positive correlation between *n* Power and estradiol for the 20 single women ( $r = 0.48$ ,  $p = 0.04$ ), but not for the 20 women in a close relationship ( $r = 0.17$ ,  $p = 0.53$ , see Fig. 2, Panel B).<sup>1,2</sup> When comparing the slopes of these two relationship status correlations using Fisher's *r*-to-*Z* transformation, we found that they were not significantly different ( $Z = 1.02$ ,  $p = 0.15$ , one-tailed).

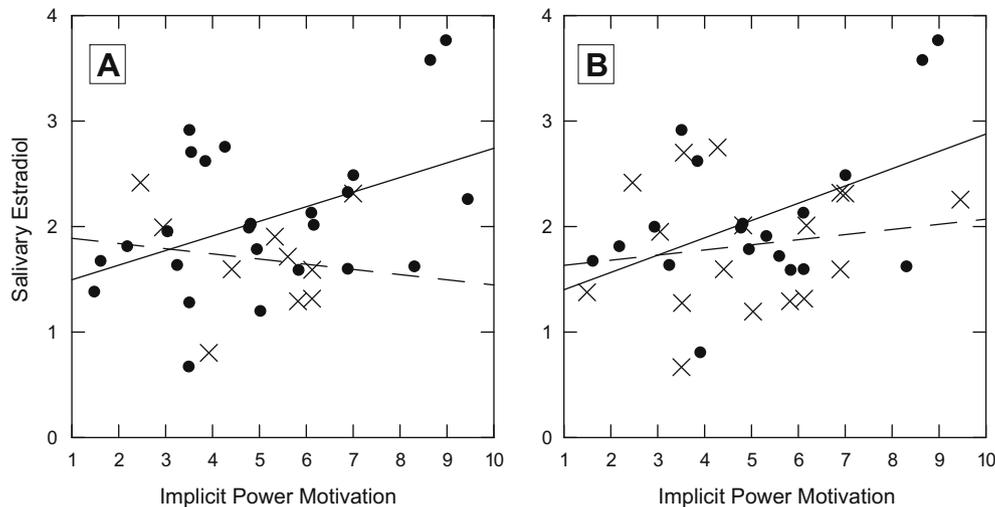
## 4. Discussion

The present data confirm our main hypothesis that *n* Power and estradiol levels would be positively associated in women, which replicates the findings of Stanton and Schultheiss (2007). In doing so, our findings also affirm the parallels between animal research and human research, in which estradiol and dominance motivation are positively associated (Michael & Zumppe, 1993). In addition, while testosterone has proven to be an unlikely candidate for a biological underpinning of dominance motivation in women (Mazur & Booth, 1998), the successful replication of the positive association between estradiol and *n* Power bolsters the assertion that estradiol plays a parallel role in dominance motivation in women to testosterone in men (Stanton & Schultheiss, 2009).

Our second hypothesis, that *n* Power and estradiol would be more strongly associated in single women than in coupled women, was also confirmed, which replicates the findings of Stanton and Schultheiss (2007). Our third hypothesis, that *n* Power and estradiol would be more strongly associated in normally-cycling women, was also confirmed, and also replicates the findings of Stanton and Schultheiss (2007). In Stanton and Schultheiss (2007), oral contraceptive use and relationship status were confounded because women in close relationships were significantly more likely to be taking oral contraceptives, yet in the present data, this relationship did not exist. Thus, we can assert that both single women and normally-cycling women, but not coupled women or those taking oral contraceptives, have significant positive associations between *n* Power and estradiol and that these effects are not

<sup>1</sup> We also tested the relationships between *n* Power and estradiol at each time-point. *N* Power was positively correlated with salivary estradiol at both time-points (Time 1:  $r = 0.39$ ,  $p = 0.02$ ; Time 2:  $r = 0.35$ ,  $p = 0.04$ ). The positive relationship between *n* Power and estradiol was significant in normally-cycling women at both time-points (Time 1:  $r = 0.47$ ,  $p = 0.02$ ; Time 2:  $r = 0.45$ ,  $p = 0.02$ ), but not in women who were taking hormonal contraceptives at either time-point (Time 1:  $r = -0.29$ ,  $p = 0.41$ ; Time 2:  $r = -0.01$ ,  $p = 0.97$ ). We found that there were significant positive correlations between *n* Power and estradiol for single women at both time-points (Time 1:  $r = 0.59$ ,  $p = 0.01$ ; Time 2:  $r = 0.45$ ,  $p = 0.05$ ), but not for women in a close relationship at either time-point (Time 1:  $r = 0.02$ ,  $p = 0.93$ ; Time 2:  $r = 0.29$ ,  $p = 0.25$ ). Thus, there were no discrepancies between the effects at each time-point and the effects derived from averaged levels of estradiol.

<sup>2</sup> We also log-transformed salivary estradiol to ensure that the reported correlations were not appreciably affected by two cases with high levels of both power motivation (1.40 and 1.53 standard deviations from the mean of *n* Power) and estradiol (2 and 2.25 standard deviations from the mean of estradiol). The results were highly consistent with those originally reported. *N* Power was positively correlated with log-transformed salivary estradiol for the whole sample ( $r = 0.31$ ,  $p = 0.06$ ), in normally-cycling women ( $r = 0.40$ ,  $p = 0.04$ ), and in single women ( $r = 0.41$ ,  $p = 0.08$ ). In contrast, *n* Power was not correlated with log-transformed estradiol in coupled women ( $r = 0.20$ ,  $p = 0.43$ ), or in women taking oral contraceptives ( $r = -0.07$ ,  $p = 0.84$ ).



**Fig. 2.** Correlations between implicit power motivation (images/1000 words) and salivary estradiol (in pg/mL) as a function of oral contraceptive use and relationship status. Panel A depicts the correlation for women who do (dashed line, exes) and do not (solid line, circles) take oral contraceptives. Panel B depicts the correlations for single women (solid line, circles) and for women in close relationships (dashed line, exes).

confounded. We also found that the slopes of the regression lines were significantly different between normally-cycling women and women taking oral contraceptives, but not between coupled and single women, which suggests that oral contraceptive use has a larger impact on the relationship between *n* Power and estradiol than relationship status does. These findings suggest that both biological (oral contraceptives) and social (relationship status) factors have the potential to influence the relationship between *n* Power and estradiol.

There are potential evolutionary benefits or explanations for the positive association between estradiol levels and *n* Power. *n* Power is positively associated with frequency of sexual intercourse in women (Schultheiss, Dargel, & Rohde, 2003b). Further, both sexual intercourse frequency and estradiol are highest in women around the time of ovulation (Udry & Morris, 1968). Thus, not only are both *n* Power and estradiol related to sexual activity, but they are also positively related to each other according to the present data. Increased sexual activity may be attributable to estradiol-facilitated increases in sexual motivation and in access to mates as a function of high levels of *n* Power and related dominance behavior (Schultheiss, 2007). This evolutionary explanation seems particularly plausible in normally-cycling women, who are periodically fertile and have ovulation-related increases in estradiol. In contrast, we hesitate to apply any evolutionary argument to women taking oral contraceptives, in whom ovarian production and release of estradiol is exogenously suppressed. Future studies that sample estradiol over the course of women's menstrual cycles will be able to more directly address whether or not increases in estradiol as a function of ovulation lead to increases in *n* Power and sexual intercourse.

Also from an evolutionary perspective, single women would benefit from increased access to mates, because they are currently without a sexual partner. In high-estradiol single women, corresponding high-levels of *n* Power could facilitate access to mates via dominance behavior. In coupled women, however, this link would be of lesser importance because these women already have access to a sexual partner. Future studies could attempt to test the effect of relationship formation and dissolution on the association between *n* Power and estradiol within the same individuals. We speculate that being in a close relationship could foster down-regulation of estradiol receptor expression in neural substrates regulating dominance motivation and behavior, as is the case for

other reciprocal relationships between neural networks and social behavior (Young, 2009). Such a modification of the neural networks would alter estradiol's ability to drive neural activity and subsequently influence the behavioral expression of estradiol-mediated behaviors and motives such as dominance and *n* Power, respectively (Michael & Zumpe, 1993; Stanton & Schultheiss, 2007). While these speculations offer a potential evolutionary explanation for the interrelatedness of these biobehavioral factors, the literature currently lacks a direct test of these hypotheses.

In conjunction, the present findings closely replicate those of Stanton and Schultheiss (2007). The consistency of this replication offers further support for a biological model of women's dominance motivation in which endogenous estradiol levels and women's dominance motivation are positively linked (Stanton & Schultheiss, 2009), and we believe that exploring the nuances of this association will be a fruitful area for future research.

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