The Influence of Relaxation with Biofeedback and Music on the Length of the Menstrual Cycle

by

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A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York.

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Abstract

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The purpose of this study was to see if relaxation at three different periods of a women's menstrual cycle would influence the duration of the cycle and day of ovulation. Students between the ages of 18-30 who were determined to have regular cycles based upon two months of record keeping, were given three relaxation sessions at different times from the start of menses, 12 during days 3, 4, and 5, 10 during days 8, 9, and 10 and 12 during days 19, 20, and 21. All participant were measured for the galvanic skin response (GSR) during a 20 minute relaxation session, 5 minutes baseline followed by 5 minute relaxation instructions and 10 minutes of music during which they were given biofeedback as to the GSR response. The GSR of 6 additional participants was measured with the relaxation manipulation 3 on days 3, 4 and 5 and 3 on days 19, 20, and 21. The findings are as follows: (1) Relaxation was most effective as measured by the GSR on days 19, 20 and 21, an important finding if one is interested in the effects of relaxation. (2) Greater relaxation as measured by the GSR within the session appeared to be effective in lengthening the start of the next menses; decreased relaxation appeared shorten the cycle. Findings were inconclusive regarding day of ovulation. This study is of importance in considering the therapeutic biofeedback treatment plans for women.

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INTRODUCTION

STRESS AND THE REPRODUCTIVE SYSTEM

Adverse influences of stress on the reproductive system have been reported in animals and in humans (Negro-Vilar, 1993). There are both anecdotal and scientific data to support the theory that stress hinders conception, and published research proposes a possible connection between stress and estrogen, and progesterone levels.

A Harvard University Medical School study reported that 33 percent of women who were taught relaxation by various techniques during infertility treatment became pregnant in the following six months (Domar, Zuttermeister, Seibel, et al., 1992). The study focused on the psychological dimensions of tension/anxiety, depression/dejection, anger/hostility, vigor/activity, fatigue/inertia, and confusion/ bewilderment. These traits were evaluated, and measures on the Spielberger Scale of Anger Expression (state and trait) (SSAE) obtained.

There was a significant positive influence of relaxation treatment on the scales of tension/anxiety, depression/dejection and confusion/bewilderment. There was also a higher level of expression of vigor/activity and a significant lower level of both state and trait anger (Domar, Zuttermeister, Seibel, et al., 1992).

Research on animal subjects (Ss) has shown that chronic stress causes anovulation (Barnea, and Tal, 1991), and that it can raise the number of natural abortions in mice (Clark, Banwatt, and Chaouat, 1993).

An Italian study found “stressed” women undergoing infertility treatments with in-vitro fertilization to have had less chance to become pregnant. In a sample of forty-nine women, stress level was measured by the Spielberger State Trait Anxiety test (SSTA), and the Stroop effect task, as well as by physiological arousal signs such as heart rate increase. Tests were conducted just prior to in-vitro fertilization.

The Stroop task, identifying the color of a "color-word" such as "black," written in red ink, requires the subjects to say "red." The discrepancy between ink-color and color-word commonly causes discomfort also manifest in heart rate acceleration. The Ss' heart rate was then measured and it was found that women who had high "trait" and "state" anxiety, and who had the greatest heart rate increase had the least successful in-vitro fertilization. Moreover, even though some of the more stressed women became pregnant, these women didn't carry the pregnancy to full term (Fanchinetti, Matteo, Artini, et al., 1997). The possibility of carrying a pregnancy to full term is dependent partly on progesterone levels.

In another study of the relationship between stress and fertility conducted in Finland at regular intervals over a period of six months, one-hundred ninety-one healthy women (without a history of infertility) who wished to bear children where examined both psychologically and gynecologically. (Vartiainen, Saarikoski, Halonen, et al., 1994). The women who were the most fertile typically had the following general profile:

- They showed no body-weight fluctuation before pregnancy
- They consumed less coffee
- They were younger then their spouses and looked younger than their age
- They scored lower on psychosomatic symptoms
- They had fewer negative life changes

Coffee consumption and life-changes are known to cause stress, and so also do frequent weight changes. It therefore may follow that women with lower coffee consumption and fewer life changes may be more fertile (Vartiainen, Saarikoski, Halonen, et al., 1994).

In a study by Nielsen, Zhang, Kristensen, et al. (2005), women who were more stressed than controls were less prone to develop breast cancer (the stress levels were based on self report). In this study, seven thousand women where followed over a period of eighteen years. An inverse relationship was found between daily stress levels and breast cancer. The explanation given was that the stress impaired the levels of estrogen

synthesis, and the higher the stress levels, the lower the levels of estrogen, and the less the chance to develop breast cancer.

Stress is also well known to raise noradrenaline and other stress hormone levels. Among the hormones found to be related to stress are cortisol, adrenocorticotropic hormone (ACTH) (Rivier, Rivier, and Vale, 1983), prolactin, and growth hormone (GH) (Uhde, 1994; Kant, Meyerhoff, Brunnell, et al., 1982; Pancheri, Biondi, Fierro, et al., 1982). It has also been suggested that stress influences the level of follicle stimulating hormone (FSH) and luteinizing hormone (LH)) (Pancheri, Biondi, Fierro, et al., 1982).

Corticotropin releasing factor (CRF) has been found to inhibit the activity of luteinizing hormone-releasing hormone (LHRH), and that influences the LH, as noted above. FSH and LH are directly connected to the process of ovulation: FSH contributes to the maturation of follicles, and lutenizing hormone helps the ovum get released from the ovaries. Growth hormone (GH) has also been found to play a role in ovulation (Lunenfeld, Menache, Dor, et al., 1991). Suppression of sex hormones in girls with "central" precocious puberty lowers the levels of GH (Manfield, Rudlin, Crigler, et al., 1988).

Morse, Dennerstein, Farrell, et al. (1991), reported that Pre-Menstrual Syndrome (PMS) (related to hormones and psychological factors), can be treated hormonally. But, it has been found that cognitive-behavioral therapy can also relieve those symptoms.

The basis for treatment with a behavioral technique is that many of the PMS symptoms are psychological, i.e., anxiety, crying spells, depression, fatigue and irritability (Speroff, Glass, and Kase, 1989). In a sample of forty-four women between the ages of twenty-two and forty-five, divided into three groups, one group underwent behavioral therapy, a second group received hormonal therapy, and the third group was given a kind of relaxation therapy.

The relaxation group outcome was found to be equivocal—perhaps due to poorly supervised relaxation: The women were instructed on how to relax at home without

supervision, and most of them dropped out of the experiment. Yet, the behavioral therapy was found to be very beneficial even three months following treatment (Morse, Dennerstein, Farrell, et al., 1991). The fact that PMS can be treated with both a psychological therapy and hormonal therapy might suggest that these may compliment each other.

Estrogen is a known factor in asthma attacks as well. When estrogen levels decrease around the time of the menses (day twenty-six, to day four), women are more prone to asthma attacks (46 percent of women are admitted to hospitals during that period, as compared to 20 percent admissions at the pre-ovulation period; or 24 percent around the time of ovulation, and 10 percent just after ovulation) (Skobeloff, Spivey, Silverman, et al., 1996). Day twenty-six to day-four corresponds in part to the pre-menstrual period where some symptoms of depression and anxiety also often appear.

The onset of anxiety seems related to decreasing estrogen. Since decreased estrogen levels occur contiguously to psychological changes, one cannot rule out that the anxiety may trigger the asthma attack. Asthma symptoms have been treated successfully with biofeedback (Kotses, Harver, Segreto, et al., 1991; Leherer, Hochoron, McCann, et al., 1986).

In studies relating stress and hormones, stress induced by putting rats in a tube for six hours a day, for twenty-one days, resulted in Ss having higher levels of cortisol. When they were stressed and subsequently tested in a maze task that required memory, they did poorly as compared to non-stressed controls (Luine, Spencer, and McEwen, 1993). When these same Ss were administered estrogen, they responded to the maze task as if they had not been stressed. We may assume that improvement in memory for the maze test was due to estrogen supplement. In a related experiment by Galea, McEwen, Tampa, et al. (1997), it was found that physically restricting female rats for twenty-one days resulted in lower plasma estradiol levels.

Stress has also been found related to levels of growth hormones (GH) . The GH level decreases after a long exposure to stress: Consistently stressed Pointer pups (especially

females) grew shorter than their estimated optimal height (Uhde, Malloy, and Slate, 1992). This seems to support the finding that, in the long run, stress depresses levels of growth hormone (Uhde, 1994).

It has been shown that estrogen up-regulates the level of growth hormone (Adashi, 1994). Therefore, lower levels of estrogen, if indeed lowered by stress, could explain why the female dogs were more affected by stress then the male dogs. In the short term GH levels have paradoxically also been found to rise as a result of stress (Faracce, Bisseli, Urbani et al., 1996)

The way that stress influences reproductive hormones is related to ACTH (River, and Vale, 1983), to cortisol, and to DHEAS (Dehydroepiandrosterone sulfate) (Suh, Lui, Bergas, et al., 1988). ACTH plays a role in the synthesis of estrogen as a precursor of pregnenolone (Goldfien, 1988).

Corticotropin-releasing hormone (CRF) is activated during stress and influences the function of the pituitary gland, where the sexual hormones are secreted. The CRF also influences the secretion of cortisol: As increased level of cortisol reaches a certain threshold, it inhibits the the action of CRF and thus influences the secretion of sexual hormones in the pituitary gland (Sternberg, and Gold, 1997).

A study by Berga (2006) reported that twenty sessions of behavioral therapy for women suffering from Hypotalamic-Amenorrhea (anovulation) caused 75 percent of them to ovulate again, as compared to 25 percent in a no-therapy control group. Berga attributed this improvement to lowering the levels of stress and, consequently, lowering the levels of cortisol.

Altemus, Redwine, Leong, et al. (1997), contend that glucorticoids (cortisol being one of them) play a role in attenuation of the stress levels at the early follicular stage, but not to the same extend at the luteal stage. The glucocorticoids (cortisol) function as a feedback loop lowering cortisol concentration when it reaches a certain level.

In their study, dexamethasone (a glucocorticoid) used to lower cortisol levels, was administered to women at the early follicular and the luteal phase. Dexamethasone was found more effective at the early follicular stage. This is thought to be mediated by the presence of a greater number of receptors for glucocorticoids at the early follicular stage, as compared to the luteal stage.

In a more resent publication by Goldstein, Jerram, Poldrack, et al. (2006), women were put under the scrutiny of a functional MRI and presented with neutral as well as arousing stimuli, at the early follicular stage and the late follicular stage:

It was found that there was a significantly larger change in blood oxygenation (SaO2) level when the arousing stimuli were presented during the early follicular stage, as compared to the late follicular stage. This change in SaO2 was noted in the central amygdala, the paraventricular and the ventromedial hypothalamic nuclei, hippocampus, orbitofrontal cortex (OFC), anterior cingulate gyrus (aCING), and peripeduncular nucleus of the brainstem. All these areas are involved in the response to stress:

Arousal , as measured by Electro Dermal Activity, (see chapter on Biofeedback for definition) correlated positively with brain activity in the amygdala, OFC, and aCING during the late follicular stage, but not in early follicular stage, suggesting less cortical control of the amygdala during early follicular stage, when arousal was higher. This is the first experimental evidence suggesting that estrogen may likely attenuate arousal in women via cortical-subcortical control within HPA circuitry.

Ovarian steroids have been found to modulate the pathophysiology of depression in the stress responsive hippothalamic-pituitary-adrenal (HPA) axis. However, there is no clear indication whether it is estrogen or progesterone that is critical (Young, and Korszun, 1999). It seems that when the testing was done at the late follicular stage prior to ovulation (Goldstein, Jerram, Poldrack, et al. (2006), estrogen might be the one that modulated stress levels, as progesterone was still low then.

In a study by Young, Altemus, Parkison, et al. (2001), it was found that female rats are less sensitive then males to hypothalamic-pituitary-adrenal feedback inhibition by exogenous glucocorticoid administration. The study concluded that estradiol decreased the levels of the stress hormone, ACTH, in restricted Ss, underscoring the importance of estrogen in the HPA axis of stress.

It has also been reported that:

- anxiety at menopause lessens with estrogen treatment (Campbell, and Whitehead, 1977),
- barbiturates increase estrogen metabolism, but also increase metabolism of corticosteroids (Hansten, 1989), and
- estrogen levels influence the sensitivity to barbiturates.

The higher the estrogen levels, the more the women became sensitive to barbiturates. The most sensitive to barbiturates were the pregnant women and, conversely, the less sensitive were the postmenopausal women (Bergman, Niv, David, et al., 1987). This suggests the possibility that higher levels of estrogen enable relaxation.

If estrogen levels affect relaxation, then it may follow that biofeedback might be more effective at times when estrogen levels are highest at the late follicular and at the luteal phase of the menstrual cycle.

Lowering stress can be achieved with biofeedback (Schwartz, and Olson, 1995). But the conventional treatment for menopausal “hot flashes” is estrogen with progesterone (progesterone is added to reduce the risk of cancer). Biofeedback, using the paced respiration technique (to be explained in a later section), has also been found useful in reducing the frequency of hot flashes (Lee, and Taylor, 1987; Freedman, and Woodward, 1992). This suggests a connection between relaxation and estrogen level.

Stress may cause spontaneous abortions perhaps because the stress hormones adrenaline, noradrenaline and cortisol influence the metabolism of other hormones, i.e. a decrease of serum progesterone, and an increase of thyroxine-level. Stress also plays a

role in immune function, and immune function plays a role in preventing the rejection of the embryo (Lapple, 1988).

Suh, Lui, Bergas, et al. (1988), found that women with Functional Hypothalamic-Amenorrhea (HA) had lower levels of estrogen, and higher levels of cortisol, compared to women who ovulated regularly. But, this was only found when these hormones were measured between 8 AM and 11AM. When these same hormones were measured over a period of twenty-four hours, the difference mostly disappeared. The only difference found over a period of twenty-four hours was in the elevated level of DHEAS and of cortisol, especially between 8AM and 4 PM, and between 12 PM and 8 AM. The level of LH was the same in both population, but the pulses of secretion of LH were more time-distant from each other, and less regular in the HA women, then those cycling normally.

A confounding factor was that the HA women were compared to normally cycling (NC) women in the early follicular stage (days two-to-five). That is the time of their "period" where estradiol levels are at their lowest.

This comparison between HA and NC women just shows that the level of estrogen of non-ovulating women is not different from the one found at the beginning of a normal cycle: In other words, in non-ovulating women with Hypothalamic-Amenorrhea, the level of estrogen is at a constantly low level, and they also have a higher level of cortisol that is associated with stress.

Perhaps, by lowering their stress levels, these women might be brought back into a cycle where estrogen levels fluctuate. This was indeed found by Berga (2006) in a study where behavioral treatment helped 75 percent of twenty women suffering from Hypothalamic-Amenorrhea, by helping to lower cortisol levels so that they were able to ovulate again.

Can we change the levels of cortisol with the help of biofeedback and thus influence the levels of estrogen?

BIOFEEDBACK AND CORTISOL

A series of experiments (McGrady, Williams, Utz, et al.,1986; McGrady, Woerner, Argueta, et al., 1987; McGrady, Nadsady, Schumann-Brzezinski, et al., 1991; McGrady, Conran, Dickey, et al., 1992; McGrady, 1994) examined the influence of biofeedback on human hypertension. In these studies, plasma cortisol and urinary cortisol were also measured. In a 1981 study (McGrady, Yonker, Tan et al.), thirty-eight patients, most of them medicated participants, took part in an eight-week EMG, biofeedback-assisted, relaxation program:

The participants came twice a week for eight weeks, thirty minutes each time. The measurements were blood pressure as well as measurements of urinary cortisol, plasma aldosterone, and EMG readings. A significant decrease in the blood pressure was found between the pretest and the post treatment in the experimental group. The control group (no biofeedback) stayed at the same level of blood pressure. In the experimental group, there was a decrease in all the other readings, i.e., the EMG, the urinary cortisol levels and the aldosterone level. In the control Ss, none of the measurements changed from the pre-test to the post-test levels.

Another study sought to find out, by establishing the preliminary profile of patients with high blood pressure, which ones would benefit from treatment with biofeedback and relaxation (McGrady, 1994). Here again, cortisol levels were measured as well as blood pressure and heart rate, muscle tension, depression and anxiety. Treatment was relaxation training and thermal biofeedback. Eighty percent of the participants were medicated.

The experimental group received eight sessions of relaxation and thermal biofeedback. They were also given relaxation tapes and thermometers and told to exercise at home for ten to fifteen minutes a day. The Ss were seen on three consecutive weeks, and then followed up after ten months for long-term effect of the relaxation. A profile emerged showing those who had higher levels of urinary and plasma cortisol to

begin with had the most benefit from the relaxation treatment, and they had the most chances over time to keep their blood pressure lower.

In another study by McGrady, Williams, Utz, et al., (1986), one group of Ss suffered from high blood pressure and controls did not. The EMG biofeedback treatment administered to both groups had no influence on the blood pressure of control Ss, and it had also no influence on their levels or cortisol. Biofeedback only influenced the cortisol levels of the Ss with elevated blood pressure.

DeGood, and Redgate (1981), reported that Ss suffering from headaches, gastrointestinal upset, pain, and other psycho-physiological symptoms were given EMG (muscle tension) biofeedback sessions while heart rate, vasodilatation, and plasma cortisol were recorded. After baseline recordings, the Ss received eight weekly biofeedback sessions. It was found that the higher the cortisol levels, the higher the heart rate. In the follow up and the eighth biofeedback session, there was a significant inverse relationship between cortisol levels and vasoconstriction: The higher the cortisol levels the more constricted the blood vessels.

CORTISOL AND ESTROGEN

Biofeedback has been shown to influence levels of cortisol, but is there a relationship between cortisol levels and levels of estrogen? In unpublished data (Davydov, Shapiro, Goldstein, et al. (2005), the only significant correlation was that between estrogen levels and cortisol levels on a day off from work, and at the luteal phase (that might be compared to the effect of relaxation).

At the luteal phase the lower the cortisol levels, the higher the estrogen levels. But, no such relationship was found on the early follicular stage. Perhaps, this relationship was not found at the early follicular stage because estrogen levels are then very low to begin with, and the buffering system may be more active at that phase of the cycle (Altemus, Redwine, Leong, et al. 1997).

Novy, and Walsh (1983), showed that the administration of dexamethasone to pregnant rhesus monkeys caused their levels of cortisol to decrease as well as the levels of estrogen—dexamethasone is a glucocorticoid like cortisol and probably influences levels of cortisol via the HPA axis feedback-loop. Another finding was that this caused a prolonged gestation in the monkeys. The administration of dexamathasone, a glucocorticoid like cortisol, prevents the biosynthesis of estrogen.

Komesaroff, Essler, and Sudhir (1999), have found that administering estrogen for eight weeks to perimenopausal women attenuated the amount of cortisol secreted, and attenuated the rise in blood pressure during the performance of a mental arithmetic task, as compared to Ss receiving a placebo—who also performed the same task.

Higher levels of cortisol result in lower levels of estrogen, and conversely, lower levels of cortisol result in higher levels of estrogen. Del Rio, Valardo, Menozzi, et al. (1998), found that administration of estradiol and progesterone to menopausal women reduces their cardiovascular response to a stressor. Estrogen and progesterone seem to serve as buffers to stressors at menopause.

In that study of menopausal women it was found that acute administration of these hormones reduced the response to a Stroop effect task. This reduction in effect was noted as a less pronounced rise in adrenaline levels during the Stoop effect task in the group that was administered estrogen and progesterone as compared to a group that was administered a placebo. There was also a lesser rise in systolic blood pressure in the experimental group as compared to the placebo group. There was no significant difference in heart rate, or cortisol levels, between the experimental, and the control group (Del Rio, Valardo, Menozzi, et al., 1998).

The secretion of cortisol has been found to be influenced by biofeedback. Estrogen levels might be influenced by relaxation at least in the luteal phase. I found no data for the late follicular phase. If estrogen is influenced by relaxation then one would expect it to influence the level of FSH and LH in different ways, depending on whether the relaxation is being introduced in the early follicular stage of the cycle (from the second to the seventh day of the cycle), or later, at the late follicular phase (from the seventh day until ovulation).

Low levels of estrogen enhance FSH and LH synthesis and storage, and have little effect on LH secretion—but they inhibit FSH secretion (at the early follicular stage). High levels of estrogen induce the LH surge at mid-cycle, and high steady levels of estrogen lead to a sustained elevated LH secretion (Speroff, Glass, Kase, et al., 1989).

Assuming that estrogen levels are indeed influenced by relaxation, introducing biofeedback at the early follicular stage might be expected to postpone ovulation. On the other hand biofeedback at the later stage might cause earlier ovulation as the estrogen contributes to higher levels of LH, in turn causing an earlier LH surge and, therefore earlier ovulation.

Previous research on the effect of biofeedback on LH, and FSH hormones, did not produce unequivocal results (Pancheri, Biondi, Fierro, et al., 1982). But this could be explained by the fact that the stage of the cycle might not have been taken into account when the measurements were made.

In a private communication of the data from an article by Davidov, Shapiro, Goldstein et al.(2005), one-hundred eighty-seven nurses were monitored for different hormones and mood changes over one cycle. Questions centered on the influence of the cycle stage—early follicular versus luteal stage—as well as a day of work—versus a day off from work.

In the follicular stage, women on a day off from work had higher levels of cortisol in the morning, as compared to the morning after a work-day. Could these differences between the early follicular stage and the late follicular stage be connected to sleeping pattern? One can find no report of such differences (Baker, and Driver, 2004**).** The only difference found was of more REM sleep during the follicular stage. Some women also report feeling more rested at the follicular stage. (Tworoger, Davis, Vitiello et al., 2005).

Other findings include:

- at the end of the day of rest, there is no decrease in cortisol level as compared with a work-day,
- on a day off from work (PM) the women in the follicular stage had significantly higher levels of cortisol then women at the luteal stage,
- comparing a work day to a day of rest in the luteal phase (PM), the work day sees significantly higher levels of cortisol,
- at the luteal stage women are more prone to the influences of stress than women at the follicular stage.

The explanation for these findings was a feedback system that keeps women in homeostasis in the follicular stage (Altemus, Redwine, Leong, et al., 1997). But, it is a feedback tool that doesn't work quite as well at the luteal phase when estrogen levels are higher. In that article, the late follicular stage is not mentioned (Altemus, Redwine, Leong, et al., 1997).

The research by Davidov, Shapiro, Goldstein, et al., (2005), seems to deal not so much with stress per se, but perhaps with "stressability": Women are more "stressable"

at the luteal phase, i.e., they are more easily disturbed from homeostasis than women at the follicular stage. But, women are also more “relaxable” at the luteal stage than in the follicular stage, as shown by the cortisol data.

It seems that it is easier for women to relax at the luteal phase and at the late follicular phase, than at the early follicular phase. Here again, women in the luteal phase and women in the late follicular stage can be disturbed out of their state of homeostasis more easily than women at the early follicular stage.

Women react in different ways to stress: In some, stress lengthens the cycles, while in others it shortens them. Therefore, it could be hypothesized that stress influences the hormonal system if any consistent and significant change in cycle length is detected.

REGULARITY OF THE MENSTRUAL CYCLE

Are there women with regular menstrual cycles,? First, what does “regular” mean? It means having menstrual periods at regular fixed intervals give or take two days In general, the term “regular” is often misunderstood or, perhaps, regular cycles may be uncommon.

The women that participated in this experiment during the five years that it has been ongoing were asked, “Do you get your period regularly” and the meaning of *regularly* was detailed for them with an example—for instance every twenty-six day. If the answer was “yes,” then they were eligible to participate in the experiment. At the third month, when the participants came and showed the dates on which they’d got their period for the last two month, it could be seen that there was sometimes more then three days difference between each cycle, i.e., twenty-six to twenty-nine days.

Creinin, Keverline, and Meyn (2004), conducted a survey on menstrual diaries obtained from women who participated in studies of barrier contraceptives. The diaries were to check their eligibility for a study on contraception. It was required that they be “regular.” The Ss were one hundred thirty women between the ages of eighteen and forty years who reported a “regular cycle.” The participants recorded their actual cycle length and the number of days they bled. This was over a period of a few months.

The average menstrual cycle length often differed by three days from the subjects’ estimated cycle duration. The median cycle range was seven days (seven days difference between the shortest cycle and the longest cycle); the average cycle range was a little more than ten days. Forty-six percent of the participants had a cycle range greater than, or equal to seven, meaning more then seven days difference between the shortest cycle and the longest cycle. Twenty percent had a cycle range greater than, or equal to fourteen days. In another study (Steiner, Hertz-Picciotto, Taylor, et al., 2001), a similar pattern was also found: in 53 percent of cases, women’s report of the average length of their cycle was within one week of their original report.

Clearly, many women were not quite as regular as they claimed to be. Their perception of regularity is interesting in itself, but the real question is what influences the length of a women cycle?

The menstrual cycle is also influenced by stress and by other factors also. At the onset of puberty, the cycle does not occur regularly—the cycle interval at the beginning can be as long as forty days. Furthermore, it may take up to twelve cycles, or more, to regulate and then the cycle is about twenty-eight days long on average.

The first cycles before regularity is reached may not involve proper ovulation (Boros, Lampe, Balogh, et al., 1988). But, once the cycle is regular, girls also start ovulating regularly with each cycle and might conceive. Later in life, when reaching menopause, the cycle becomes longer and longer in duration until it stops entirely.

Diet has also been reported to influence the length of the cycle. Jones, Judd, Taylor, et al. (1987), it was found that changing from a high fat diet (40 percent of daily energy from fat) to a low fat diet (20 percent from fat), there was a significant lengthening in mean cycle duration: The cycle lengthened by an average of 1.3 days and the menses increased in duration by 0.5 days.

Cholesterol is a precursor of estrogen and progesterone (Goldfien, 1989). Thus, a corresponding change in cycle length might be expected as its dietary intake increases, or decreases. In a similar study by Gann, Chatterton, Gapstur, et al. (2003), consistently changing to a low fat, high fiber diet, results in a 7.5 percent decrease in estrogen levels over a period of twelve months.

These findings largely explain why it took the present study so long to complete, as the question that also needed to be addressed was “why do women claim regularity when in fact they are not regular?” There is no published explanation for this phenomenon. One possible explanation is the common practice of mixing interchanging the terms “regular” and “normal.”

Do we generally assume that “regular” is the norm? Do we prefer to see ourselves as “normal,” and therefore regular? In an informal pilot survey, participants in a women’s learning group were asked to address this question.

Ten women completed a questionnaire about the regularity of their cycle. Then, they were asked how many days elapsed between their cycles. The last question asked what percentage of the population they thought to be regular? From this sample, the following emerged:

- five women of the ten women said that 80 percent of women are regular,
- one woman said 60 percent of women are regular,
- another said most are regular, and
- only one said that 10 percent of women are regular.
- Two women said they did not know.
- All ten women considered themselves to be regular.
- When asked what is their definition of regular three women said getting a period once a month, six women said to get it at fixed intervals, and one said getting your period every month or every two month.

If this sample is representative, it seems that there may be a general belief that most healthy women are regular. It is now well known, however, that this is not the case.

All the subjects in my sample reported themselves to be regular. So, if regular is just getting a period every month then, yes, they might be regular. However, only six of the Ss gave the right definition, i.e., getting a period at fixed intervals, and still they saw themselves as regular. This would suggest that 60 percent of my sample were regular, and that is highly improbable. So even if a woman knows the definition of regular, there might still be a discrepancy between her report and her actual menstrual cycles.

The menstrual cycle is known to be influenced by stress, but there are other factors too.

THE ELECTRODERMAL RESPONSE (EDA) AND THE MENSTRUAL CYCLE

Electrodermal activity (EDA)—EDA is measured as change in conductance of an electrical impulse between two adjacent locations on the surface of the skin. It is influenced by the secretion of sweat that is, itself, influenced by stress: the more stress, the more sweat glands are activated. The more sweat produced, the higher the electrical conductance of the electrical impulse.

Historically, most of the research on EDA centers on the effects of some stressful stimuli. Findings on the relationship between EDA and the menstrual cycle have been contradictory, result varing from:

- no differences in EDA along the cycle (Kopel, Lunde, Clayton, et al., 1969; Wineman, 1971; Zimmerman, and Parlee, 1973; Slade, and Jenner, 1979; Strauss, Schultheiss, and Cohen, 1983), to
- the EDA is higher at the luteal phase (Menhem, Hansson, Milsom, et al., 1996), to
- one finds a higher EDA in the phase just before the menses (Asso, and Braier, 1982; Chattopadhyay, and Das, 1983; Asso, 1986), to
- the EDA is greater at the early and late follicular stages (Uno, 1973; Little, and Zahn, 1974; Rosenberg, 1980; Plante, and Denney, 1984; Gomez-Amor, Martinez-Selva, Roman, et al., 1990).

It was also found by Cevik (2006), that as estrogen levels increase so does the amount of nitric oxide (NO) generated in the body—NO is a known vasodilator. This dilatation of the blood vessels decreases resistance also allowing for better blood flow through tissues (Chen, and Ma, 2005; Durand, Davis, Cui, et al., 2005).

How do these disparate findings shed light on the relationship between relaxation and the menstrual cycle?

In an experiment by Little, and Zahn (1974), twelve women were tested on a simple reaction time task, depressing a button for what they perceive to be five seconds, or

pressing it when hearing a sound. These Ss were tested for twenty minutes, six days per week, for the duration of the cycle.

Heart rate, skin temperature as well as skin conductivity (base line) where measured before and during the button-press task. When at rest, there was an increase in heart rate and respiration during the luteal phase, as compared to the other phases of the cycle, as well as lower skin conductivity. During the button pressing task, the skin conductance changed the most at the ovulatory stage (the largest amplitude, and the latency, the time it took to reach this amplitude was the fastest). These results indicate that skin conductance is lower at the luteal phase of the cycle probably when progesterone prevails.

Mackinnon, and Harisson (1961), found that injection of progesterone decreases palm sweat. This would explain the lower conductivity in the luteal phase. But, in Kopel, Lunde, Clayton, et al. (1969), we see a very different picture: Here, eight women where studied on the third, fourteenth, twenty-fourth, twenty-sixth and twenty-eighth day of the cycle, over two cycles:

The two tasks in that study were detecting the threshold between two flashes, and also pressing on a button for a time that is perceived as being thirty seconds. Skin conductance was measured as well as plasma cortisol. The differences that were found over the length of the cycle were in the estimation of time, as well as in the task of detecting the threshold between two flashes. The time needed to detect two light flashes as separate became longer at the luteal stage, but there was no difference in conductivity over the cycle. Cortisol and time estimation were found to be related but no relationship was found between cortisol and skin conductance.

The galvanic skin response (GSR) is the immediate change in skin conductance following presentation of a stimulus. A study by Uno (1972), examined GSR monitored at three different stages of the cycle, i.e., days five to seven, comparable to the early follicular days twelve to fourteen (ovulatory), and days twenty-four to twenty-eight (pre menses). It was found that only when the subjects are stimulated (in this case by an 80 dB, 1000 Hz sound) can one detect a difference between the different phases of the cycle.

In that study, the largest change in conductivity (as a reaction to the sound) was found at the early follicular stage, and the smallest change in GSR was at the late luteal stage just prior to the menses. In a similar experiment by Gomez-Amor, Martinez-Selva, Roman, et al. (1990), the increase in conductance to a sound was at the early follicular stage and the magnitude of the conductivity was the highest at the early follicular stage. Yet another finding from this study, as well as others (Chattopadhyay, and Das, 1983) concerns habituation.

Habituation in these experiments is observed as "no change" in conductivity as a result of repeated exposure to a given sound stimulus. Gomez-Amor, Martinez-Selva, Roman, et al. (1989), showed that habituation was reached maximally in the early follicular stage of the cycle.

Another measure of habituation is a steady decline in conductivity as the Ss are continuously exposed to the same situation. This is illustrated in Gomez-Amor, Martinez-Selva, Roman, et al. (1990). Twenty minutes a day, six days a week, for a whole cycle.

In a study by Chattopadhy, and Das (1983), it was found that when women are repeatedly presented with a light flash, their level of arousal rises higher in the premenstrual phase than in the menstrual phase. When the same stimuli are presented in the mid cycle, this rise in arousal level is not detected. Habituation to the stimuli was found to be the fastest in mid cycle, as compared to the menstrual and premenstrual stage.

BIOFEEDBACK AND RELAXATION TECHNIQUES

Biofeedback provides indirect but immediate and continuous information about certain involuntary autonomic responses. The biofeedback clinical and research application are based on the assumption that psychological changes translate into such involuntary physiological changes that can then be controlled also only indirectly. There are several different physiological modalities that respond to biofeedback.

1. Skin Temperature

Skin temperature measured at the fingers tips has been reported to decrease with stress, because stress constricts peripheral blood vessels there thus diminishing the local blood circulation. The effects of counter stress relaxation therapy can be measured by increase in finger tip temperature as one relaxes.

In biofeedback, the person observing his/her temperature rise is encouraged to continue the trial and error behavior that led to temperature rise. The finger skin surface temperature can also be transduced into either a visual "feedback" stimulus, i.e., a line graph rising on a computer display, or a tone that decreases in pitch or loudness as temperature rises. Simply getting feedback from a small finger thermometer can also serve same the purpose.

These visual or auditory feedback signals are also thought to be "reinforcing" when contiguous to relaxation.

2. Electro-dermal activity (EDA)

Generally, the higher the baseline (control) electrical resistance (measured in ohms) to a current passed between two adjacent points on the skin surface, the less stressed one is assumed to be. The reciprocal of resistance is conductance measured in Siemens—usually micro-Siemens (1000/kohm) (Schwartz, 1995). This exosomatic measure of skin resistance consist of phasic changes—changes that occur at a relatively fast rate (Venables, and Christie, 1980).

3. Electromyography (EMG)

Muscle tension is also a sign of stress. It is not ordinarily measured directly but as muscles tense, their emitted electrical signals can be measured in microvolts. The higher that voltage, the greater is the tension. Relaxation training serves to lower those microvolts (Schwartz, 1995).

Several different methods may be used to reach a relaxed state:

- *Paced Respiration* is a relaxation technique where duration of inhale and exhale are paced at a predetermined rate (Clark, and Hirschman, 1990; Lichstein, 1988).
- *Breath Meditation and Breath Mindfulness* including passive breath mindfulness and breath holding are methods derived from yoga, that require that one concentrate on breathing and by so doing, prevent other thoughts from intruding the mind and causing stress (Schwartz, 1995).
- *Muscle relaxation*, the subject learns to alternately tense and then relax skeletal muscles.

RELAXATION AND SELF-REGULATION

There is a difference between relaxation and self-regulation: The first is passive and the second is active. In passive relaxation, for instance, the subject can listen to relaxing music that can help him/her to relax. Active relaxation employs techniques such as progressive muscle relaxation (Jacobson, 1938), visual imagery, paced breathing, or meditation.

The purpose of the present study is to determine the influence of relaxation, introduced at different phases of women's menstrual cycle on the length of their cycle as a whole, and on the day of ovulation. This is intended to be an indirect way of determining the influence of relaxation on specific hormone levels.

There is a clear advantage to the active relaxation techniques because their effects is known to usually be longer lasting, and also because they can be exported from the laboratory. In the present study, a combination of instructions for breath mindfulness, and relaxing music seemed to be the fastest and most effective way to reach a desired relaxed state.

STATEMENT OF THE PROBLEM

In the present study, it is hypothesized that the “treatment” variables, i.e., stress-reducing relaxation, and music, will regulate or make predictable:

- 1) the cycle duration, and
- 2) the day of ovulation so as to make it possible to regulate the first (1) and predict the second (2).

Furthermore, it is hypothesized that the possible relationship between stress and estrogen levels may make the effects of relaxation comparable in some way to those of exogenous estrogen, thereby supporting the theory that it allows the body to promote producing its own.

To accomplish this, comparison is made between:

- the variation in the length of the cycle on the third month (when relaxation is or is not administered), and a baseline of the two first months (where no experimental manipulation is done), versus
- variation in the day (in the cycle) of ovulation and in the same two baseline months.

SUBJECTS

The Ss were forty Hunter college women psychology students ranging in age between eighteen and thirty. Only those Ss with a very regular menstrual cycle served, i.e., every month the same cycle length, or up to two days off, not more. Based on two month base line recordings by the participants.

INSTRUMENTS

The biofeedback device used was the Ultra Mind unit that measures Electro-Dermal Activity (EDA). The instrumentation consists of two parts: 1) a set of highly sensitive biosensor electrodes that measure the EDA change, and 2) a computer that recorded and interpreted the information transmitted to it by infra-red link.

The data were available in visual form via a video monitor, and in audio form, and also fed back to the computer. Feedback to the Ss was given visually as a line graph rising with relaxation. The sessions were recorded and then printed to track Ss' progress on each session.

Ovulation detection sticks (Mediplex, New Jersey) were used to detect luteinizing hormone (LH) in the urine stream. LH surges twenty-four to thirty-six hours prior to ovulation. When it surges, two lines appear in a window on the ovulation detector stick. Prior to ovulation only one line appears there. This measurement was done from day 8 of the cycle until the two lines were detected.

Tapes that were provided to the Ss began with a five-minutes-of-quiet period, followed by relaxation instructions. A few examples of relaxation instructions included: "Breathe comfortably and easily, focus on your exhalation, feel as if a balloon is expending in your stomach, continue to breathe like this for a while." These instructions take about five minutes. That is followed by relaxing music for another ten minutes. All together this procedure took about twenty minutes.

METHOD

The Ss were told that the purpose of the experiment is to monitor menstrual cycle. They were instructed on how, and when, to use the ovulation detection sticks (from day eight of the cycle, once a day—the second urine of the day—until two lines are detected), and they were given an informed-consent form to execute.

They were also given twenty-four ovulation detector sticks (for single use, disposable) and a short explanation from printed manufacturer-instructions on how to use the sticks. They were also provided with the chart to be filled out.

For two month the Ss only had to chart the days of menses, and the days when the two lines appeared on the ovulation detector stick. In the third month, on the first day of the cycle, the Ss were asked to telephone the experimenter who randomly assigned them to either the relaxation group, or the control group; and who instructed them to come to the lab, for three days in a row, either on day-three, four and five (early follicular phase), or on day-eight, nine and ten (late follicular phase) of the cycle, or on day-nineteen, twenty, and twenty-one (luteal phase) of the third cycle.

On the third month, the biofeedback device sensors were attached to the Ss and, if they were in the experimental group, they were made to listen to the tape. The five first minutes were quiet and were used as a baseline. Just prior to the presentation of the relaxation instructions, they were told that they are going to be given instructions on how to relax, and that the more they relax, the higher the line they were seeing on the computer monitor would rise.

From the fifth minute to the tenth minute of the session, instructions were given on how to relax with paced respiration. The last ten minutes, they heard relaxing music and they were asked to continue to breathe the way they had been taught.

The control group Ss were also connected to the biofeedback device, but the tape was not turned on. Each session lasted twenty minutes.

RESULTS

There were twelve Ss in the early follicular-stage group, and twelve in the luteal-stage group. There were ten Ss in the late follicular-stage group. The measurements were those that the biofeedback device manufacturer called EDA (Electro Dermal Activity) units. These units were translated to resistance units (micro-ohms) with the help of formulas provided by the manufacturer (see table 1).

The ohm units were then translated into μSiemens- units of conductivity (see tables 1, and 2). Measurements were based on data time-sampled every fifteen seconds during the twenty minute sessions. These data were then averaged over five minute periods, resulting in four such averages per session: minute-zero to five, five to ten, ten to fifteen and fifteen to twenty.

Complete data include three sessions and a data sheet in which ovulation day was detected for three consecutive months (two base line months and the third month when the experiment was conducted), as well as data of the day the period occurred over the same period of time. Some participants had only results for the length of the cycle, and did not record the day of ovulation.

RELAXATION RESULTS

Comparing the conductivity over the 3 sessions in all 3 experimental groups and the control group (Figures 1-9). It was found that when looking at the conductance of all 3 sessions together, the lowest conductance was found to be at the luteal phase. (Figures 1, 1a). When looking at each session individually for all 3 experimental groups we can see how at session 1 there is no significant difference between the conductance at the early follicular stage and the late follicular stage and even the luteal stage, but as we go to the next sessions the difference between the early follicular stage and the late follicular stage and the luteal stage deepens. (Figures 2-4). Conductivity over the 3 sessions individually in the 3 experimental groups is showed in Figures 5-7.

For the twelve Ss in the early follicular stage, five (41.67 percent) showed a consistent improvement between the sessions. Two did not improve at all, and their conductivity rose from session to session. Five Ss had equivocal results—decreasing the conductivity in one session, and augmenting it in the other.

For the ten Ss in the late follicular stage, eight (80 percent) decreased their conductivity from the first session to the next two sessions. One had mixed results, and one increased her conductivity from the first session to the other two sessions.

In the luteal experimental group, eight (66 percent) of the twelve Ss showed a decrease in conductivity from the first session to the next two sessions; three had mixed results, and one increased her conductivity from session to session. The difference in conductivity from session to session is shown in percentages:

- Conductivity on session two, less conductivity on session one, divided by conductivity on session one, times 100.

The procedure was repeated for the third session (see Figure 10).

A one-way ANOVA was used to test the significance of the difference in the changes in conductivity between the three experimental groups. When all the participants were compared in the three experimental groups including participants for whom Biofeedback was ineffective, i.e., whose conductivity did not decrease over the three sessions, $P(F_{2,65} = 1.31) > .05$; but when one extreme value was omitted from each experimental group, a change was noted: $P(F_{2,61} = 4.16) < .05$.

In order to pin point what contributed to significant difference found between the different experimental groups, a comparison between pairs of experimental groups was done. Between the early follicular stage, and the luteal stage: $P(t = 2.21) < .05$, df = 37; and between the late follicular stage, and the luteal stage: $P(t = 0.41) > .05$, df = 39; and between the early follicular stage, and the late follicular stage: $P(t = 2.48) < .05$, df = 37.

In order to find out if there is a difference in the change of conductivity from the first session to the second and from the first session to the third session, separately in the three experimental groups, an ANOVA was done for each the two sessions separately—session 2—yielded: $P(F_{2,30}= 3.62) < .0.5$. For session, 3: $P(F_{2,29} = 1.63) > .05$.

When looking more closely at all the experimental groups on session two, between the early follicular and the late follicular stages, we find that $P(t = 2.59) < .05$, df = 12, and between the late follicular and the luteal stage, $P(t = 1.3) > .05$, df = 14. Then, between the luteal and the early follicular stage, $P(t = 1.72) > .05$, df = 16.

One-way ANOVA was also used to analyze the conductivities obtained in the three experimental groups, as well the control groups. There were some very extreme values that were above 4.5 micro-Micro-Siemens(i.e. 11 micro-Siemens, and 9 micro-Siemens). However, regardless of whether these values were included or excluded, statistically significant results where obtained.

When these extreme values were included, however, the variance was very high—sometimes twice the value of the mean of the sample. A comparisons of all three experimental groups including extreme values yields: $P(F_{2,405} = 11.8) < .05$. For averages and variance, see Table 3.

When parceling out the results for sessions, it was found that for session 1: $P(F_{2,133} = 4.01) < .05$. Results for session 2: $P(F_{2,133} = 5.77) <.05$, and, for session 3, $P(F_{2,133} = 6.17) < .05$.

Since there was a relatively small sample, extreme values have a great influence on the variance. Therefore, extreme values were omitted—those above 4.5 micro−Siemens. A one way ANOVA for all three sessions on all experimental groups then yielded: $P(F_{2,364} = 4.31) < .05$.

ANOVA for each session separately:

Session 1: $P(F_{2,121} = 0.32) > .05$; Session 2: $P(F_{2,213} = 3.43) < .05$; Session 3: $P(F_{2,121} = 8.92) < .05$ (see Figure 2, Table 5).

Further comparisons were made between paired experimental groups: early follicular/late follicular, early follicular/luteal, late follicular/luteal:

Session 2, early follicular/late follicular: $P(t = 1.34) > .05$, df = 44; early follicular/luteal: $P(t = 2.42) < .05$, df = 48; and late follicular/luteal: $P(t = 1.26) > .05$, df = 74 (see Figure 3, Table 6).

Session 3, early follicular/late follicular: $P(t = 3.13) < .05$, df = 65; early follicular/luteal: $P(t = 3.39) < .05$, df = 54; late follicular/luteal: $P(t = 0.01) > .05$, df = 56 (see Figure 4, Table 7).

The previous statistical tests were for the changes in conductance *between* the different experimental groups. In order to test the significance of changes from session to session in each experimental group separately, a t-test for differences was used. Two measurements were taken for each participant, one for the difference between session two and session one, as expressed in percentage, and one for the difference between session three and session one, as expressed in percentage.

In the early follicular phase group (data from three participants who did not relax, and who had at least one session) the conductance doubled as compared to the first session. For both sessions averaged together: $P(t = 0.68) > .05$, df = 23. Excluding the three participants with extreme values: $P(t = 2.11) < .05$, df = 17. A t-test was also used to compare Ss sessions separately for session 2: $P(t = 1.23) < .05$, df = 8; and session 3: $P(t = 1.66) < .05$, df = 8.

Late follicular phase, with one extreme value for both sessions: $P(t = 0.96) > .05$, df = 19; without the one extreme value, both sessions: $P(t = 7.8) < .05$, df = 17; for session two only: $P(t = 11.67) < .05$, df = 8; and session three only: $P(t = 4.68) < .05$, df = 8.

Luteal phase with one extreme value: $P(t = 1.71) > .05$, $df = 23$; without one extreme value, both sessions: $P(t = 6.04) < .05$, $df = 21$. Session two: $P(t = 3.27) < .05$, $df = 10$; and session three: $P(t = 5.32) < .05$, $df = 10.15$.

ANOVA was also used to analyze the Conductances for each experimental group, the results taking into account the large variances between the participants: any participant with an average of over 4.5 micro-Siemens per session was omitted.

The early follicular phase comparison between the three sessions yielded: $P(F_{2,117} = 0.87) > .05$ (two Ss were omitted) (see Figure 5). Late follicular phase comparison between the three sessions: $P(F_{2,93} = 8.47) < .05$ (two Ss were omitted) (see Figure 6). Luteal phase comparison between the three sessions: $P(F_{2,144} = 10.00) < .05$ (see Figure 7).

The next test was used to see if relaxation was achieved during the different segments of the sessions. Each session was divided into four segments of five minutes to see if there was a change in conductance from the first five minutes (the base line), to the next segment, minutes five to ten (with paced breathing instructions), minutes ten to fifteen, and the last (music) segment fifteen to twenty.

The data consist of percent differences. Each segment was subtracted from the baseline of that session, and then divided by the value of the baseline, times one-hundred (see Figure 11, 11a, 11b).

A t-test was used to see whether the subsequent segments were significantly different from their base line: It showed that in the early follicular phase, for the segment of five to ten minutes: $P(t = 1.38) > .05$ $df = 35$; for the segment of ten to fifteen minute: $P(t = -0.60122) > .05$, $df = 35$; and for the segment of fifteen to twenty minutes: $P(t = 0.32) > .05$, $df = 35$.

In the late follicular phase for the segment of five to ten minutes: $P(t = 0.30) > .05$, $df = 27$; for the segment of ten to fifteen minutes: $P(t = 0.38) > .05$, $df = 27$; and for the segment of fifteen to twenty minutes: $P(t = 0.45) > .05$, $df = 27$.

In the luteal phase, for the segment of five to ten minutes (over all three sessions) . $P(t = 0.66) > .05$, $df = 35$; for the segment of ten to fifteen minutes: $P(t = 1.92) < .05$, $df = 35$; and for the segment of fifteen to twenty minutes: $P(t = 1.76) < .05$, $df = 35$.

The average change in conductivity in the early follicular phase, from the first five minutes to the five-to-ten minute segment, was an increase of 7.79 percent; it was a 5.85 percent decrease to the segment of ten-to-fifteen minutes, and a 3.30 percent decrease to the segment of fifteen-to-twenty minutes.

The average change in conductivity in the late follicular phase from the first five minutes to the five-to-ten minute segment was an increase of 1.7 percent, to the segment of ten-to-fifteen minutes, a 3.09 percent increase, and to the segment of fifteen-to-twenty minutes, 4.26 percent increase.

The average change in conductivity in the luteal phase from the first five minutes to the five to ten minute segment was an increase of 4.8 percent, to the segment of ten to fifteen minutes, -14.8 percent, to the segment of fifteen to twenty minutes, -16.27 percent (see Figure 17).

When looking only at the segments where the conductivity decreased, as compared to the first segment—minutes zero to five—we find that in the early follicular stage the average decrease is 33.9 percent (48 percent of 108 segments showed a decrease in conductivity). In the late follicular stage the average decrease in conductivity between the segments is 25 percent (40 percent of the 90 segments showed a decrease in conductivity). In the luteal phase the decrease in conductivity between the segments was 41.42 percent (62 percent out of the 108 segments showed a decrease in conductivity) (see Figure 21).

THE CONTROL GROUP

The control group had only six Ss and only three of them gave back the results of the cycle length and the ovulation day. ANOVA was conducted on the three experimental groups and the two control groups (there were only two control groups one for the early follicular stage and one for the luteal stage) . The control groups had higher conductance values than the experimental groups: $P(F = 8.16) < .05$, $df_1 = 3$, $df_2 = 460$. There is no significant difference between the late follicular stage group and the control group: $P(F = 0.46) > .05$, $df_1 = 1$, $df_2 = 174$.

There is a significant difference between the control group and the early follicular stage group: $P(F = 10.76) < .05$, $df_1 = 1$, $df_2 = 210$; between the luteal phase group and the control group: $P(F = 25.6) < .05$, , $df_1 = 1$, $df_2 = 210$. (Figure 8a)

The conductivity decreases, in the control group, between the sessions from 2.47 in the first session to 1.98 in the second session, to 2.04 in the third session. But, this decrease is not statistically significant (Figures 8a, 8b, 9). There are also some changes in conductivity in each session separately, when looking at changes in conductivity in the segments after the first five minutes. On the average the change is an increase of 7.67 percent in conductivity.

When examining only the segments where the conductivity decreases, one finds a decrease of 25.57 percent. Twenty-eight out of 50 segments showed such a decrease. The initial value in micro-Siemens units (on session one) showed a relationship to the decrease in conductance in the following two sessions, each session taken separately. The initial conductance was also related to the average decrease in conductance of the two sessions.

Early follicular stage, session one conductance in micro-Siemens units, and average conductance decrease yielded: $P(t_{rxy = -0.51} = 1.87) < .05$ df = 10 (see Figures 12 and 13); the original conductance in micro-Siemens units on session one, and decrease in

conductance in session two: $P(t_{rxy = -0.29} = -0.958) > .05$, df = 10 (see Figures 11); the original conductance in session three: $P(t_{rxy = -0.47} = -1.68) > .05$, df = 10 (see Figure 12).

Late follicular stage, session one, conductance in micro-Siemens units correlation with the average conductance decrease: $P(t_{rxy = -0.357} = -1.08) > .05$, df = 10; the original conductance in micro-Siemens units on session one, with the decrease in conductance in session two: $P(t_{rxy = -0.538} = -1.8) > .05$, df = 8 (see Figure 13); the original conductance in micro-Siemens units on session one, and the decrease in conductance in session three: $P(t_{rxy = -0.111} = -0.31) > .05$, df = 8 (see Figures 14, 15).

Luteal stage, session one, conductance in micro-Siemens units and average conductance decrease: $P(t_{rxy = -0.497} = -1.81) > .05$, df = 10; the original conductance in micro-Siemens units on session one, and decrease in conductance in session two: $P(t_{rxy = -0.0997} = -0.316) > .05$, df = 10 (see Figure 15); the original conductance in micro-Siemens units on session one and decrease in conductance in session 3: $P(t_{rxy = -0.723} = -3.3) < .01$, df = 10 (see Figures 16, 17).

CHANGE IN CYCLE LENGTH

A t-test was used to determine significance of differences in the length of the cycle on the third month, as compared to the average length of the cycle in the two base line months. The second test was to determine the correlation between the change in conductance on during each session (from the first five minutes of base line) and the number of days that the cycle changed. A further correlation was sought between the change in conductance in during each session, and the change in the day of ovulation.

Correlations were obtained between average change in conductance in each session, from the initial first five minutes, and the change in cycle length; between the segments in which the conductance decreased, and the cycle length. Correlation was also sought between all these and change in ovulation day.

A t-test for differences was conducted on the data collected during the three months of the experiment: the base line for the length of the cycle and for the day of ovulation from the first two months.

The data for the experiment were collected on the third month and counted as "different" only if the third month was longer then the longest cycle (of the base line months), or shorter then the shorter cycle.

This procedure prevailed on all three experimental groups for both cycle length and ovulation day (from the beginning of the cycle). If the third month was found to be different from the base line months, only then were the results of the base line averaged and the third month results subtracted from that average.

Early follicular stage, all participants, cycle length: $P(t = 3.85) < .01$, $df = 11$ (Table 11. Figure 18); ovulation: $P(t = 1.65) > .05$, $df = 6$; late follicular, cycle length: $P(t = 0.97) > .05$, $df = 6$ (Figure 19); ovulation: $P(t = 0.84) > .05$, $df = 6$; luteal, cycle length: $P(t = 3.29) < .01$, $df = 10$ (Figure 20); ovulation, day: $P(t = 1.77) > .05$, $df = 7$.

Correlation was sought between change in conductance (as the assumption was that relaxation would influence the cycle length) and change in the cycle length—three such correlation coefficients were obtained for each experimental group: The first one was between the change in conductance from the first session to the second session and the change in the cycle length. The second one was between the change in conductance between session one and session three, and the change in cycle length, and the third one was between the average change in conductance from the first session, and the change in cycle length. A similar analysis was also done with ovulation day changes.

Early follicular phase correlation between decrease in conductance (sessions two and three), and the change in the length of the cycle (Figure 21): Session two: $P(t_{rxy = -0.21} = -0.67) > .05$, df = 10; session three: $P(t_{rxy = -0.59} = 2.31) < .05$, df = 10; ovulation day, session two: $P(t_{rxy = -0.19} = 0.43) > .05$, df = 5; with session 3: $P(t_{rxy = -0.25} = 0.58) > .05$, df = 5.

In this experimental group there were a large number of participants who did not relax. Therefore, another analysis was done that separated the relaxed participants from the ones who did not relax. Relaxed: $P(t_{rxy = -0.72459} = -1.8211) < .05$, df = 3, not relaxed: $P(t_{rxy=0.741} = 2.46) < .05$ df = 5; with the change in conductance on session 2, relaxed: $P(t_{rxy=-0.455} = 0.88) > .05$, df = 3; not relaxed: $P(t_{rxy =0.481} = 1.227) > .05$, df = 5.

Late follicular phase—correlation between change in conductance (sessions two and three) and change in cycle length, session two (Figure 22): $P(t_{rxy =- 0.2} = -0.456) > .05$, df = 5; session three: $P(t_{rxy = -0.47} = -1.06) > .05$, df = 5. There was only one participant who reported that the ovulation day changed.

Luteal phase—correlation between change in cycle length and average change in conductance on session 2 (Figure 23): $P(t_{rxy = -0.45} = -1.59) > .05$, df = 10; correlation with session three: $P(t_{rxy = -0.038} = -0.12) > .05$, df = 10; No correlation was sought with ovulation day since change in ovulation day was not notable.

A multiple regression analysis was conducted to see if the average change in conductance on sessions two and three from the first session can explain the change in the cycle length. No significant results were found in any of the experimental groups. (all the participants are included).

A correlations also obtained between the average decrease in conductance in each session, from the first five minutes of each session (for all three sessions), and the change in the length of the cycle, and the change in the day of ovulation. There were two correlations that were obtained between the change in cycle length (on the third month), from the base line (the first two month) and

- a correlation between the segments that showed a decrease in conductivity (from the first five minutes of base line of each session, using an average value), and
- between the average of total changes in conductivity (all segments included), in each session (from the first five minutes of base line of each session).

Early follicular stage—the correlation between the average of the segments that decreased in conductivity and the change in the length of the cycle was -0.47. $P(t_{rxy = -0.47} = -1.6) > .05$, df = 9. Correlation between average change of all the segments from the first five minutes and change in the length of the cycle was -0.55. $P(t_{rxy = -0.55}\ (r_{xy}) = -2.07) < .05$, df = 10. (Figure 24)

Late follicular stage—the correlation between the average change in conductivity of the segments (from the first five minutes of each session) that showed a decrease in conductivity, and the change in the length of the cycle was 0.597.
$P(t_{rxy = 0.597} = 1.66) > .05$, df = 5. (Figure 25)

Luteal stage—correlation between the average of the segments that decrease in conductivity and the change in the length of the cycle was -0.194. $P(t_{rxy = -0.194} = 0.62) > .05$, df = 10; with average change in conductivity from all segments $P(t_{rxy = -0.066} = 0.2) > .05$, df = 10. (Figur

Early follicular stage—the correlation between the average decrease in conductivity and the change in the day of ovulation was -0.218. $P(t_{rxy = -0.218} = 0.497) > .05$, df = 5.

Late follicular stage—the correlation between the average decrease and conductivity and the change in the day of ovulation was 0.89. $P(t_{rxy = .89} = 4.36) < .05$, df = 5.

Luteal stage: the correlation between the average decrease in conductivity and the change in the day of ovulation was 0.096. $P(t_{rxy = .096} = 0.236) > .05$, df = 6.

A multiple regression analysis was conducted to see if the change in conductance during sessions one, two and three from the beginning of each session, the five first minutes, to the next three segments of five minutes (five to ten, ten to fifteen, fifteen to twenty) can explain the change in the cycle length. The only experimental group in which the change in conductance during the session explained the change in the cycle length was at the early follicular stage where 96.6% of the results can be explained by the 3 segments of each of the relaxation sessions. The more relaxed the participant was the longer was the menstrual cycle. (Figure 27) There were no significant results when regression analysis was conducted between the relaxation segments and the change in the day of ovulation.

DISCUSSION

The first part of the discussion will explain the results of the relaxation sessions, i.e., how the electrical conductance changes from session to session, and what happens to tension/relaxation within one session. This section will also explain the differences that were found between the relaxation patterns in the three different parts of the cycle, i.e., the early follicular, the late follicular and the luteal phases. This will be followed by examination of the influence of relaxation on the length of the cycle, and on the ovulation day.

RELAXATION: TESTING THE EFFECTS OF PACED BREATHING

The results of the biofeedback sessions are expressed in conductance units, micro-Siemens. A higher conductance indicates higher stress levels. Here are the results for forty Ss, twelve in the early follicular phase, ten in the late follicular phase, twelve in the luteal phase, and six in a control group:

- There is no significant improvement between the four segments of the sessions for the early follicular phase of the cycle, and in the late follicular stages.
- No significant changes were noted in conductance, nor in percentage of change from the beginning of the session (minutes zero to five), to the next three segments in which the instructions for paced respiration were introduced (minutes five to ten), and where the music was introduced (minutes ten to twenty).
- A decrease in conductance was only detected in the luteal phase (see Figure 17).

Examining only the segments where there is a decrease in conductance, there is a difference between the stages of the cycle. It seems that the lowest decrease in conductance is in the late follicular stage. Not only is the average decrease lower (as

compared to the early follicular stage and the luteal stage), but the percentage of segments in which there was a decrease is also the lowest.

In the early follicular stage, the average decrease in conductance (only in the segments that showed a decrease) is higher then at the late follicular stage, and the percentage of segments that show a decrease is higher then in the late follicular stage:

- The highest average decrease in conductance was found in the luteal phase and the percentage of segments that showed a decrease in conductance was the highest.

The results obtained in the late follicular stage are not different from the results obtained in the control group. Each participant came for three sessions lasting twenty minutes from to end:

- The results of the relaxation instructions were greater in the luteal experimental group, than in the other experimental groups. This shows the influence of the cycle on the ability to relax, and it constitutes a heretofore unknown physiological coincidence.

There is a relationship between the phase of the cycle and the EDA response: The response to stress was strongest in some of the Ss at the premenstrual period (late luteal phase) (as noted also by Asso, and Braier, 1982; Chattopadhyay, 1983; Asso, 1986). Indeed if we look at the data by Davydov, Shapiro, Goldstein, et al. (2005) (private communication, not appearing in the article), we can see that:

- at the luteal phase, a work day is more stressful than at the early follicular phase, but
- a rest day has more of an impact in the luteal phase than in the early follicular phase.

There is little, if any, previously published evidence of the degree to which stress and relaxation are affected physiologically by entirely different hormone status in women.

> In fact, the present data suggest that stress and relaxation may not constitute opposite poles of a single continuum, as previously thought.

The luteal phase is more prone to influence one way or another.

There is another way to interpret the data: Instead of comparing the sessions individually and comparing four segments of one session**,** one can ask if there is an improvement from the first session to the second, and to the third session? Are the Ss more relaxed the second time they come for their biofeedback session than at the first session? The answer is a "conditional yes."

There are significant differences between the first, second, and third session. These are dominant in the luteal phase, but not so in the early follicular phase. At the late follicular phase (ovulatory phase) again this difference is significant.

As mentioned previously, 41 percent of the Ss relaxed at the early follicular stage but when we look at the late follicular stage, 80 percent of the participants relaxed. Then, at the luteal phase, 66 percent relaxed—as defined by decreased conductance between the first session, and the two following sessions.

Not only are the participants more relaxed from session to session, but the data show more consistency insofar as the variance is considerably lower progressing towards the third session. This decrease in variance strongly suggest a learning process as the behavior becomes more focused. All these differences appear in the luteal phase and the late follicular phase:

In the early follicular phase, the learning effect is much less pronounced but the difference between sessions is not significant. That difference between the sessions was obtained in two ways: repeated measurements were made between the second session and the first session, and between the third session and the first session. Similarly, analysis was also conducted on the Conductance data.

The resulting analyses suggest some special facilitation for learning to relax at the late follicular, luteal phase, yet not at the early follicular phase. It is possible that this is just habituation. But that raises other questions about female hormone status facilitating habituation that cannot be addressed here.

When comparing the three phases of the cycle in Siemens-units data, there is no significant difference between them on the first session. But, there is progress to the second and third session, when the difference between the early follicular stage, the late follicular stage, and the luteal stage become more pronounced.

On the third session there is no significant difference between the late follicular stage and the luteal stage, but there is a significant difference between the early follicular stage in the two other experimental groups.

Similar findings were reported in a study by Gomez-Amor, Martinez-Selva, Roman, et al. (1990). The highest level of habituation to a stimulus as measured by EDA, was found to be in the late follicular stage. In the experiment by Goldstein, Jerram, Poldrack, et al. (2005), cited in the introduction, arousing stimuli seem to have more effect in the HPA axis on the early follicular stage. It was also found there that at the late follicular stage the HPA axis reaction is tempered by the higher levels of estrogen contributing to less excitability.

It seems from all the experiments mentioned above (Davidov, Shapiro, Goldstein, et al., 2005; Gomez-Amor, Martinez-Selva, Roman, et al., 1990; Goldstein, Jerram, Poldrack, Ret, al., 2006), and in the experiment of this thesis that:

- change—no matter in which direction, towards more stressed or more relaxed—happens in the same phase of the cycle: the late follicular and the luteal stages being the most prone to changes.

The question is why?

One explanation is that there might be a feedback mechanism that works at the early follicular phase and not at the late follicular, nor at the luteal phase. The feedback mechanism described by Altemus, Redwine , Leong, et al. (1997), comes to mind: that at the early follicular stage there are more glucocorticoid receptors than at the luteal phase, thus helping relaxation at the follicular stage.

At the early follicular stage the levels of estrogen are at their lowest, as ovulation approaches, these levels are much higher. After ovulation at the luteal stage, estrogen levels are still high, but just prior to the menses these levels of estrogen drop again.

It would seem that there are two "calming" mechanisms, the feedback loop described by Altemus, Redwine, Leong, et al. (1997), and the mechanism described by Goldstein, Jerram, Poldrack, et al. (2006), that take over once the estrogen levels are higher. Their model explains why we are able to relax better on the late follicular and the luteal phase, then at the early follicular stage.

A two-phase model is also suggested by the present findings:

- At the beginning of the cycle there is the glucocorticoid loop that arises when stress rises, and it serves as a buffer.
- In the second half of the cycle, when estrogen levels are higher, the HPA axes has a general calming effect, but not a buffering effect.

Perhaps, the HPA axes permit more fluctuations for arousal and for relaxation.

What can the reason be for a strong tendency toward homeostasis at the early follicular stage? When looking at the ability to relax at the late follicular stage (days eight to ten), and the luteal stage (days nineteen to twenty-one), the common denominator is higher levels of estrogen as compared to the early follicular stage. We can also see that the habituation effect is not limited to in one session, but it seems to progress even between the sessions.

The relaxation in this experiment was accomplished by mindful breathing. Breathing is not declarative insofar as it doesn't deal with content, but rather with the procedure, how to breathe according to instructions given. Once the instructions are followed the breathing exercise becomes second nature and the learning becomes implicit, meaning it can not be put into words.

If the glucocorticoid feedback mechanism works at the early follicular stage the women are kept at a more relaxed level (any aggravation might be counteracted by this feedback mechanism). Therefore the biofeedback sessions might not teach them anything as they are already relaxed.

It appears from the data collected here that the facilitation is more pronounced at the late follicular phase and at the luteal phase. In an experiment by Fisher, Hallschmid, Elsner, et al. (2002), habituation was observed after giving two sessions of learning on the same day and one on the next day. The larger improvement was attained on the next day. In an experiment by Gomez-Amor, Martinez-Selva, and Roman (1990), it seems that not only is the EDA reaction the highest at the ovulatory stage the ability to habituate is also the highest at that stage.

THE RELATION BETWEEN INITIAL CONDUCTIVITY AND CONSEQUENT RELAXATION

Correlations were obtained between the initial conductivity on the first session and that on the following sessions to determine if the initial conductivity could in any way be a good predictor of future success in relaxation.

Lacey, and Lacey (1962), reported that the initial level of an autonomic response is positively related to the degree of change that can be detected. They called this the "Law of initial value." This phenomenon was also found in the present study: the higher the initial conductivity the more the participant relaxed proportionally (noted as a decrease in Conductance units).

- A significant correlation obtained between the initial conductivity on session one and the decrease in conductivity on session three in the luteal phase.

There was no such significant correlation found in the early follicular stage. (Figure 12)

- In the late follicular stage there was again a significant correlation between the initial Conductance values and the decrease in conductivity in the second session.

In the early follicular phase, as previously noted, only 41 percent of the participants relaxed. Therefore, it was interesting to see the relationship in this experimental group between the relaxed and the non relaxed participants.

Once the relaxed and the non-relaxed Ss data were separated, a much clearer picture emerged: There is a significant relationship between the initial value in Conductance units on session one, and an increase in conductivity in session two, and three (in the non relaxed sub-group).

In the relaxed sub-group, the correlation was high but not significant. What emerges from this division is that the relaxed group data are negatively correlated with the change in conductivity—the higher the initial conductivity the more we can expect a decrease in conductivity in the next sessions. This complies with Lacey and Lacey's "Law of initial value" (1962). In the group that failed to relax, the higher its initial conductivity the more this conductivity increase in the following sessions.

- The present study also finds that a correlation between the initial conductivity value and the outcome of a biofeedback session is also dependant on the phase of the cycle.

THE INFLUENCE OF RELAXATION ON THE LENGTH OF THE CYCLE AND ON OVULATION

- The results of this study show that biofeedback does influence the total length of the menstrual cycle in both the early follicular stage and the luteal stage.

Parenthetically, a curious finding in the present study is the heretofore undocumented difficulty of finding regularly cycling women: A change in the length of the cycle is defined as a change from the base-line—the two first months, if the length of the cycle exceeded the upper and the lower limits of the two month base line, i.e., when:

a) a participant had a base line cycle-length of twenty-six days on the first month, and
b) she had a base line cycle length of twenty-eight days on the second month, then, it was counted as a difference but only if the
c) participant had twenty-five days or less, or twenty-nine days or more on the third month.
d) If there where more then two days difference, in the length of the cycle, on the base line month (month one and two). The participant was not asked to come back on the third month, as the participant was not considered to be regular.

As previously noted, only forty of the more than one-hundred women who signed an informed consent form actually completed the study. Some Ss just dropped out, but others who wanted to complete the study and had attested to their being regular were found not quite as regular as they had originally thought they were, and they had to be disqualified.

An average change in conductivity from the beginning of each session was calculated for each participant. This value was correlated with the change in the cycle length.

- In the early follicular stage: there was a significant negative correlation between the average changes in conductance during each session, and the

change in the cycle length: the more the participants relaxed the longer the cycle became.

- In the late follicular phase, the correlation between the change in conductivity, and the change in the day of ovulation was statistically significant: The positive correlation indicated that the more the participants relaxed, the earlier the ovulation occurred.

Changes in the length of the cycle from the two first months baseline was not significant, but as not every participant relaxed belonging to any experimental group does not guarantee that relaxation has occurred. Therefore the correlation results between relaxation and change of cycle length/ ovulation and the multiple regression results are the most important if found to be significant.

In the late follicular stage, when ovulation is very near, there is no detectable influence of the biofeedback session on the length of the cycle (only three out of the seven participants had any change in the cycle length). In the luteal phase, in the experimental group it was found that the length of the cycle changed on the third month. But, there is no significant correlation between the average change in conductance, over the individual sessions (as compared to the five first minutes of each session), and the change in cycle length.

Menses start twelve to fourteen days after ovulation, so if there were a change in the length of the cycle, then we could assume that there might have been a change in the ovulation day as well. But, the LH detection kits that predict ovulation in the next twelve to thirty hours have a large “window” so that there might still be a difference in the ovulation day that cannot be pinpointed.

The percent of Ss actually capable of relaxing is relatively small at the beginning of the cycle. This may be partly due to the fact that the glucocorticoid feedback loop is stronger at that part of the cycle.

Considering the number of segments where participants relaxed, as compared to the first five minutes of every session, early follicular-stage participants relaxed on only fifty-two segments (48 percent) out of one-hundred eight segments. In the luteal phase 62 percent out of the one-hundred eight segments showed relaxation (see Figure 21).

It stands to reason that if the procedures result in relaxation, then we can also influence their cycle that way:

- there was a significant change in the total length of the cycle, yet, not in the day of ovulation.

When relaxation did occur in the second half of the cycle, there was no expectation of change in the day of ovulation and, indeed, no such change was found. But, there was a change in the total length of the cycle (but not correlated to the relaxation). This group, the luteal experimental group, was the only one that showed a change in conductance in the individual sessions, as well as habituation between the sessions.

IN CONCLUSION

Relaxation with biofeedback, at least the set of procedures employed in this study, works in a different way at different stages of the menstrual cycle. This heretofore unreported phenomenon may need to be taken into consideration in any therapeutic biofeedback treatment plan for women.

Perhaps biofeedback treatment failures can be avoided by- and treatment outcome results could be optimized by starting it in with women just after ovulation.

Furthermore, these results also indicate that in any study of relaxation with or without biofeedback, one cannot justify the common procedures where men and women are typically randomly assigned to treatment and control groups without regard to the phase of the menstrual cycle of the women at the time of the study. Likewise, initiating such treatment in a clinical setting also requires knowledge of the phase of the woman's menstrual cycle if one is to optimize treatment.

As it turns out, it is difficult to find "regular" women, and even in such women, a "simple procedure" such as relaxation can divert some women from their "regular" mode.

The purpose of this study was to see if the biofeedback/relaxation could lengthen the cycle and postpone ovulation. The procedure did indeed change the cycle: But in some women the cycle lengthened, while in others it became shorter.

One reason for seeking a procedure that can control the cycle duration—it was hoped to lengthen it—and predict the day of ovulation is practical: The Jewish orthodox community adheres to certain guidelines pertaining to sexual marital relations. One of them is that these are permitted only after seven days free of "bleeding" and the woman has immersed in a ritual bath. This means that the earliest day for marital relations is day twelve, depending on the duration of bleeding.

Adhering to these laws can cause women to miss the day of ovulation by as little as one day, making conception difficult.

While biofeedback appears to influence the cycle, it can either shorten or lengthen it, but when looking at the ability to relax not just the mere fact that a participant had undergone a biofeedback session, then the picture is clearer. At the early follicular stage relaxed participants caused their cycle to be longer .

Biofeedback sessions seems also to be able to "stabilize" women, when talking about their relaxation ability. The data has showed some near-chaotic, large fluctuation in the conductivity on the first session. By the second and third sessions, variation decreases significantly—even this observation is cycle dependant: this ability to "focus" the behavior is more pronounced at the late follicular and the luteal stage.

Clearly, any relaxation/biofeedback procedure with women must take the menstrual cycle phase into account.

A further intriguing finding was the mostly false perception of regularity that many Ss reported: For the most part, women tend to overestimate their "regularity." Parenthetically, these findings explain why this study has taken so long to complete.

Relaxation can change the cycle and, paradoxically, so can stress both making it either longer or shorter in duration.

It would have been ideal to be able to have measured estrogen levels to see if there exists a more direct connection between relaxation and estrogen. Questions arise:

- Estrogen influences relaxation, but can relaxation increase estrogen?
- Could an intervention longer than three session, let's say, seven days in a row, stabilize the findings?
- Should relaxation/biofeedback be considered only in the luteal stage? After all, Ladisich (1977) found that the influence of the first session on subsequent sessions depend on the phase of the cycle in which the initial session was begun.

In that study (Ladisich, 1977), Ss were given a mild electric shock to the hand either eight days before their period, in the luteal phase, or on the day prior to the menses when hormonal levels are low. The dependant variable was heart rate.

If the first session took place in the luteal phase, then the second session did not show any different response—the heart rate remained the same. However, if the first session took place on the day just before the menses, then the second session was in the luteal phase and in that case, the heart rate was slower as a response to shock.

Unlike the present study featuring relaxation, their study induced stress. If indeed there are more receptors for steroids at the time of the menses, that fact would explain why there is greater habituation when the first encounter with a stressful event occurs then: That is because the elevated cortisol feedback loop would be activated.

The activation of such a feedback loop is thought to yield association between stressful stimuli and a more relaxed state. The next encounter on the luteal phase, therefore, would show greater habituation to the stimuli.

But in this study, there is no activated feedback loop as there is no stress. In this case habituation is greater when estrogen levels are higher.

This suggests a follow-up study based on the knowledge that relaxation/biofeedback is more effective at certain times in a woman's life. It should also be noted that perhaps diurnal changes need also be taken into account since into there is a surge in cortisol levels early in the morning.

- The significant negative correlation between the lengthening of the cycle and the ability to relax seems to support the present hypothesis that relaxation increases the overall cycle length but only if induced in the early follicular phase.
- The failure to find the same correlation with the day of ovulation, does not in itself mean that the ovulation day was not influenced by relaxation. But,

rather that it is possible that the diagnostic tool—ovulation sticks that were used—leave some margin of twenty-four hours for the ovulation to occur.

In pre-menopausal women, the ability to relax seems very clearly menstrual cycle-phase-dependent. Mastering relaxation should be achieved before a repetition of this experiment. At the early follicular stage it is difficult for Ss to reach relaxation, but a negative correlation between relaxation and the cycle length seems to indicate the more relaxed they became the longer the cycle became. Relaxation, therefore, should be first taught in the luteal phase of the cycle, to influence the duration of the cycle.

E D A	**Kohm**
EDA<4500	1071.706 -0.9766399*EDU+ .0002684*EDU^2 -2.24*10^(-8)*EDU^3
4500>EDA<7000	-341.5146+.1234316*EDU-.0000139*EDU^2+1.57*10^(-9)*EDU^3
7000>EDA<10100	25.4547+ .0737562*EDU-.0000189*EDU^2+2.26*10^(-9)*EDU^3
10100>EDA<1250	-37436.9 + 11.10762*EDU -.0011063*EDU^2+ 3.81*10^(-8)*EDU^3
12500< EDA	-25880+2.2993*EDU

Table 1 conversion table from EDA the units used by the device to resistance units in Kohm

E D A	**Kohm**	**micro-Siemens** **1000/kiloSiemens**
EDA<4500		13.5 ≤ micro-Siemens≤ 20
4500>EDA<7000		2.0 ≤ micro-Siemens≤ 13.0
7000>EDA<10100		0.9 ≤ micro-Siemens≤ 1.9
10100>EDA<12500		0.4 ≤ micro-Siemens≤ 0.8
12500<EDA		0.2 ≤ micro-Siemens< 0.3

Table 2 range of equivalence between EDA the units used by the device to conductivity units in micro-Siemens.

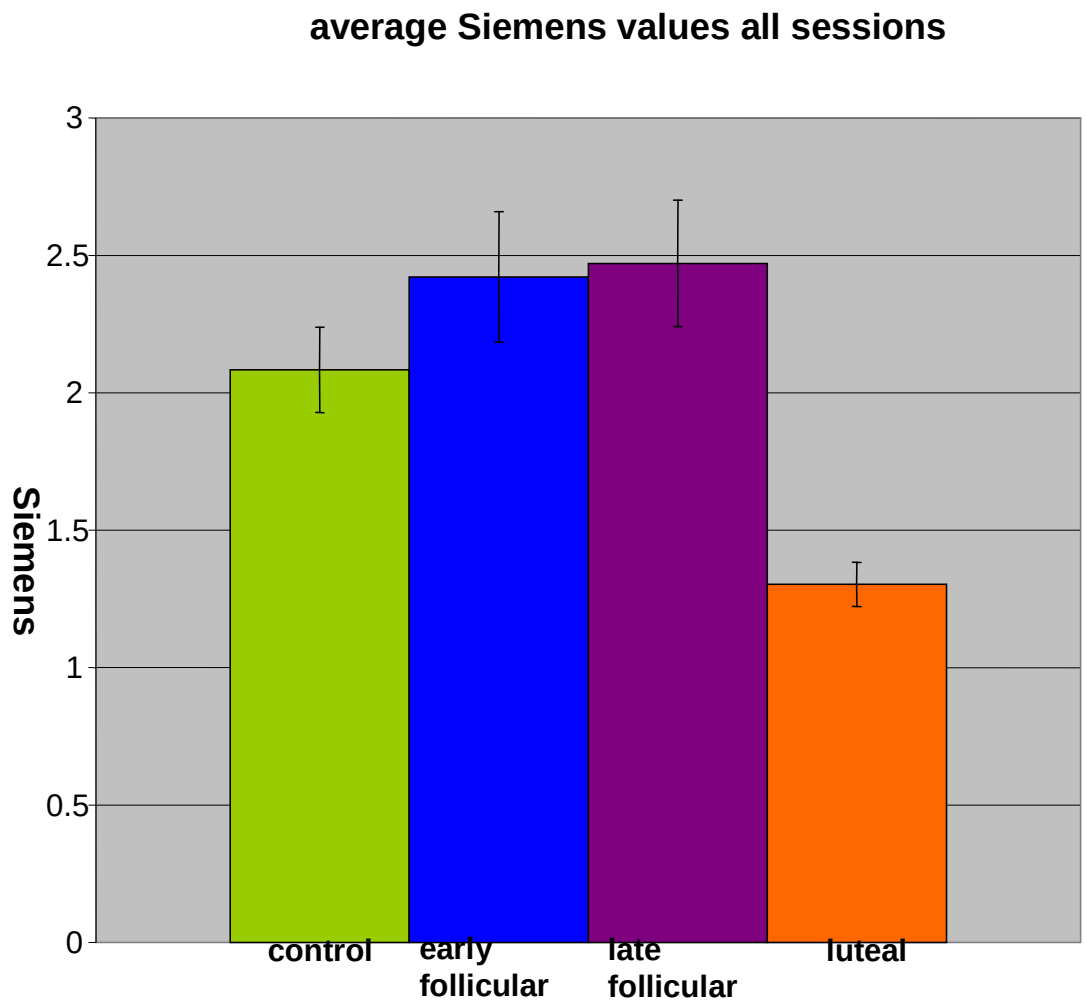


Figure 1: Means of all 3 experimental groups and control groups all 3 sessions in micro-Siemens (including extreme values)

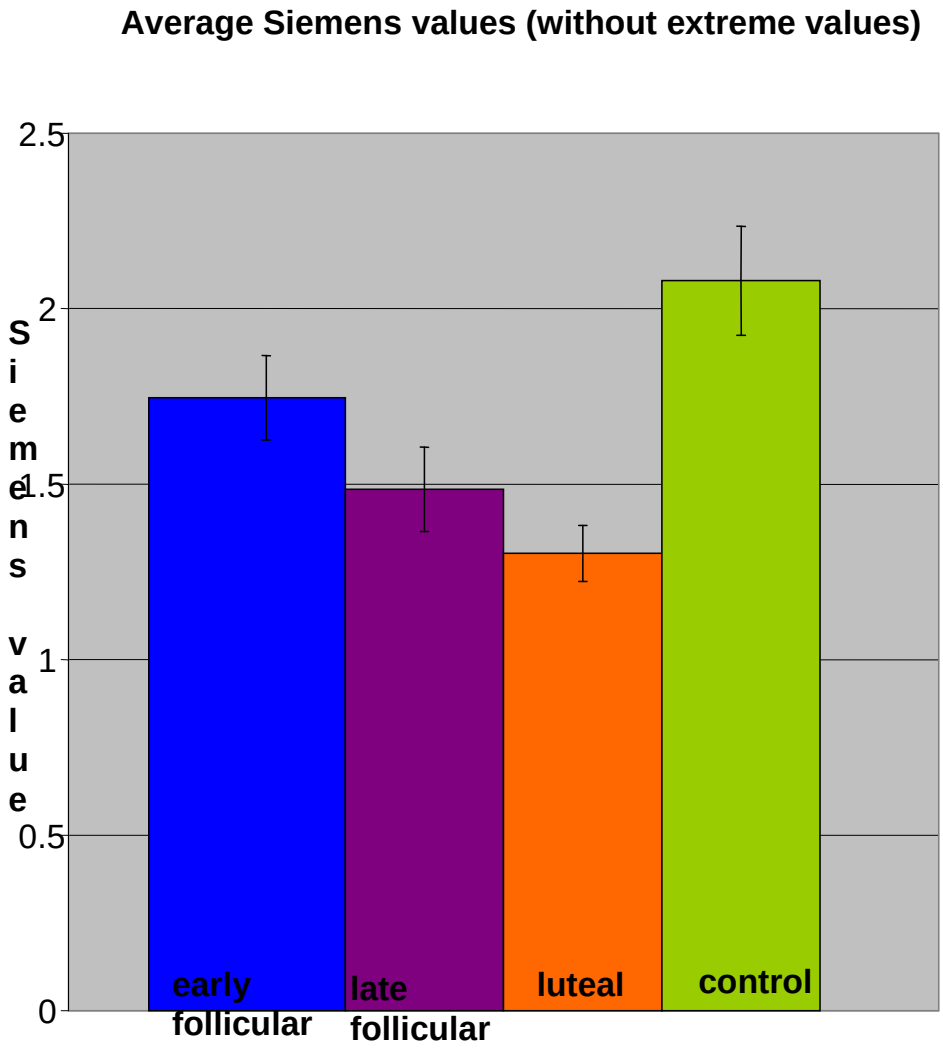


Figure 1a: Means of all 3 experimental groups and control groups all 3 sessions in micro –Siemens (without extreme values)

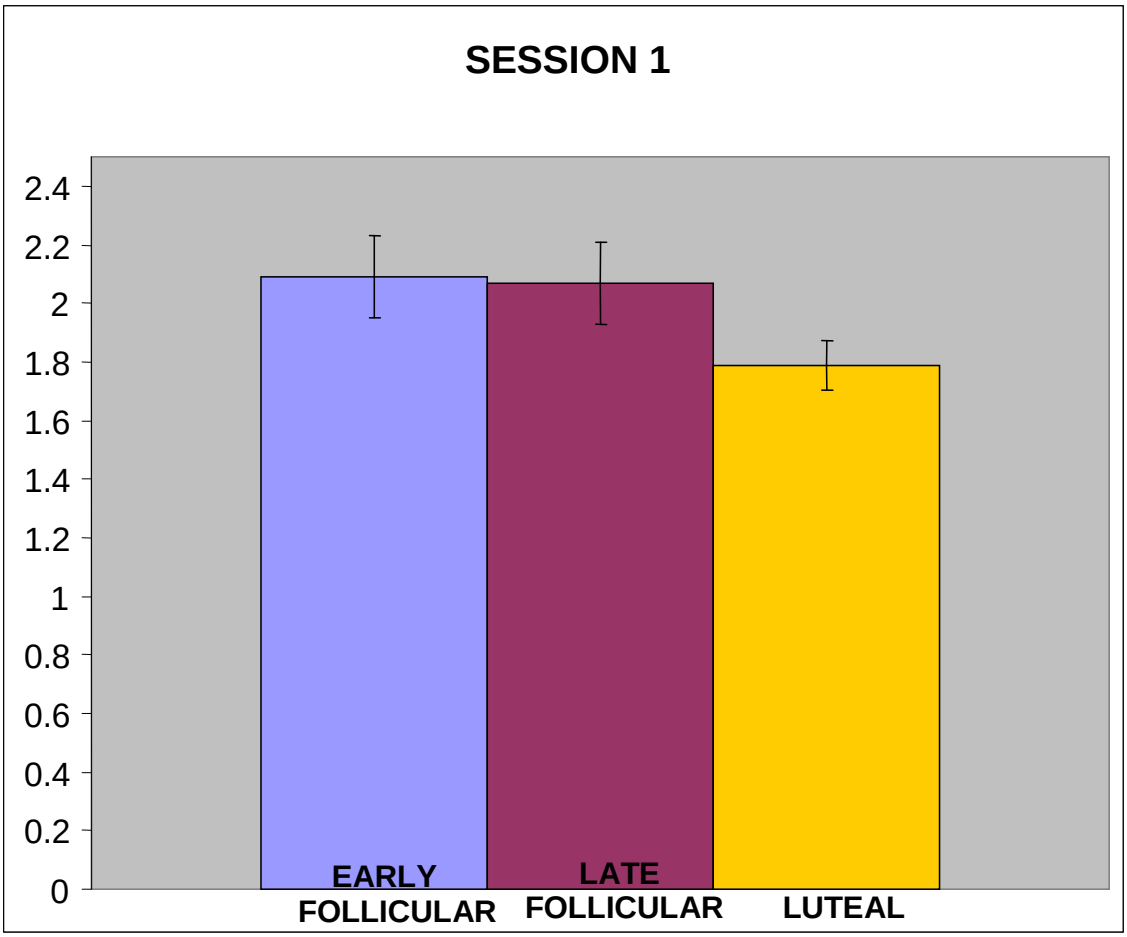


Figure 2: Means of all 3 experimental groups in sessions 1 in micro-Siemens

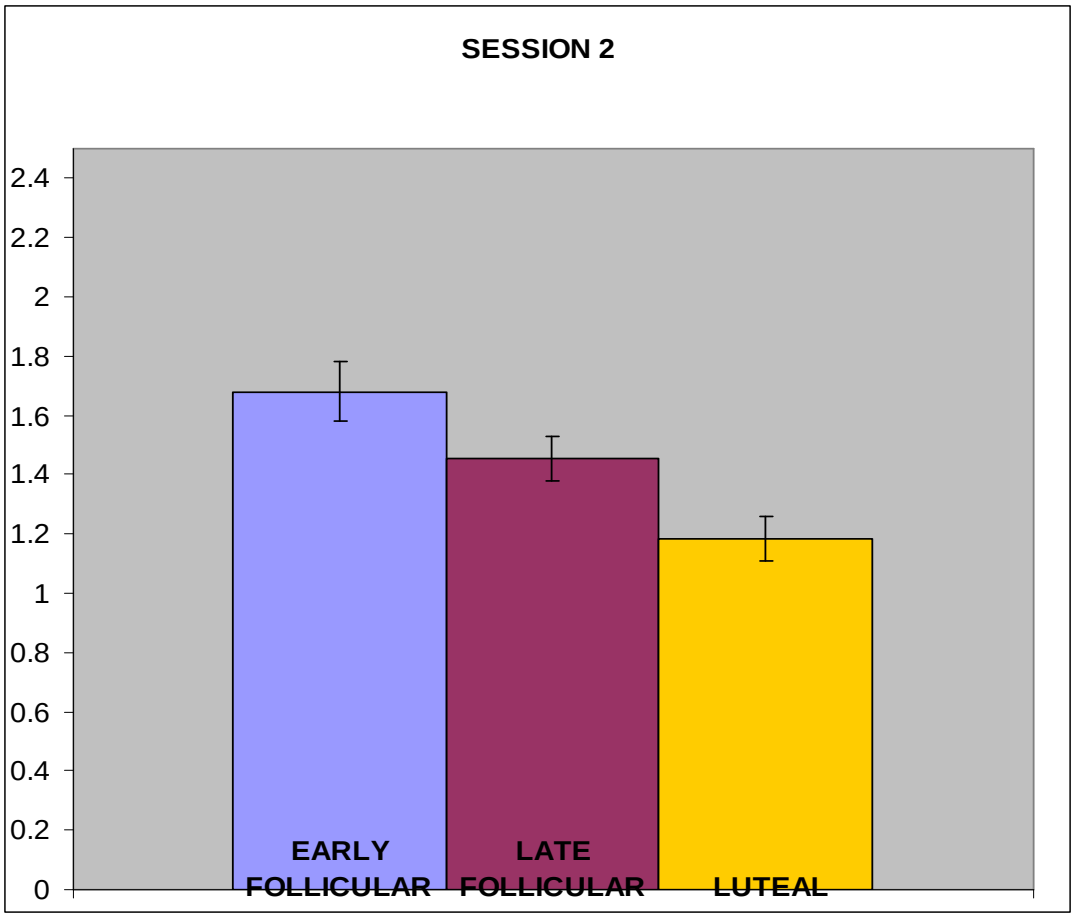


Figure 3: Means of all 3 experimental groups in sessions 2 in micro-Siemens

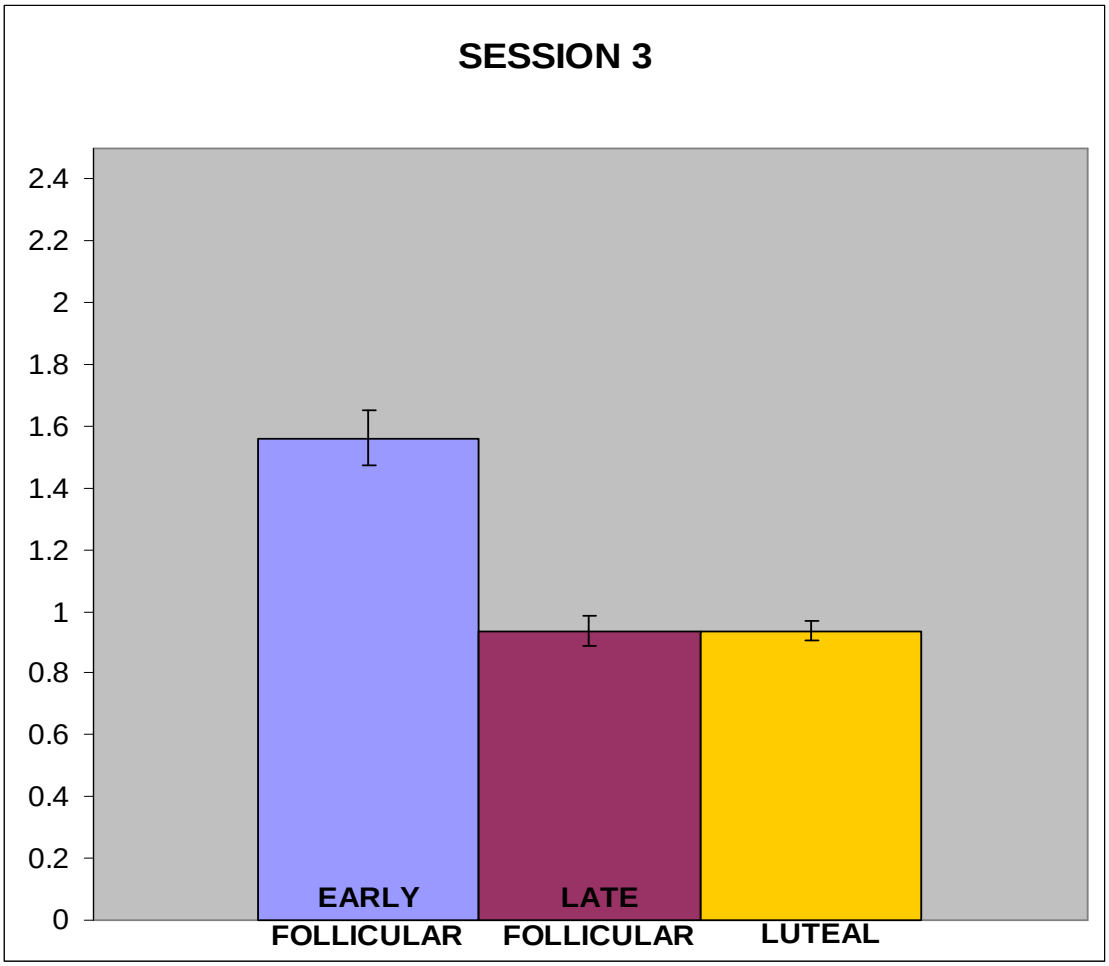


Figure 4: Means of all 3 experimental groups in sessions 3 in micro-Siemens

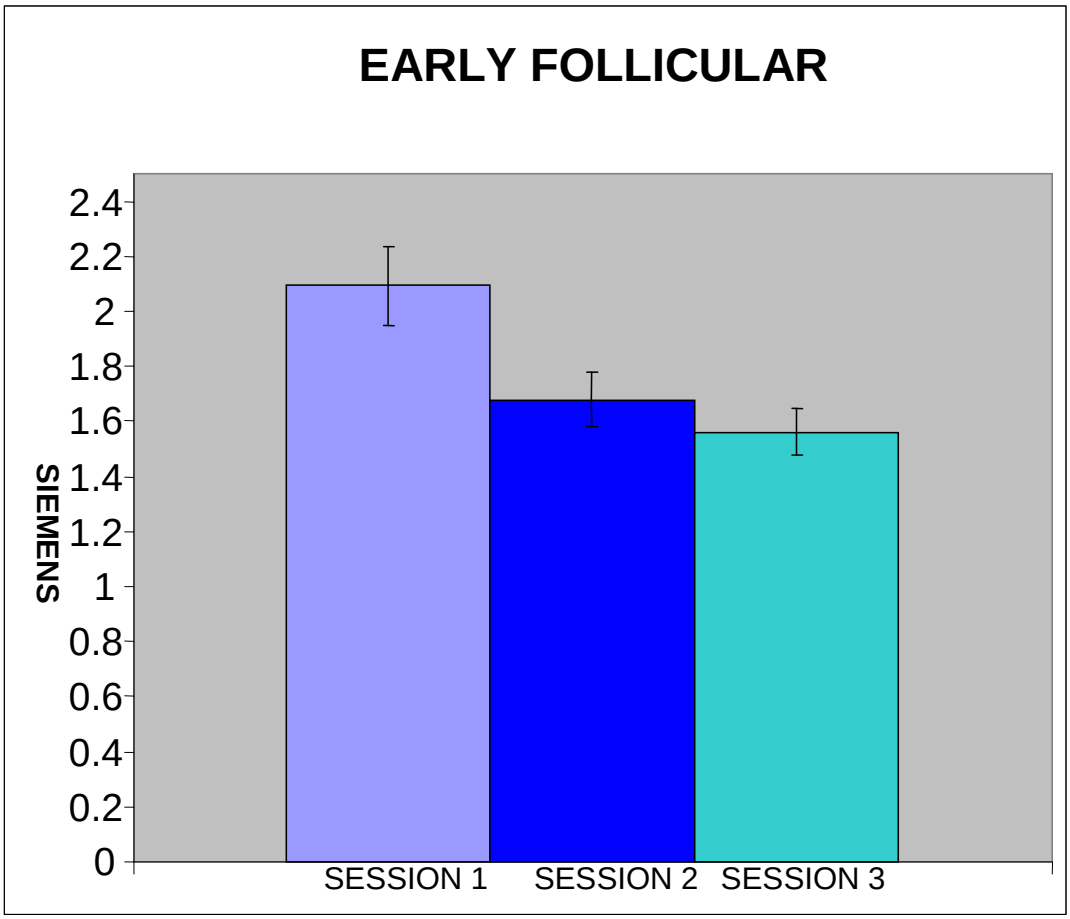


Figure 5: Means in micro-Siemens for the early follicular experimental group over all 3 sessions.

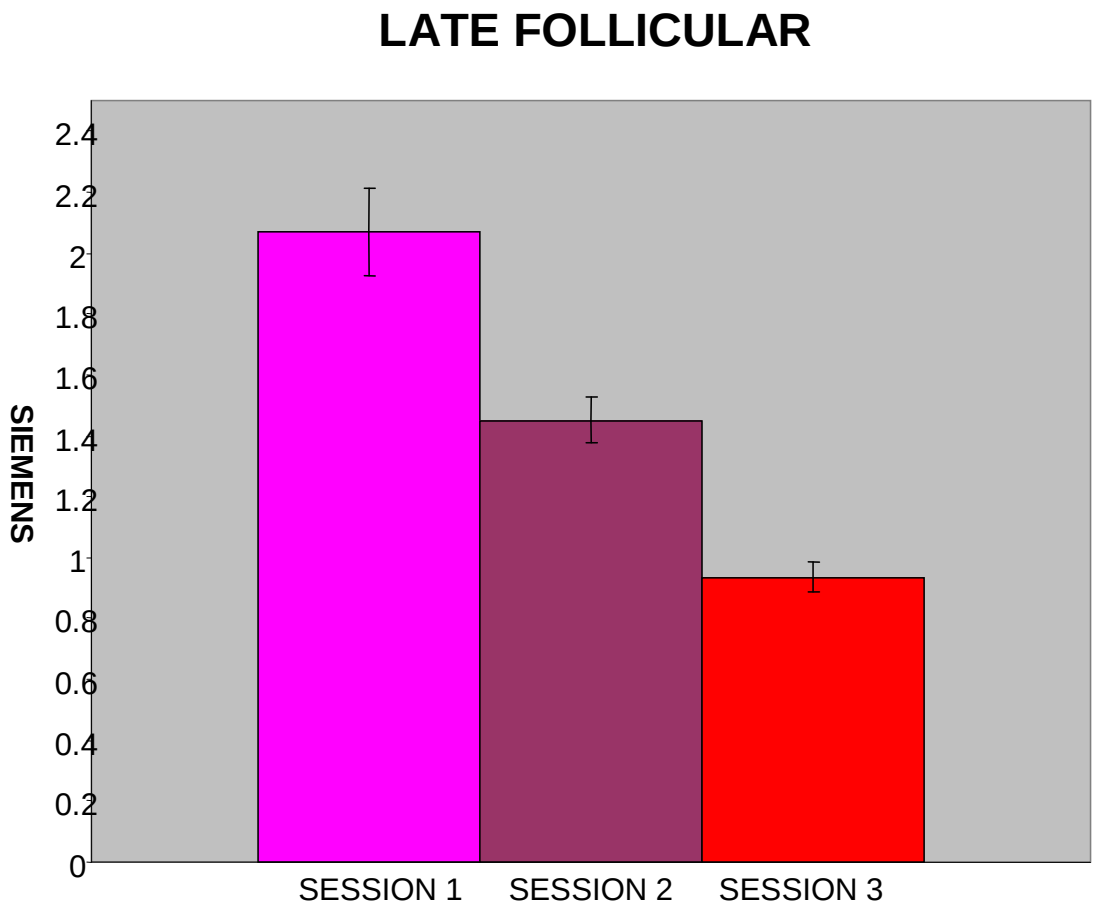


Figure 6: Means in micro-Siemens for the late follicular experimental group over all 3 sessions.

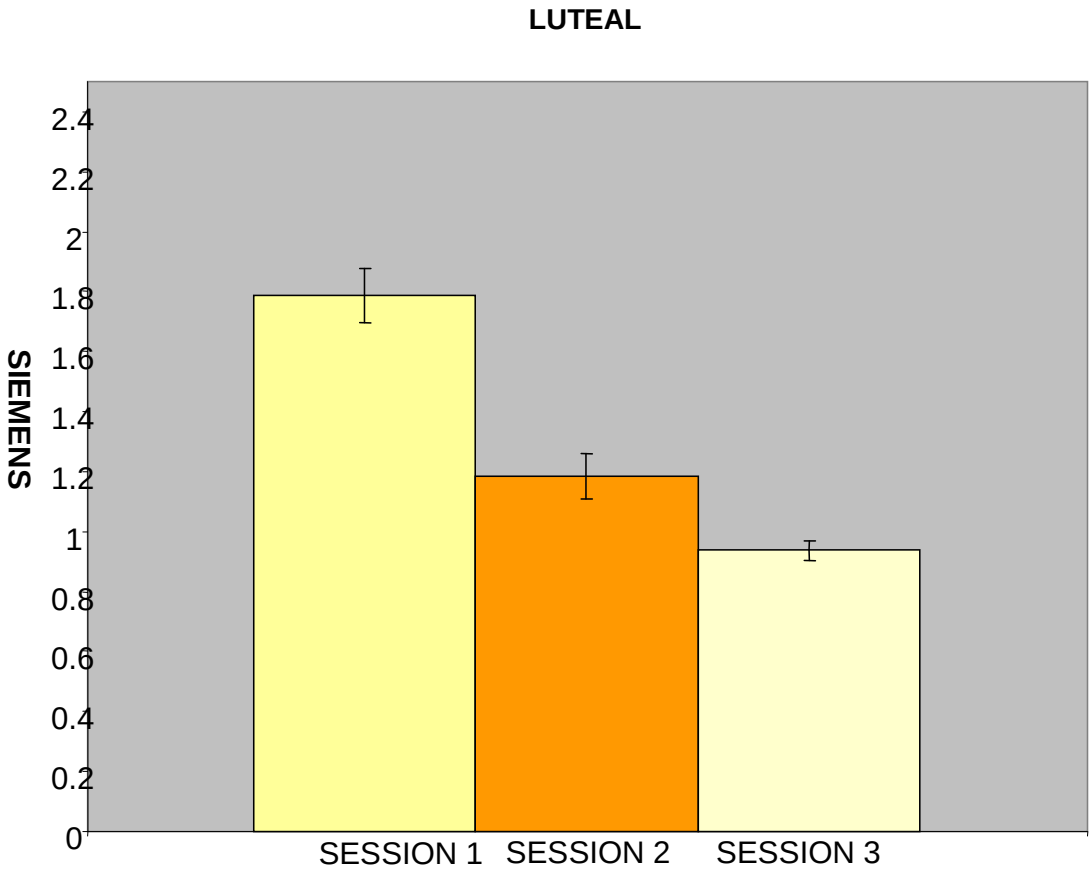


Figure 7: Means in micro-Siemens for the luteal experimental group over all 3 sessions.

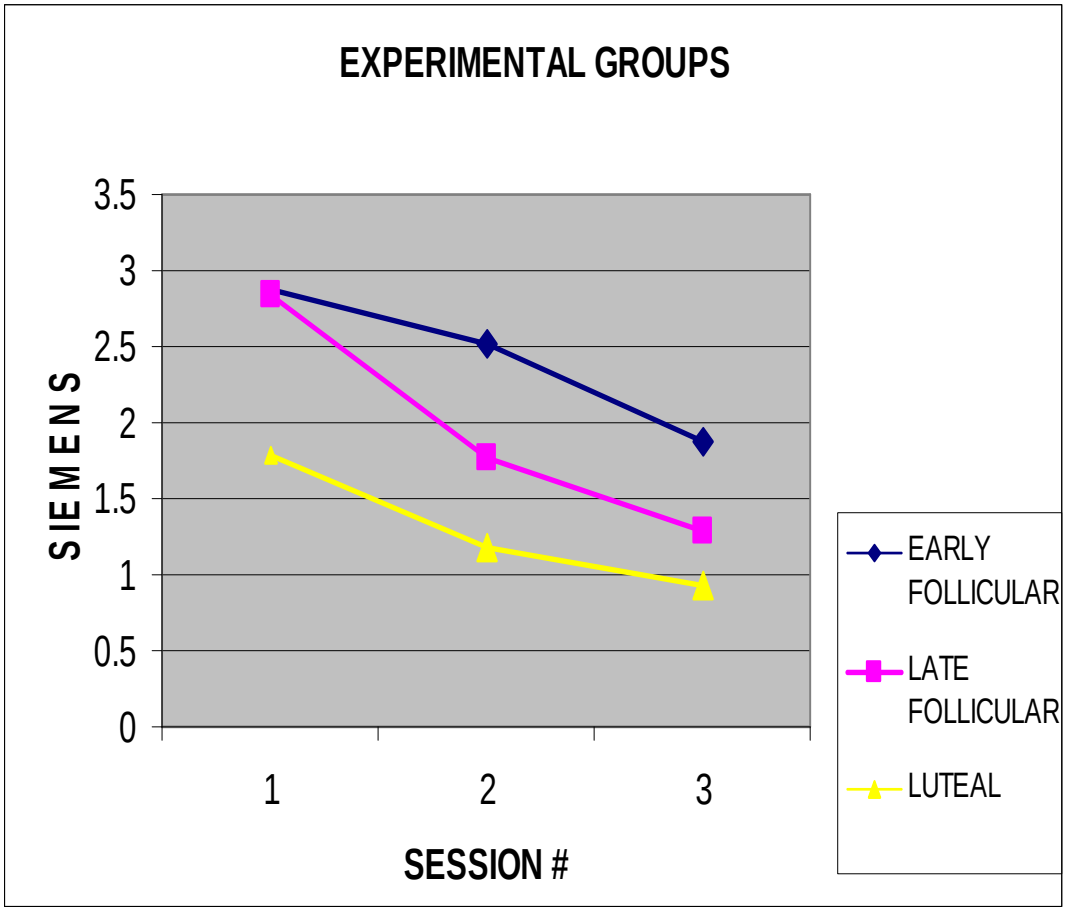


Figure 8: All 3 experimental groups over all 3 sessions in micro-Siemens.

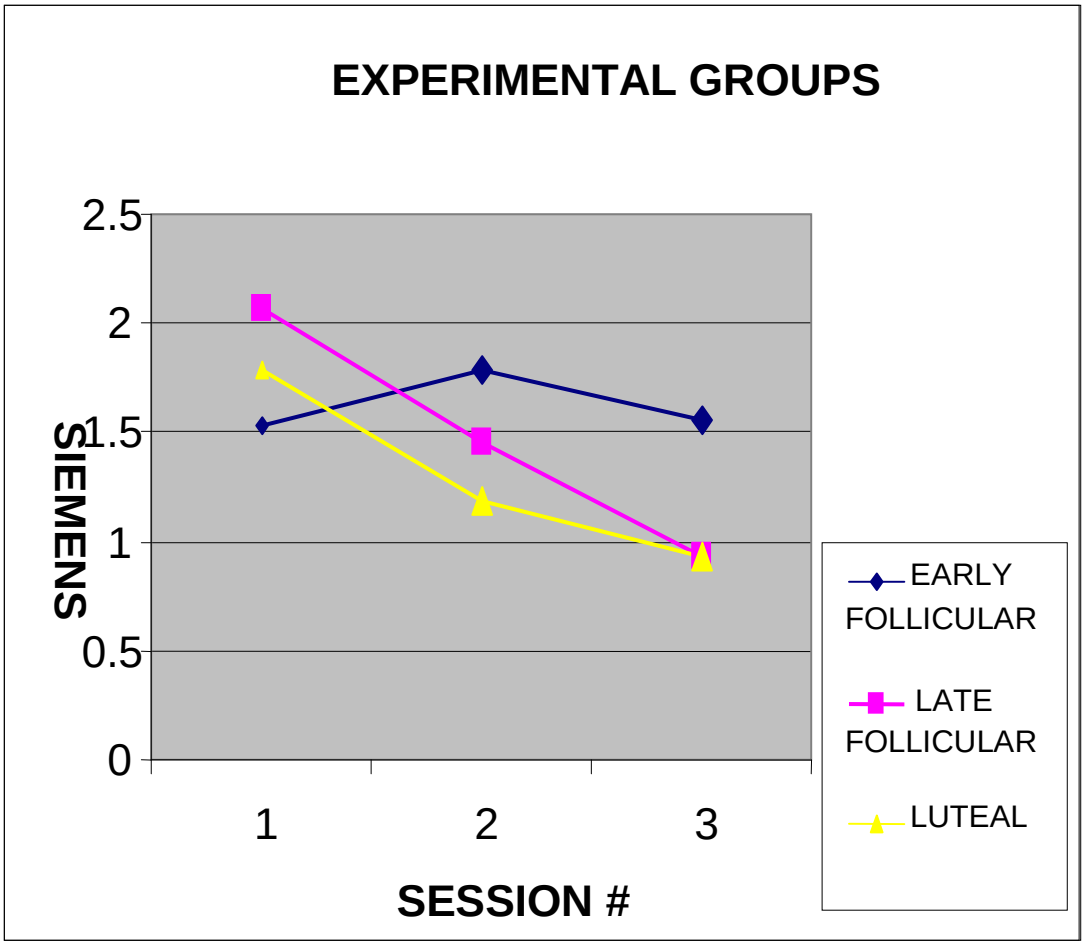


Figure 8': All 3 experimental groups over all 3 sessions in micro –Siemens (without extreme values).

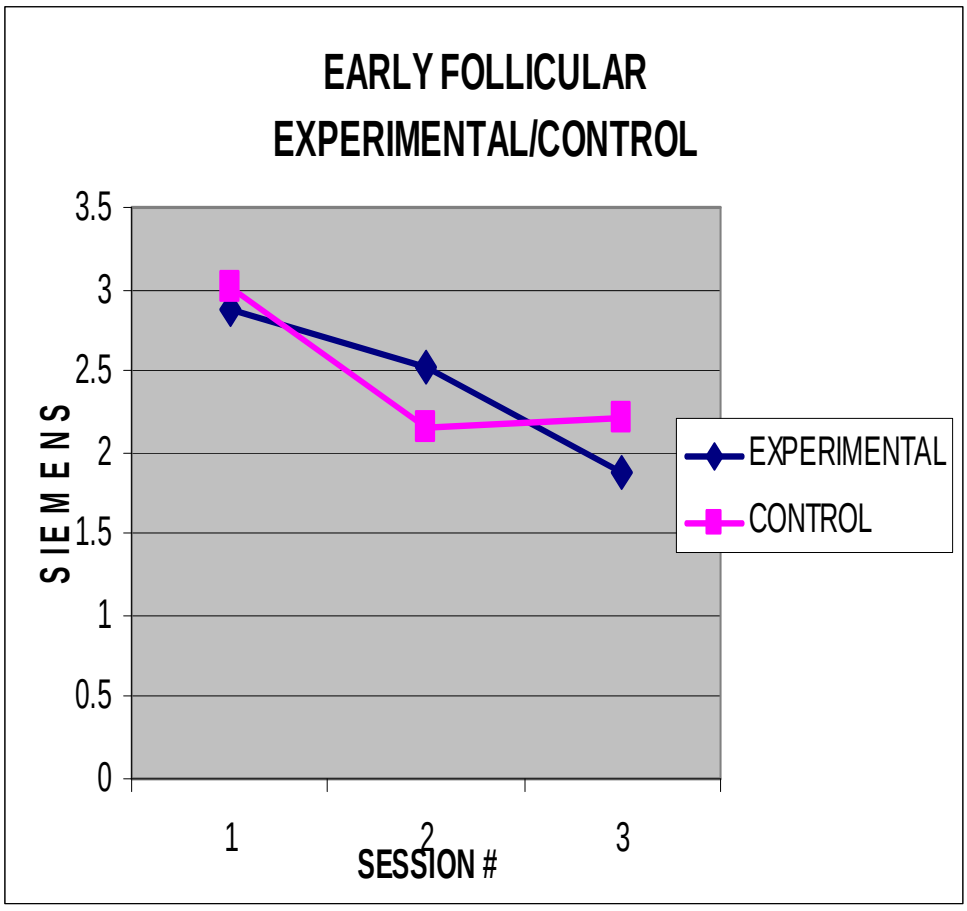
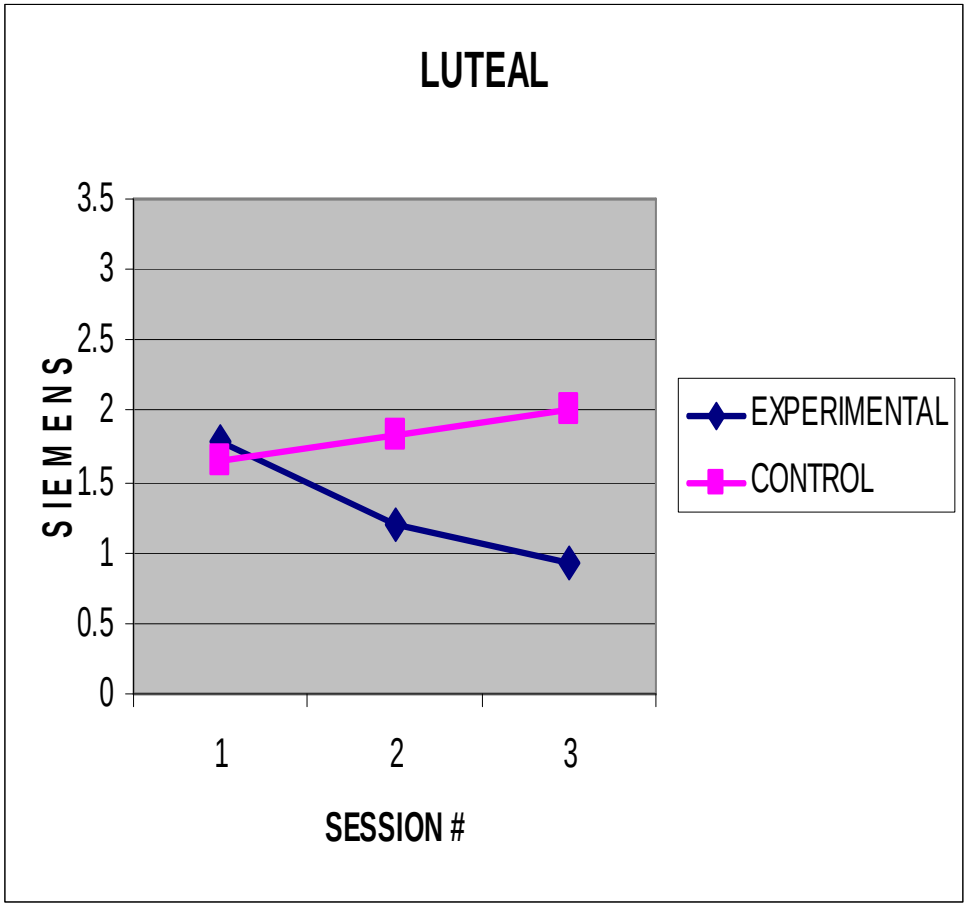


Figure 8a: Early follicular groups experimental and control over all 3 sessions in micro - Siemens.


LUTEAL
3.5
3
2.5
2
1.5
1
0.5
0
SIEMENS
1
2
3
SESSION #
EXPERIMENTAL
CONTROL

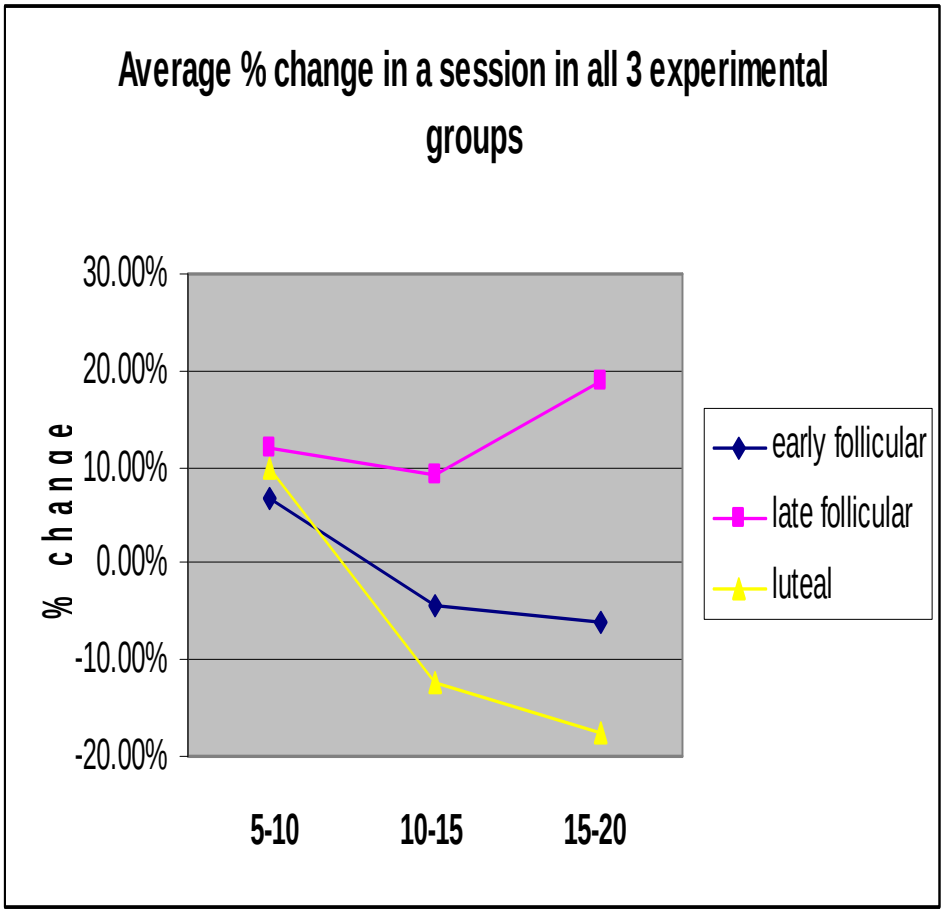


Figure 11: Changes in percentage in conductivity from the first 5 minutes of each session to the next 3 segments 5-10 minutes, 10 -15 minutes and 15 to 20 minutes. (In all three experimental groups)

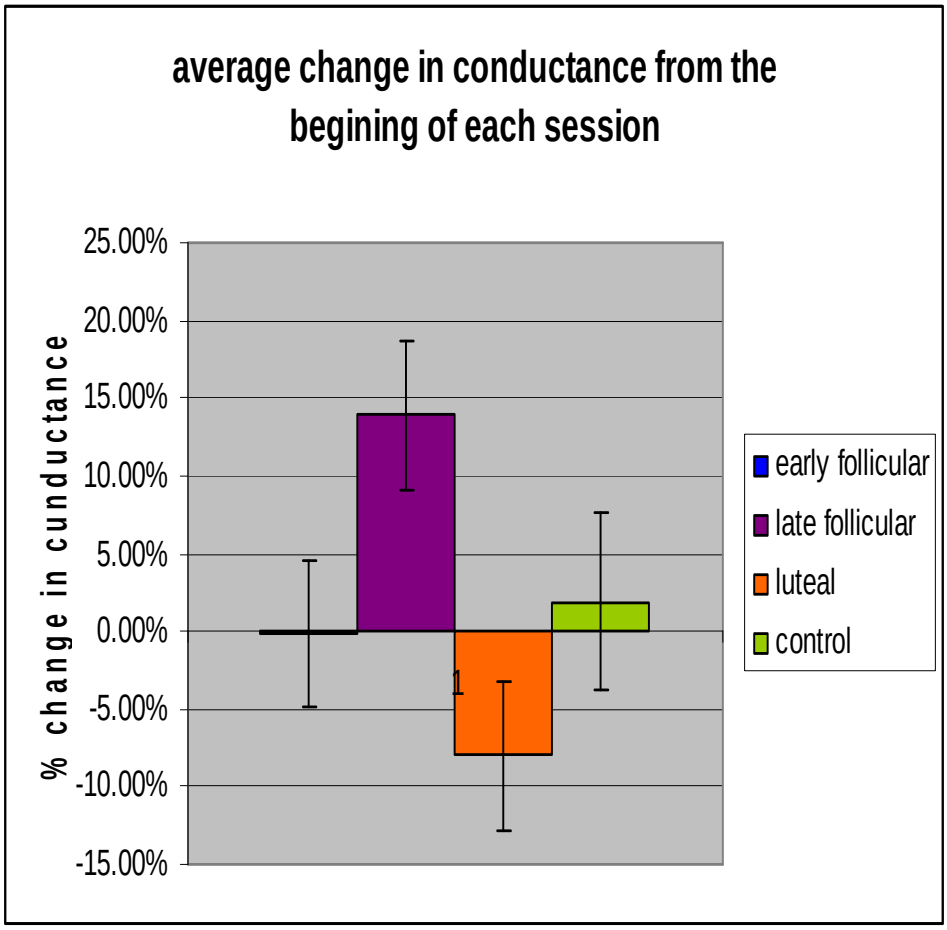


Figure 11a: Average change in conductance, from the first 5 minute segment to the 3 other segments (5-10 minutes, 10 to15 minutes, 15 to 20 minutes) in each session.

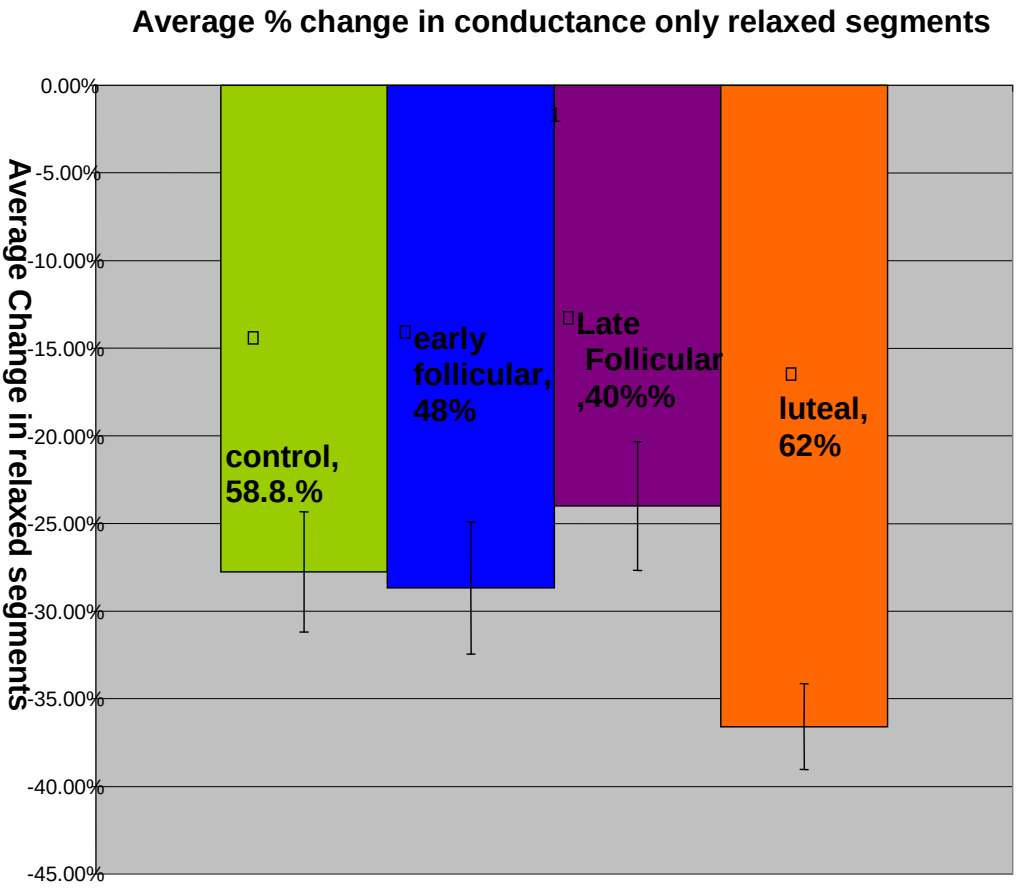


Figure 11b: Average decrease in conductance (only segments that decreased in conductance), from the first 5 minute segment to the 3 other segments (5-10 minutes, 10 to15 minutes, 15 to 20 minutes) in each session. On the bars: the percentage of segments out of all the segments that showed a decrease in conductance.

Descriptive	Early follicular	Late follicular	Luteal
Mean	1.62	1.48	1.3
SD	1.23	1.18	1.01
SE	0.109	0.12	0.08
Variance	1.53	1.39	1.037
N	128	96	144

Table 3: All 3 experimental groups all 3 sessions in conductance units micro-Siemens (without extreme values) nearly reaches significance p=.06

Descriptive	Early follicular	Late follicular	Luteal
Mean	1.53	2.06	1.78
Median	1.157	1.77	1.68
SD	1.26	1.6	1.23
Se	0.29	0.28	0.178
Variance	1.597	2.586	1.519
Range	4.22	4.81	4.74
N	40	32	48

Table 4: Session 1 All 3 experimental groups (without extreme values), in conductance units micro- Siemens, no significant differences.

Descriptive	Early follicular	Late follicular	Luteal
Mean	1.78*	1.45	1.18*
Median	1.35	1.34	0.755
SD	1.3	0.84	1.02
Se	0.197	0.149	0.148
Variance	1.715	0.718	1.055
Range	4.25	2.78	4.14
N	40	32	48

Table 5: Session 2 All 3 experimental groups (without extreme values), in conductance units micro-Siemens (without extreme values).
* p=0.016 significant

Descriptive	Early follicular	Late follicular	Luteal
Mean	1.56*+	0.93*	0.93+
Median	1.58	0.8	0.84
SD	1.15	0.55	0.43
Se	0.17	0.093	0.06
Variance	1.32	0.3	0.18
Range	4.41	2.16	1.719
N	40	32	48

Table 6: Session 3 All 3 experimental groups (without extreme values), in conductance units micro-Siemens (without extreme values).
* p= 0.005857 significant , + p=0.00071 significant.

Session	Early follicular	Late follicular	Luteal
All	-2.97% *+	-44.87% *	-35.56%+
Session 2	-2.5%# >	-42.62%#>	-33.333%>
Session 3	-3.3%##	-47.10%##	-43.49%##

Table 7: Average change in conductance from the first session to the next two sessions as expressed in percentage, change in percentage from the first to the second session, and change in percentage from the first to the third session.
p=0.0032, + p=0.008, # p=0.019, > p=.03, ##p=.04

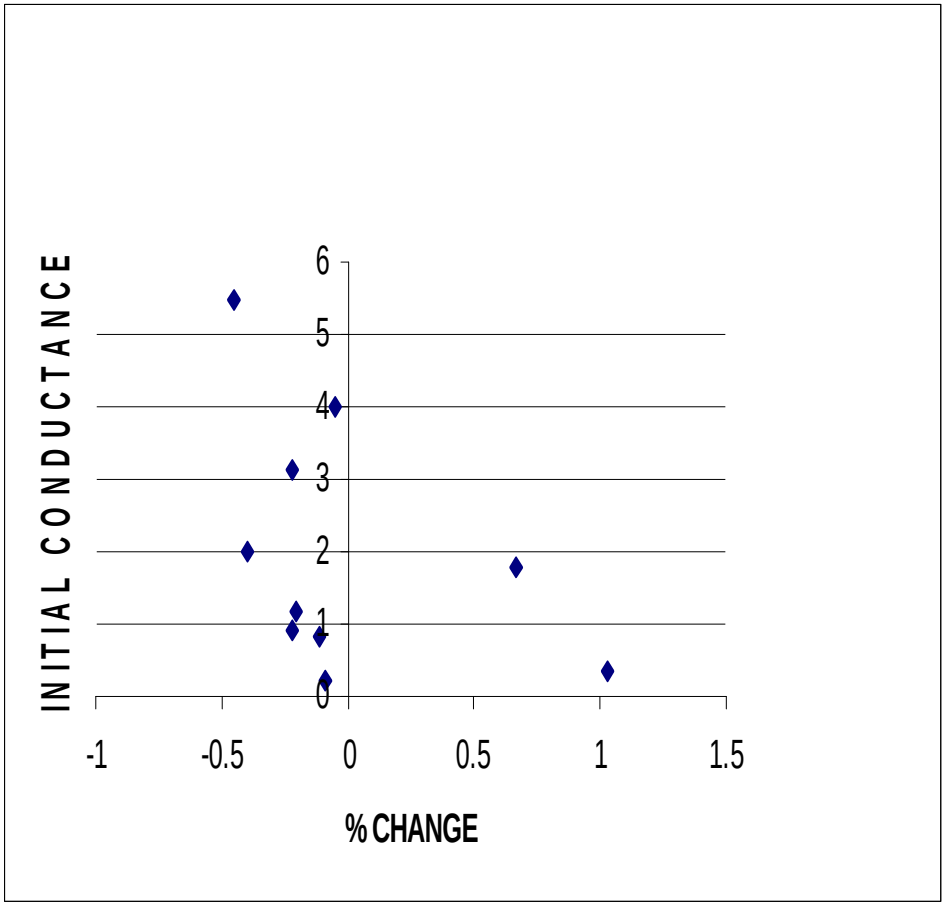


Figure 12: correlation between the initial conductivity on the first session and the change in conductivity on the second session in the early follicular stage. R(10)=0.41854, t=1.3, p>.05

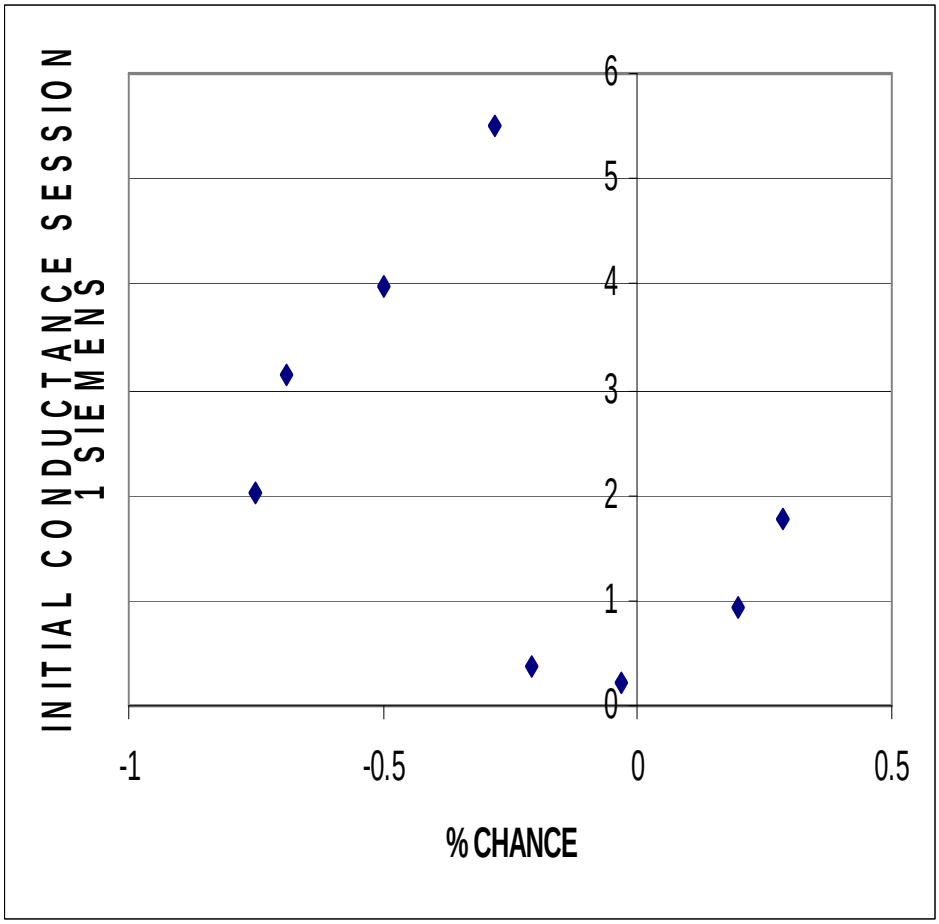


Figure 13: correlation between the initial conductivity on the first session and the change in conductivity on the third session in the early follicular stage. r=0.19879 t=0.5

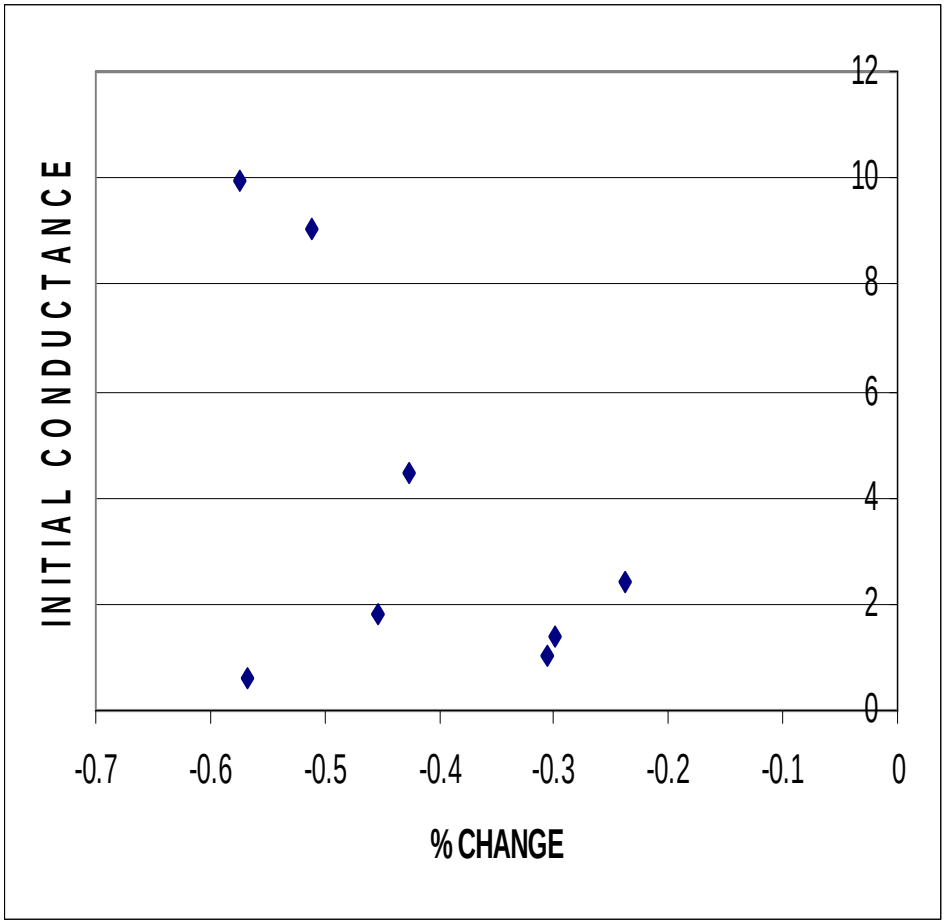


Figure 14: correlation between the initial conductivity on the first session and the change in conductivity on the second session in the late follicular stage. r=0.53 t=1.65

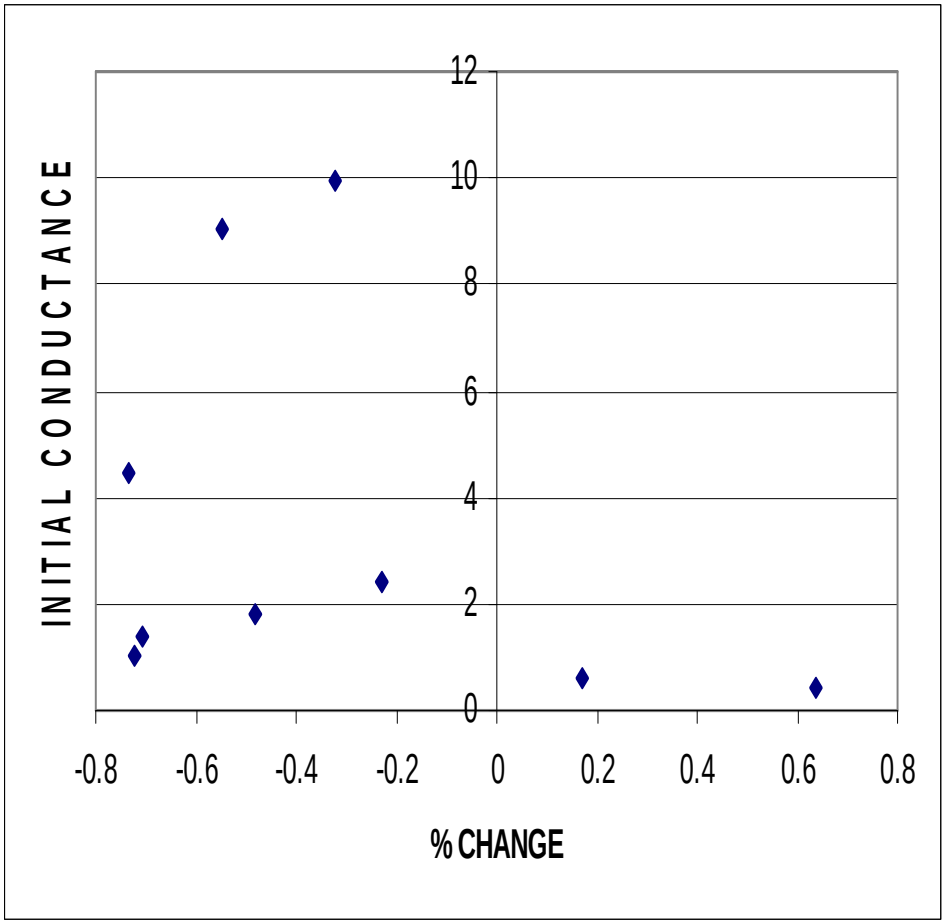


Figure 15: correlation between the initial conductivity on the first session and the change in conductivity on the third session in the late follicular stage r=0.30334 t=0.78

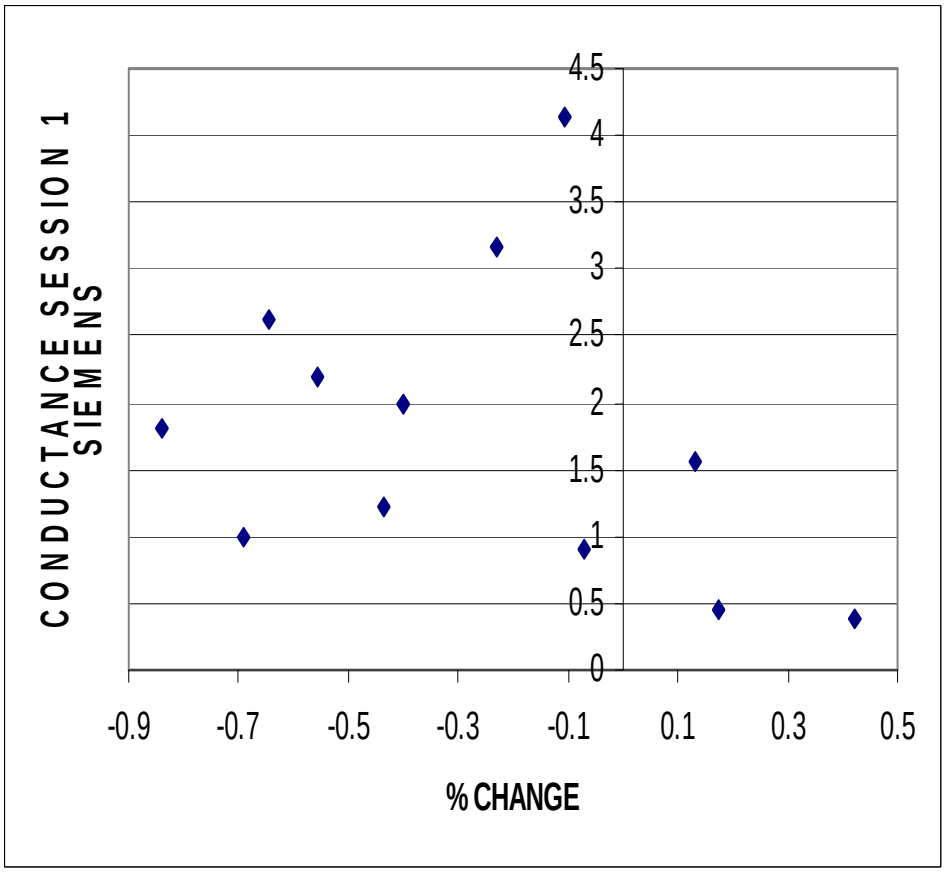


Figure 16: correlation between the initial conductivity on the first session and the change in conductivity on the second session in the luteal phase. r=-0.297 t=0.98

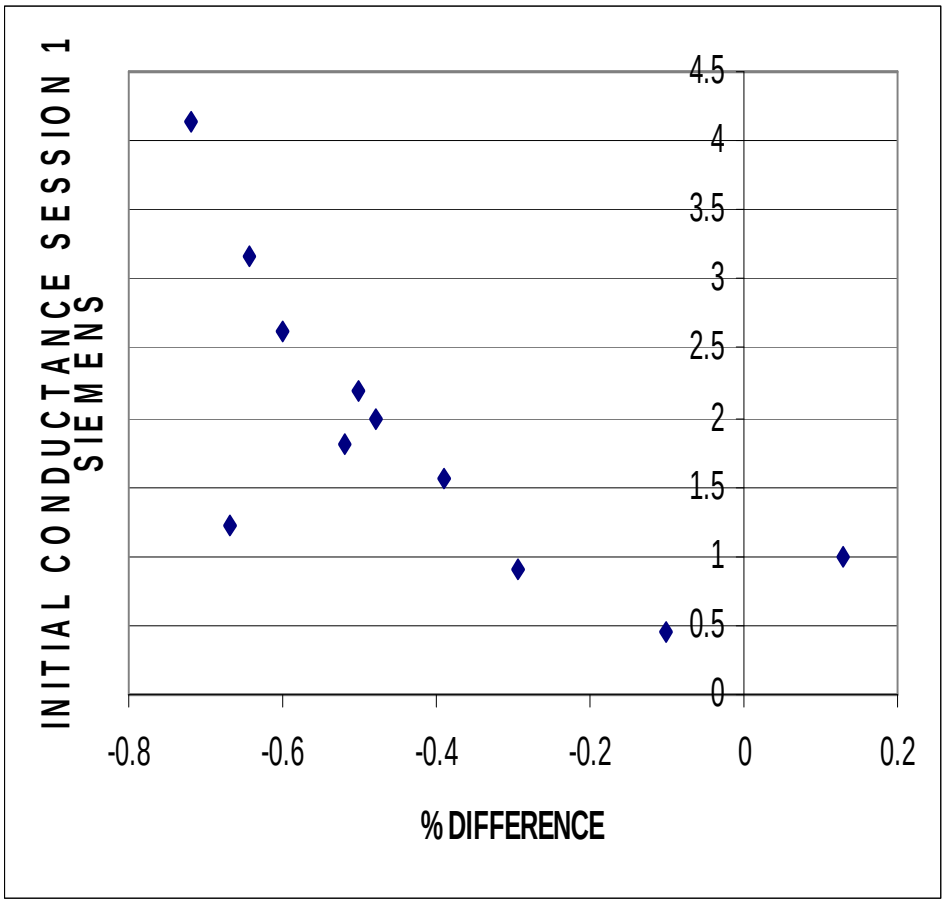


Figure 17: correlation between the initial conductivity on the first session and the change in conductivity on the third session in the luteal phase. r=-0.72312 t=3.14 p<0.01

	Early follicular	Late follicular	Luteal
% change session 2	r=-0.21	r=-0.19	r= -0.45 t=1.59
% change session 3	r=-0.59 t=2.3*	r=-0.1	r=-0.038

Table 8. Correlation between the average change in conductivity from the first session to the next two sessions and the change in the length of the cycle.
*p<0.05

	Early follicular	Late follicular	Luteal
% change session 2	r=-0.19	r=-0.2	
% change session 3	r=-0.25	r=-0.47 t=1.06	

Table 9: Correlation between the average change in conductivity from the first session to the next two sessions and the change in the ovulation day.

	Early follicular	Late follicular	Luteal
Change in each session	r(10)=-0.55 t=-2.07*		r= -0.06
Change relaxed segments	r(10)=-0.47 t= 1.59		r= -0.2

Table 9a: Correlation between average change in conductance, from the first 5 minutes of each session to the rest of the session, and change in cycle length.
Correlation between change in cycle length and change in conductance in relaxed segments only. *p<0.05

Participant	Phase of cycle	First month	Second Month	Third Month
sa40603	Early follicular	13	14	12
dr89205	Early follicular	11	9	14
gr29802	Early follicular	15	16	15
ej45604	Early follicular	15	16	16
bw39905	Early follicular	14	14	13
sv67504	Early follicular	15	15	15
nj86505	Early follicular	16	16	16
fa68605	Late follicular	15	12	13
sf54105	Late follicular	10	9	10
jw42605	Late follicular	14	14	14
ee26705	Late follicular	11	10	11
je82605	Late follicular	13	16	15
nv52805	Late follicular	13	14	15
lk43503	Luteal	13	15	15
sm65303	Luteal	13	13	14
dm73905	Luteal	10	13	13
om71505	Luteal	16	17	16
afh23405	Luteal	14	16	16
mf48605	Luteal	8	8	8
pg65305	Luteal	14	16	15
gd25104	Luteal	7	11	16

Table 10: change in the day of ovulation in the 3 experimental groups. The two first month being the base line. No significant difference was found between the base line and the third month.

Participant	Phase of cycle	First Month	Second Month	Third Month
sa40603	Early Follicular	27	28	26
dr89205	Early Follicular	32	31	30
gr29802	Early Follicular	27	28	30
ej45604	Early Follicular	30	32	30
bw39905	Early Follicular	29	29	28
sv67504	Early Follicular	27	30	27
nj86505	Early Follicular	36	33	28
fa68605	Late Follicular	29	26	26
sf54105	Late Follicular	29	29	31
jw42605	Late Follicular	26	27	27
ee26705	Late Follicular	31	29	31
je82605	Late Follicular	29	27	35
gl90205	Late Follicular	25	27	26
nv52805	Late Follicular	28	28	29
lk43503	Luteal	31	30	28
sm65303	Luteal	30	29	27
dm73905	Luteal	27	27	28
om71505	Luteal	32	32	25
afh23405	Luteal	26	28	31
mf48605	Luteal	33	31	30
pg65305	Luteal	27	29	28
gd25104	Luteal	28	28	31

Table 11: Change in the length of the cycle on the third month as compared to the first two month (the base line). There is a significant change on the third month in the early follicular stage as well as in the luteal stage. In the late follicular stage there is no significant difference

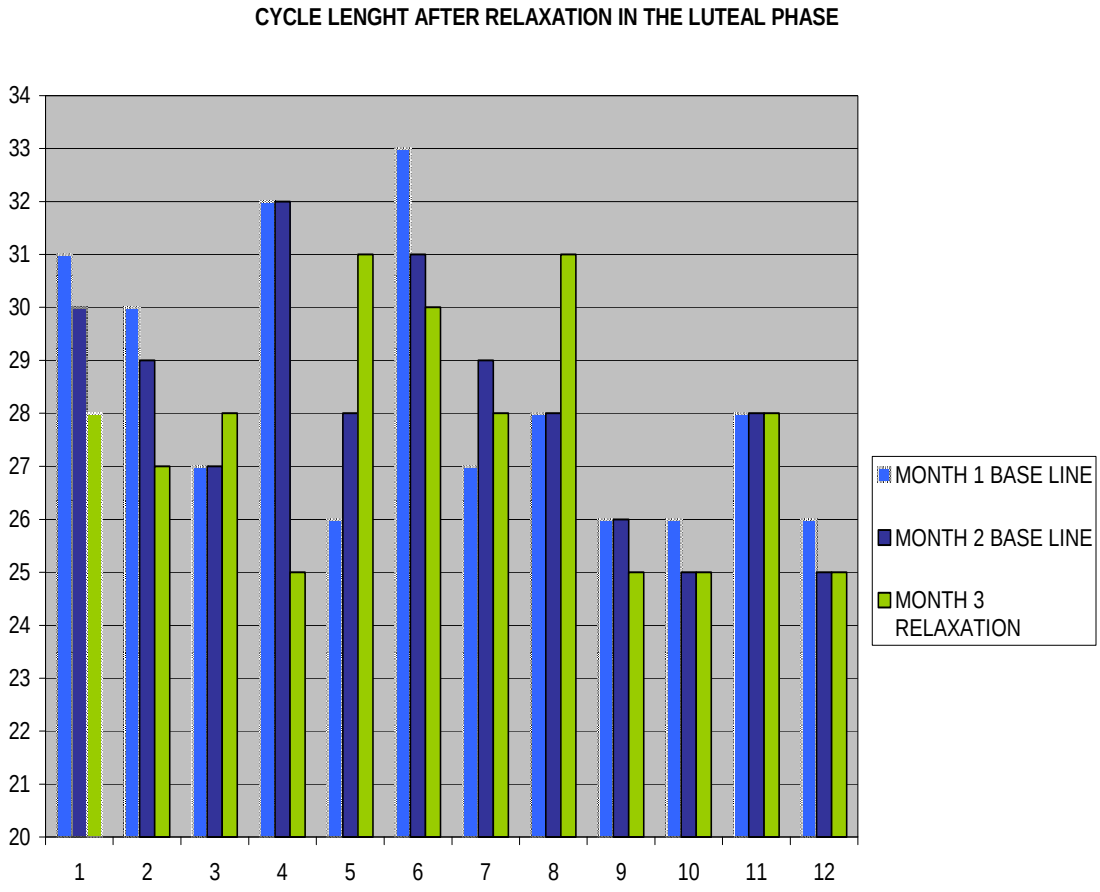


Figure 18: Length of cycle in the two base line month and on the experimental third month when relaxation was done at the luteal phase.

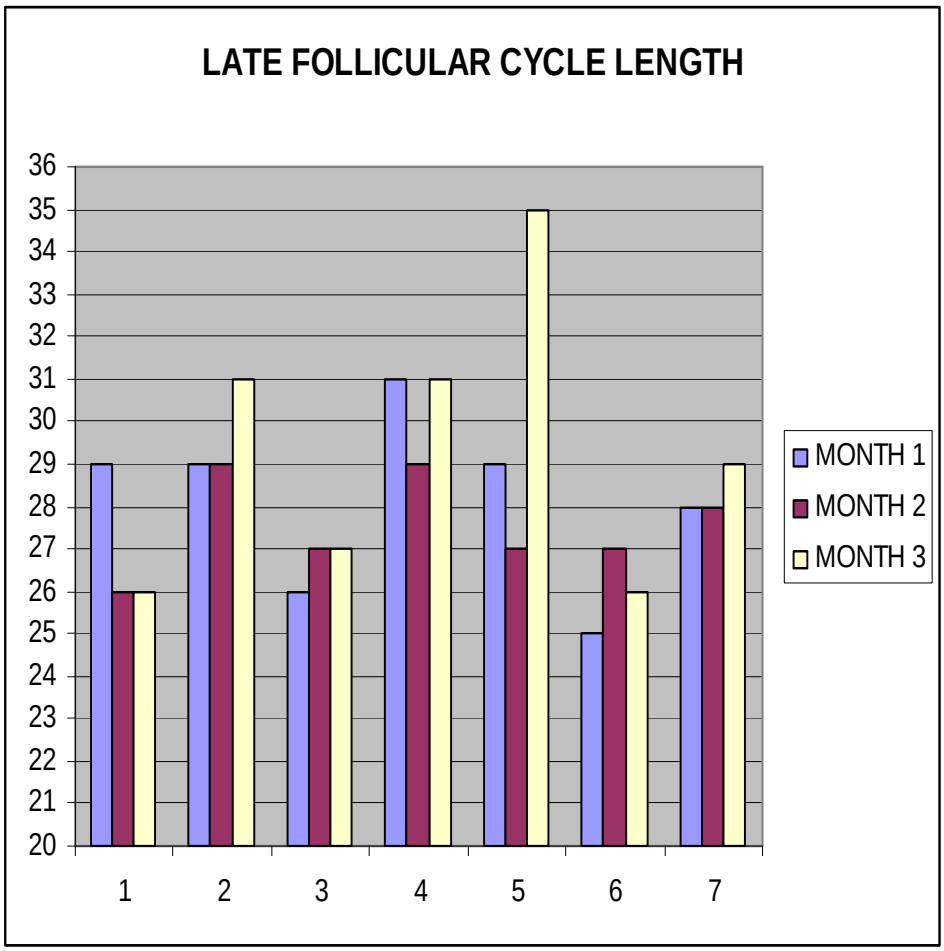


Figure 19: Length of cycle in the two base line month and on the experimental third month when relaxation was done at the late follicular phase.

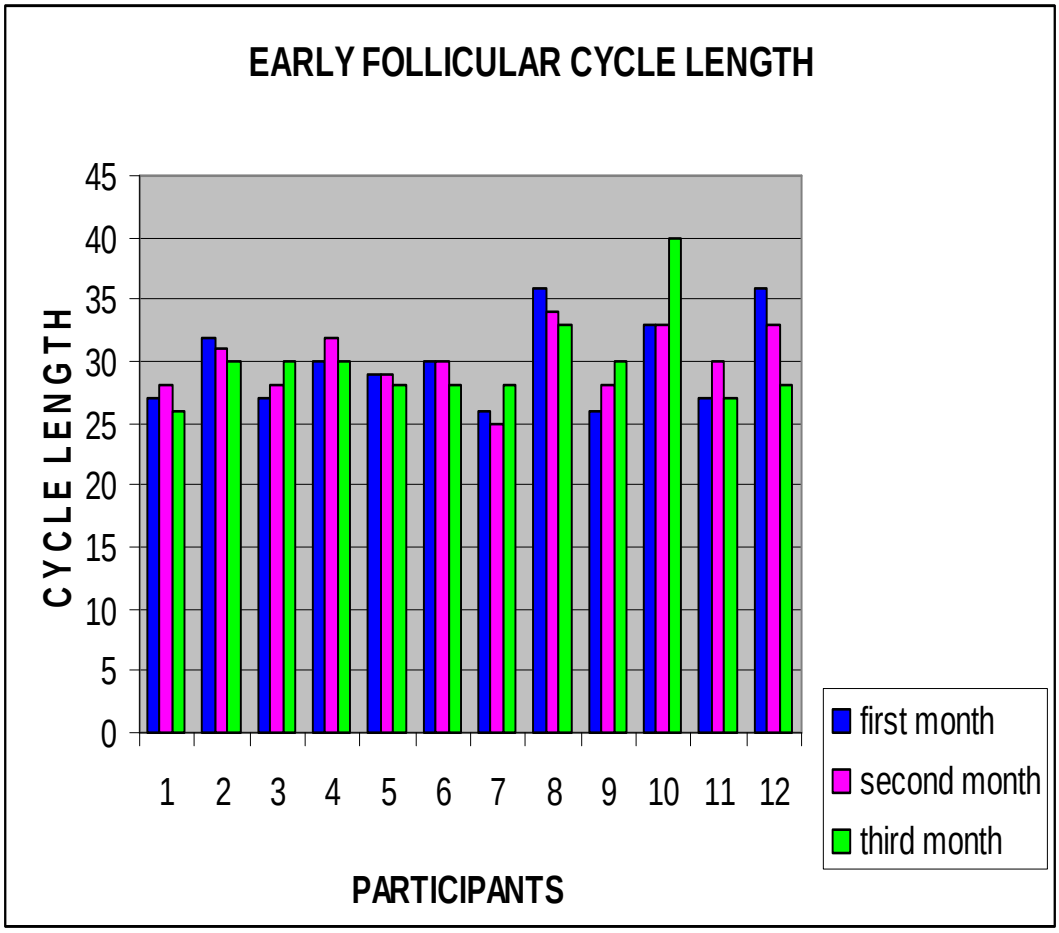


Figure 20: Length of cycle in the two base line month and on the experimental third month when relaxation was done at the early follicular phase.

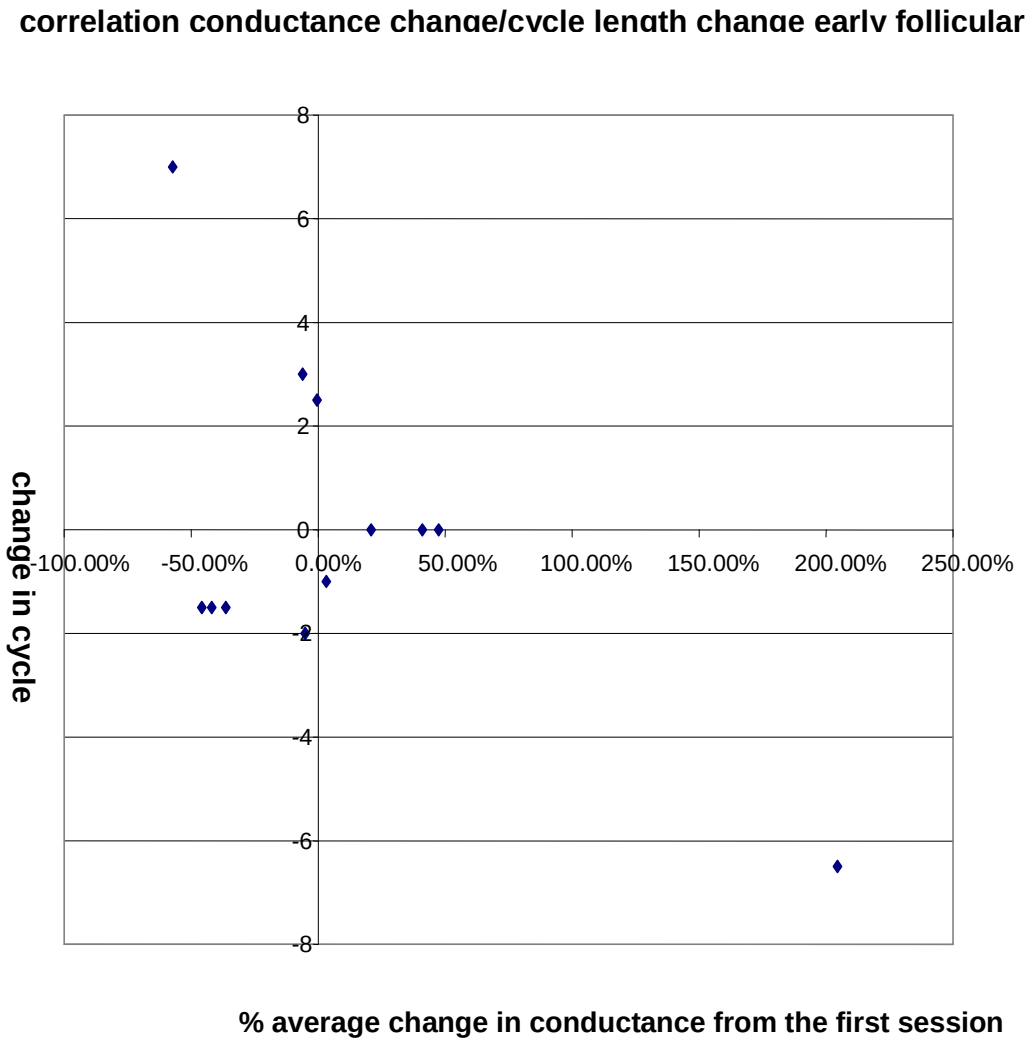


Figure 21: Early follicular correlation between the average change in conductance (in percentage) from the first session (to the subsequent 2 sessions) and the change in the cycle length. r(10)=-0.61 t(10)=-2.45 p<.01.

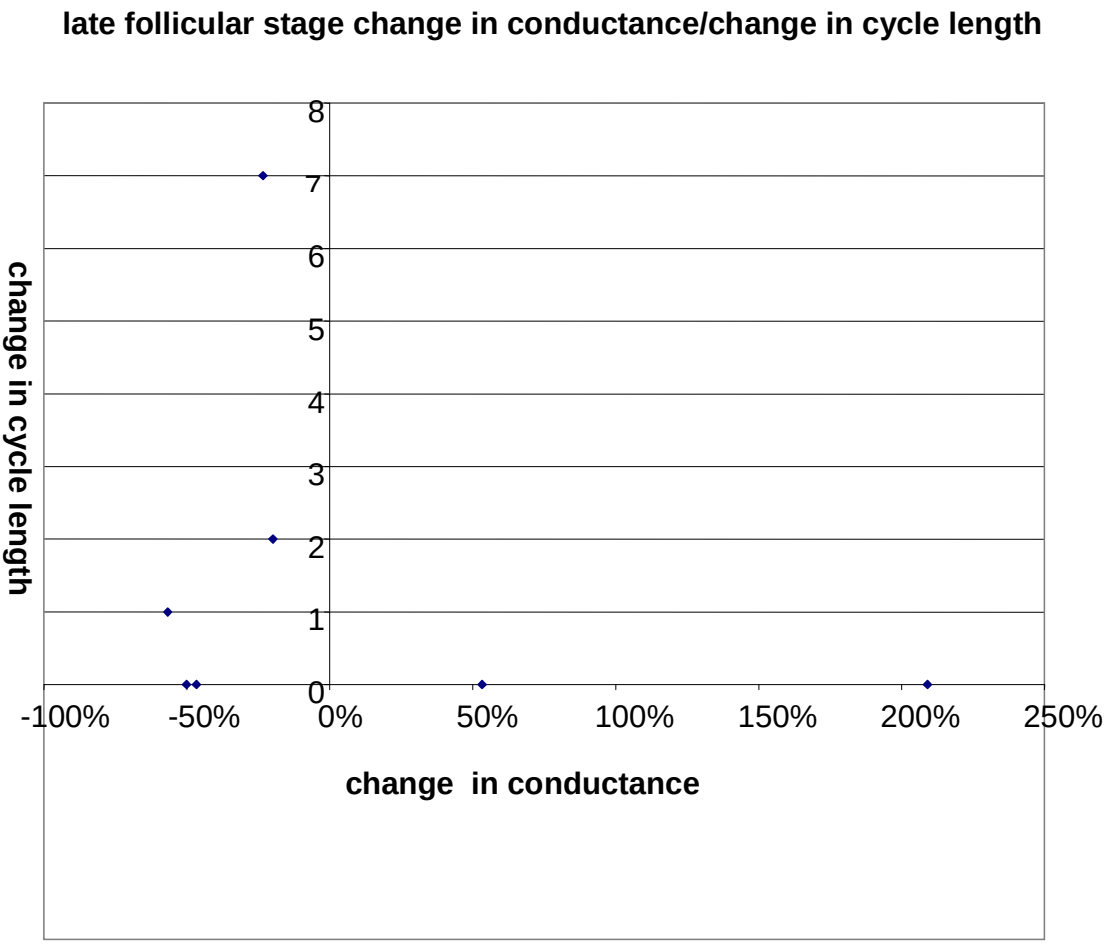


Figure 22: Late follicular correlation between the average change in conductance (in percentage) from the first session (to the subsequent 2 sessions) and the change in the cycle length. $r(5)=-0.24$, $p>.05$.

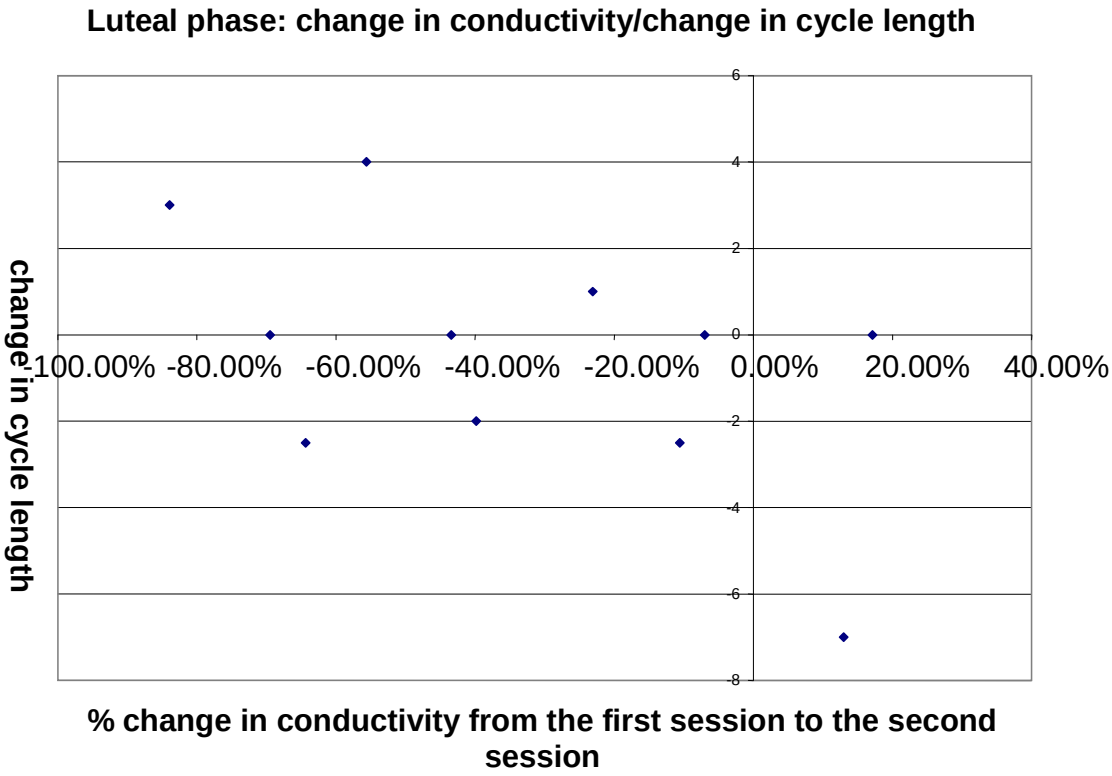


Figure 23: Luteal phase correlation between the average change in conductance (in %) from the first session (to the subsequent 2 sessions) and the change in the cycle length. $r(10)=-0.51$ $t(10)=-1.87$ $p<.05$.

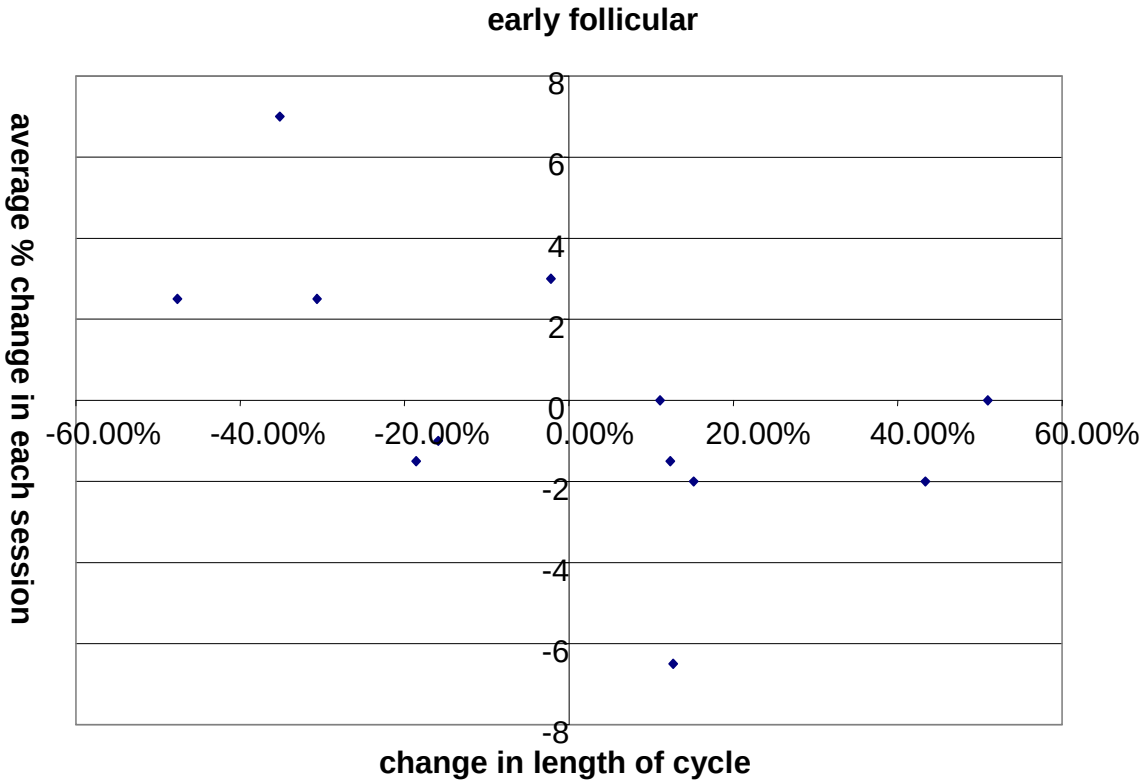


Figure 24: Early follicular correlation between the average change in conductance in each session (from the first 5 minutes of each session) and the change in the cycle length. $r(10)=-0.5489$ $t(10)=2.07$ $p<.05$

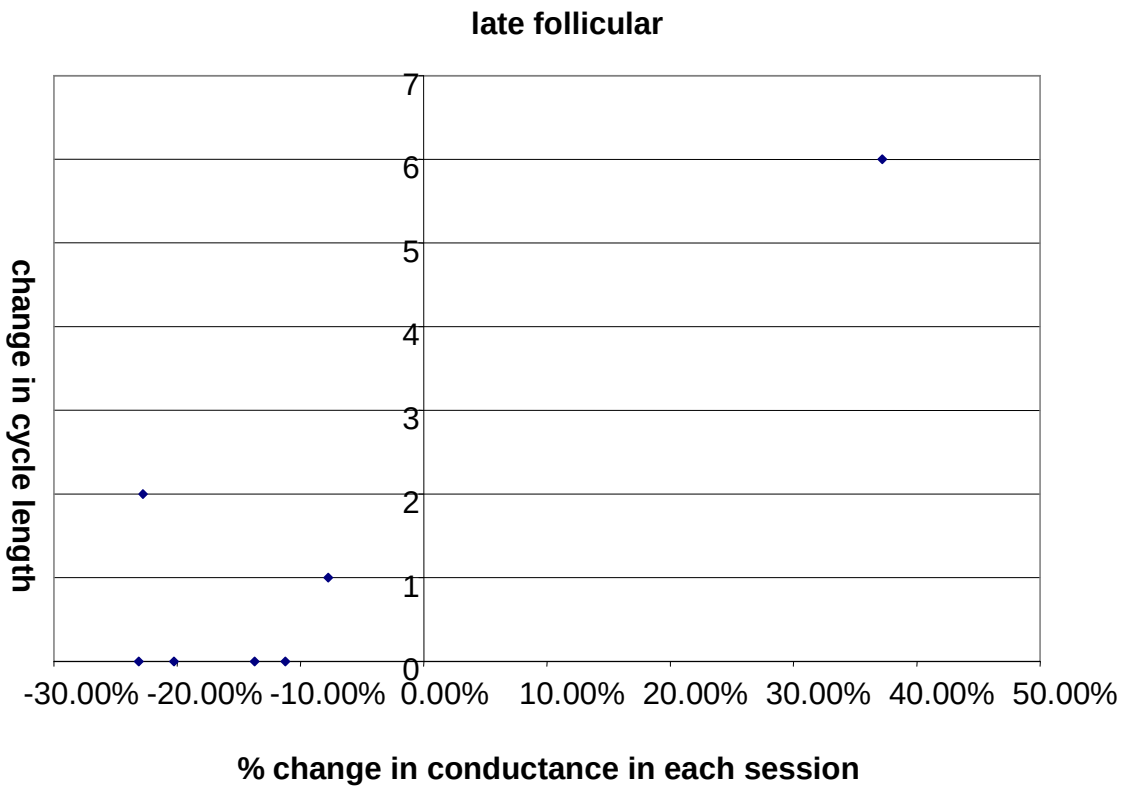


Figure 25: Late follicular correlation between the average change in conductance in each session (from the first 5 minutes of each session) and the change in the cycle length. $r(5)=0.887$ $t(5)=4.3$ $p<.05$.

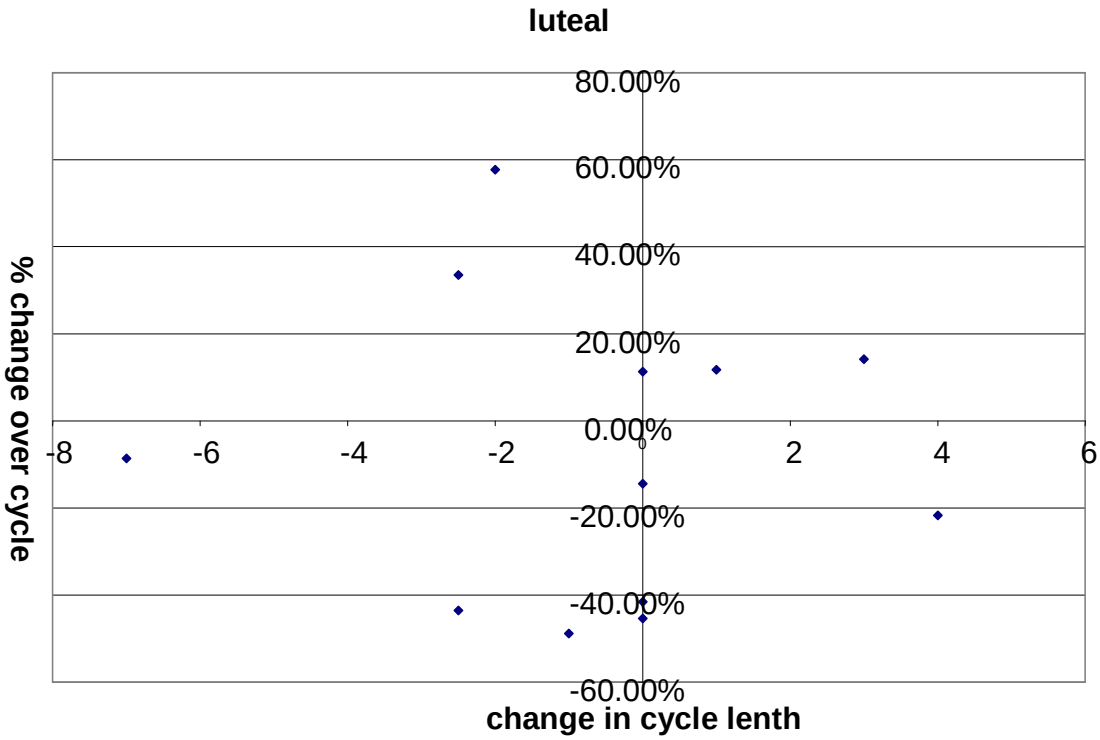


Figure 26: Luteal phase correlation between the average change in conductance in each session (from the first 5 minutes of each session) and the change in the cycle length. $r(10)= -0.065$ $p>.1$

Regression line conductance/cycle length change

Early follicular stage

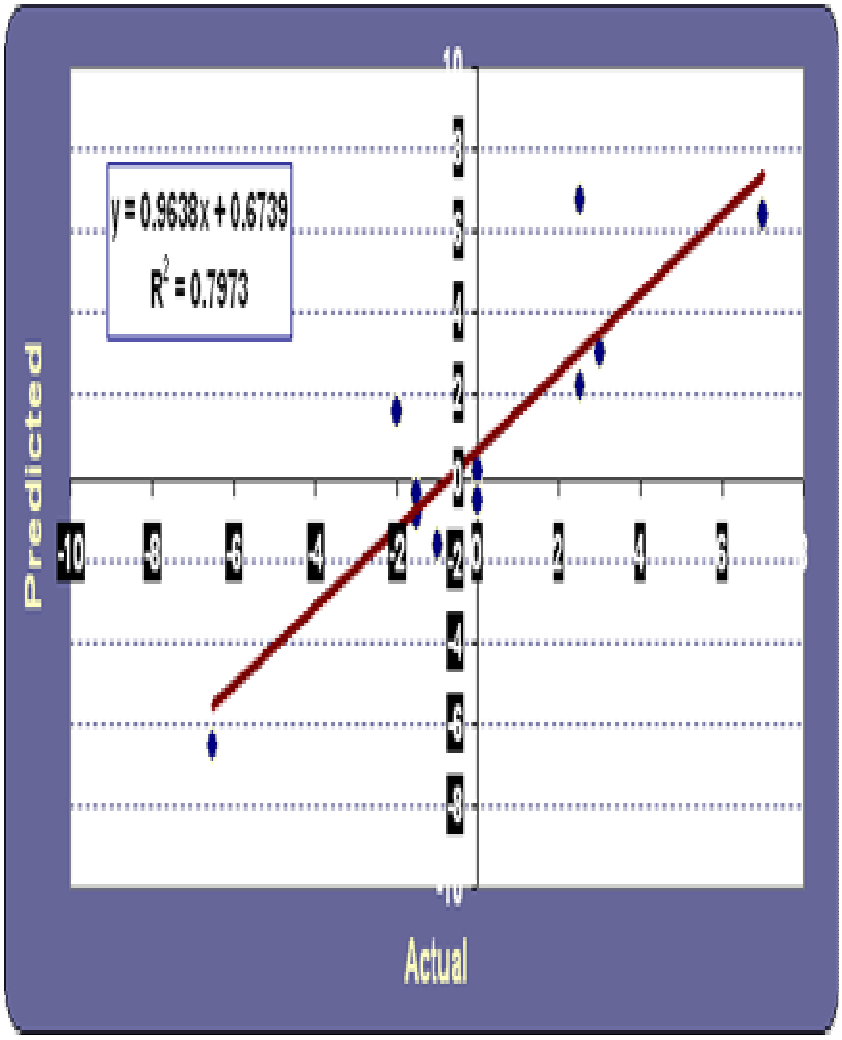


Figure 27: cycle length change as predicted by change in conductance (during the sessions) and actual change in cycle length in the early follicular phase (with regression line).

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