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DEPRESSIVE SYMPTOMS AND MONOPHASIC ORAL CONTRACEPTIVE USE

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ABSTRACT

The purpose of this study was to see if depressive symptoms varied with oral contraceptive use. Forty monophasic oral contraceptive users and 40 naturally cycling women aged 18 to 22 completed the Children's Depression Inventory (CDI), an instrument suitable for young adults. Monophasic oral contraceptives contain a stable dose of estradiol throughout the active phase of the menstrual cycle, and reduce endogenous levels of circulating estradiol. In comparison, naturally cycling women experience fluctuations in estrogens throughout the menstrual cycle, and have higher levels of circulating estradiol than oral contraceptive users. It was hypothesized that, compared to naturally cycling women, oral contraceptive users would report fewer depressive symptoms if the presence of estradiol *fluctuations* contributes to depression, but more depressive symptoms if low estradiol *levels* are a factor in depression. No significant difference in depressive symptoms was found between monophasic oral contraceptive users and naturally cycling women. Failure to see a group difference may reflect methodological limitations (e.g., sample size insufficient to detect what are likely to be small effects, small gender difference on the CDI in college students) or that depressive symptoms are associated with both estradiol fluctuations and levels which vary in different directions in women using oral contraceptives. Future research should focus on the separate effects of estradiol fluctuations and level on depressive symptoms.

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Introduction

The prevalence of depression throughout the lifespan is well documented. About half of all men and women experience at least one two-week period of depressed mood or anhedonia during their lifetime (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). A higher prevalence of depressive symptoms is evident in adolescents and young adults than individuals in childhood or middle adulthood (Kessler et al., 1993). Kessler et al. (1993) suggested that the presence of depression may be underrepresented in surveys of the general population; this may be due in part to the stigma associated with mental illness.

There is a sex difference in the prevalence and severity of depressive symptoms. Women consistently report experiencing more depressive symptoms than do men during adolescence (Angold & Rutter, 1992; Angold, Costello, & Worthman, 1998; Avison & McAlpine, 1992) and adulthood (Angst et al., 2002; Nolen-Hoeksema, Larson, & Grayson, 1999) and are almost twice as likely as men to be diagnosed with clinical depression throughout their lifetime (Kessler et al., 1993). Women are more likely than men to report individual symptoms of depression, including diminished interest in activities (Angst, 2002; Kessler et al., 1993), chronic strain and rumination (Nolen-Hokesema et al., 1999), loss of appetite, sleep, and concentration, and increased agitation, tiredness, worthlessness, suicidal thoughts, and impairment (Angst et al., 2002).

The sex difference in depression is first evident during puberty. During childhood, the prevalence of depression is fairly similar for boys and girls (Angold & Rutter, 1992; Angold, Erkanli, Silberg, Eaves, & Costello, 2002). During puberty, a significant gender difference emerges, around age 13 (Angold & Rutter, 1992; Angold, Costello, & Worthman, 1998; Ge,

Conger, & Elder, 2001; Hankin, Abramson, Moffitt, Silva, McGee, & Angell, 1998); by age 16 women are twice as likely as men to have significant depressive symptoms (Angold & Rutter, 1992). This large gender difference is a product of increasing depressive symptoms throughout adolescence, with women reporting increases in depression at a steeper rate than men (Hankin et al., 1998).

The sex difference in depression that begins in early adolescence suggests the importance of changing social roles and the biological processes of puberty on depressive symptoms. During puberty and adolescence, boys and girls may experience increased pressure to abide by stereotypical gender roles, which may account for part of the sex difference in depression (Avison & McAlpine, 1992; Ge, Conger, & Elder, 2001; Wichstrøm, 1999). Generally, masculinity and femininity, or overall gender typicality, are found to be associated with the increased gender difference in depression during puberty (Broderick & Korteland, 2002; Jewell & Brown, 2014; Whitley, 1984). Research indicates that depression is positively associated with femininity (Broderick & Korteland, 2002) and negatively associated with masculinity (Whitley, 1984), but results are not consistently found (Priess, Lindberg, & Hyde, 2009). The intensification of gender identity and roles may account for the sex difference in depression during puberty, as girls are more feminine and experience more depression. While changing social roles likely have an impact on the gender difference in depression, biological factors also play a role in depression.

The biological processes associated with puberty may be responsible for the increased prevalence of depressive symptoms in adolescent girls, as pubertal status is associated with the sex difference in depression (Angold, Costello, & Worthman, 1998; Ge, Conger, & Elder, 2001). Specifically, pubertal status, and not the age at which the pubertal transition occurs, is related to

the increased gender difference during puberty (Angold, Costello, & Worthman, 1998). Increases in reproductive hormones during the pubertal transition can account for part of the gender difference in depression (Angold, Costello, Erkanli, & Worthman, 1999), and have been suggested as an explanation for why the gender difference begins during early adolescence, instead of earlier in life (for review, see Nobel, 2005). In addition to changes in circulating levels of reproductive hormones, fluctuations in these hormones may be an important factor in depressive symptoms (for review, see Shors & Leuner, 2003). Specifically, fluctuations in the estrogen, estradiol may account for a portion of the sex difference in depressive symptoms. As discussed below, there is accumulating evidence for a relationship between estrogen and depression in women of reproductive age, from increases in estradiol prior to menarche through decreasing estradiol during menopause. Evidence has emerged from studies of women with varying hormonal statuses including pregnancy, menopause, menstrual cycle, and oral contraceptive use. The evidence is seen in studies of pregnancy, menopause, menstrual cycle, and oral contraceptive use, which generally show a link between estrogen and depressive symptoms, but do not clearly distinguish between the effects of estradiol fluctuations and estradiol levels.

Pregnancy and Postpartum Depression

Pregnancy is associated with complex hormonal changes from the time of conception through labor and postpartum. Pregnancy is also associated with depression; postpartum depression is a form of clinical depression encompassing persistent negative emotions that begin after childbirth. During pregnancy, estrogen levels increase significantly. After delivery, estrogen levels decrease rapidly and may contribute to the onset of postpartum depression (for review, see

Shors & Leuner, 2003), although the etiology of postpartum depression is suggested to be multifactorial (Payne, 2003). Women who experience postpartum depression typically have a personal or family history of depression, have low social support, and experience high levels of stress (for reviews, see O'Hara & Swain, 1996; Wisner, Parry, & Piontek, 2002). These women do not consistently exhibit atypical hormone levels in comparison to women without postpartum depression, indicating that the normal decline in estrogen alone is not sufficient to trigger depression.

Recent research suggests women with postpartum depression may actually develop depression during pregnancy (Schiller, O'Hara, Rubinow, & Johnson, 2013; Wisner et al., 2013). Some studies have found that women who experience depression during pregnancy may have improvements in depressed mood prior to delivery, but worsened mood after the sharp drop in estrogen postpartum, which suggests the drop in estrogen can exacerbate pregnancy-related depression (Schiller et al., 2013). While there is a relationship between estrogen and depression during and after pregnancy, studies do not distinguish between effects of absolute levels of versus the change in estrogen.

Perimenopausal Mood

Menopause, like pregnancy, is associated with both changing levels of estrogen and alterations in mood. The menopausal period contains three distinct states: pre-menopause, perimenopause, and menopause or post-menopause. Pre-menopause describes the years leading up to menopause, before menstrual cycles become irregular. Women enter perimenopause when they experience months of amenorrhea or changes in cycle regularity, yet have also gone through menses during the past year (Mayo Clinic, 2013). Perimenopause is characterized by cycle

irregularity, reduced fertility, and fluctuations in estrogen production (Mayo Clinic, 2013; for review, see Shors & Leuner, 2003). The menopausal period concludes with menopause, in which a woman has not experienced a menses within the past year (Mayo Clinic, 2013).

The menopausal period is associated with an increased prevalence of depressive symptoms. Typically, women do not experience increased rates of depression during pre-menopause or at the onset of menopause, and rates of depression decrease in post-menopausal women (Avis et al., 1994). During perimenopause, however, women may experience more depressive symptoms than during pre- or post-menopause (Avis et al., 1994), which is thought to be due to increased fluctuations in estrogen during perimenopause (for reviews, see Schmidt, 2005; Payne, 2003). This research shows estrogen fluctuations during perimenopause are associated with increased depressive symptoms, whereas low estrogen levels post-menopause are not. It is possible that increased estrogen fluctuations are responsible for increased depression at other points in a woman's life (e.g., the menstrual cycle), as discussed below.

The Menstrual Cycle

Throughout reproductive age, women experience monthly fluctuations in sex hormones during the menstrual cycle. The menstrual cycle begins on the first day of a woman's menses, which is also the first day of the follicular phase. During the follicular phase, an ovarian follicle matures in preparation for fertilization. The follicular phase is characteristic of steadily increasing levels of estrogen until the ovulatory period, when estrogen rapidly increases (Punnonen, Nummi, Ylikorkala, Alapiessa, Karvonen, & Viinikka, 1975). Ovulation occurs around day 14 of the menstrual cycle and marks the transition from the follicular to luteal phase. During the luteal phase, the uterus prepares for implantation of a fertilized ovum. If fertilization

does not occur, the luteal phase begins with rapidly decreasing estrogen levels from the initially high level during ovulation. After this sharp decrease, estrogen levels continue to fluctuate before steadily decreasing throughout the remainder of the menstrual cycle (Punnonen et al., 1975).

The luteal phase is associated with both somatic symptoms and negative mood, which may manifest themselves in disorders such as Premenstrual Syndrome (PMS) or Premenstrual Dysphoric Disorder (PMDD). PMS and PMDD symptoms include breast pain, bloating, headache, changes in appetite and energy, and anxiety or depression (American Psychiatric Association, 2013). Logue & Moos (1986) found that at least 40% of women of reproductive age experience negative premenstrual symptoms to varying degrees, regardless of a diagnosis of a premenstrual disorder. While some researchers have found the prevalence of premenstrual symptoms to be higher than 40%, others have found a smaller prevalence, which is largely due to the strictness of criteria used for diagnosis (for review, see Halbreich, Borenstein, Pearlstein, & Kahn, 2003).

Premenstrual disorders are thought to reflect hormone fluctuations throughout the menstrual cycle being primarily responsible (for review, see Cunningham, Yonkers, O'Brien, & Erikkson, 2008). Lower levels of estradiol during the late luteal phase are closely linked to negative mood (Backstrom, Sanders, Leask, Davidson, Warner, & Bancroft, 1983; Meaden, Hartlage, & Cook-Karr, 2004; Sanders, Warner, Backstrom, & Bancroft, 1983; for review, see Yonkers, O'Brien, & Eriksson, 2008). While low estradiol may be linked to increased depressive symptoms, some research has failed to find an association between estradiol levels and premenstrual depression (Bayer, Schultz, Gamer, & Sommer, 2014; Jacobs & D'Esposito, 2011). Additionally, while Reed, Levin, & Evans (2008) found a difference in depressive symptoms

between women in the follicular and luteal phases, there was not a significant difference in estradiol levels between the follicular and luteal groups. The inconsistent association between estradiol levels and depressive symptoms indicates that estradiol levels may not entirely account for depressive symptoms, and that there may be a different explanation for the association. Fluctuations in estradiol throughout the luteal phase, irrespective of estradiol level, may also play a role in increased premenstrual depression (Bao, Ji, Van Someren, Hofman, Liu, & Zhou, 2004; for reviews, see Payne, 2003; Shors & Leuner, 2003). While this research indicates an association between estradiol and mood throughout the menstrual cycle is likely, it is unclear whether estradiol fluctuations or levels are responsible for an association.

Oral Contraceptives and Well-being

Women using hormonal contraception provide researchers with an opportunity to further explore the relationship between hormones and depression. Oral contraceptives are a type of hormonal contraception where controlled dosages of hormones are administered daily throughout the menstrual cycle. Oral contraceptive pills are designed to inhibit ovulation and reduce circulating estradiol levels (Spona, Feichtinger, Kindermann, Wunsch, & Brill, 1996), although different types of pills (e.g., monophasic, biphasic, triphasic) accomplish this through varying combinations of reproductive hormones (discussed in Walker & Bancroft, 1990). Monophasic oral contraceptives contain a stable dose of combined estrogen and progestin for the entirety of the menstrual cycle, with the exception of the menstrual period. Biphasic and triphasic oral contraceptives utilize similar estrogen and progestin hormones to monophasic pills, yet the amount of hormones, particularly progestin, varies throughout phases of the menstrual cycle in an attempt to mimic the hormone profile of a natural menstrual cycle. Typically three dosage

variations are administered for one week each in triphasic oral contraceptives, increasing the hormone dosage as the menstrual cycle progresses.

Research on the mood effects of oral contraceptives is generally inconsistent. As Böttcher, Radenbach, Wildt, & Hinney (2012) suggest, combining oral contraceptive types (e.g., monophasic and triphasic), hormone compositions, and effects of estradiol and progestin within studies may confound results and make it difficult to distinguish between the effects of estradiol fluctuations or levels. Oral contraceptive users report reduced premenstrual breast pain, bloating, impairment in functioning, and fatigue than non-users (Graham & Sherwin, 1987; Graham & Sherwin, 1992). There is also some evidence demonstrating that oral contraceptives improve negative mood, or protect against worsening mood (Keyes, Cheslack-Postava, Westhoff, Heim, Haloossim, Walsh, & Koenen, 2013; Nyberg, 2013; Toffol, Heikinheimo, Koponen, Luoto, & Partonen, 2012). Oral contraceptive users also may report fewer depressive symptoms than non-users (Graham & Sherwin, 1987; Young et al., 2007) although this finding is not consistent (for review, see Oinonen & Mazmanian, 2002). The inconsistent evidence may be due to the lower level of circulating estradiol in oral contraceptive users, in comparison to naturally cycling women, may be responsible for inconsistencies in the literature, as lower circulating estradiol is related to increased depressive symptoms (Backstrom et al., 1983). Variations in depressive symptoms throughout the menstrual cycle may also account for inconsistencies in previous research, as naturally cycling women typically experience depression prior to menstruation, and oral contraceptive users report more depressive symptoms during menstruation (Bancroft & Rennie, 1993), when exogenous hormone administration stops.

Research differentiating between monophasic and triphasic oral contraceptive users provides additional insight into the relationship between estradiol and depressive symptoms. In

comparison to triphasic oral contraceptive users and non-users, monophasic oral contraceptive users are less likely to show peaks and troughs of well-being throughout the menstrual cycle, with less cyclical changes and variability in mood (Warner & Bancroft, 1988). This stabilizing effect of oral contraceptives is evident in community samples (Ott, Shew, Ofner, Tu, & Fortenberry, 2008) and in women with treated clinical depression who reported additional premenstrual depressive symptoms (Joffe, Petrillo, Viguera, Gottshcall, Soares, Hall, & Cohen, 2007). Along with a consistent dose of estradiol across the active menstrual cycle phase in monophasic pills, exogenous progestin is also administered in stable doses. In comparison to triphasic pills and naturally cycling women, the stabilized progestin may be responsible for the stabilizing effect of monophasic oral contraceptives on mood (for review, see Oinonen & Mazmanian, 2002).

In addition to less variability in mood, monophasic oral contraceptive users may also report lower levels of depressive symptoms throughout the menstrual cycle than triphasic pill users and naturally cycling women (Bancroft & Rennie, 1993), supporting the hypothesis that stabilized hormone levels in monophasic oral contraceptives are responsible for decreased depressive symptoms. However, other studies have not reported significant differences in depressive symptoms between monophasic oral contraceptive users and non-users (Cinar, Harmanic, Demir, & Yildiz, 2012; Duke, Sibbritt, & Young, 2007; O'Connell, Davis, & Kerns, 2007; Rapkin, Morgan, Sogliano, Biggio, & Concas, 2006).

The Current Study

The literature on the association between estrogen and depressive symptoms during pregnancy, menopause, the menstrual cycle, and in oral contraceptive users provides evidence for an association between estrogens and depressive symptoms. This literature, however, does not clearly distinguish between the effects of estradiol levels and estradiol fluctuations on depressive symptoms. The purpose of this study is to see if depressive symptoms varied with oral contraceptive use. Monophasic oral contraceptives provide a controlled measure of stable, lower estradiol levels in comparison to naturally cycling women with fluctuating, higher levels of estradiol throughout the menstrual cycle. As previously noted, it is possible that depressive symptoms increase as a result of both increased fluctuations in estradiol (Bao et al., 2004; for reviews, see Payne, 2003; Shors & Leuner, 2003) and lower estradiol levels (Backstrom et al., 1983; Bancroft & Rennie, 1993; Sanders et al., 1983). It was hypothesized that, compared to naturally cycling women, oral contraceptive users would report fewer depressive symptoms if the presence of estradiol *fluctuations* contributes to depression, but more depressive symptoms if low estradiol *levels* are a factor in depression. While this sample provides power to detect a moderate to large effect of estradiol levels or fluctuations on depressive symptoms, it cannot separate these two effects.

Methods

Participants

Participants were undergraduate students at a large public university. They were selected over two consecutive semesters from a psychology subject pool for a larger research study involving oral contraceptive users, naturally cycling women, and men. Men self-selected themselves into the study by signing up online for a test session. All women initially completed a mass screening questionnaire in which they indicated if they currently use oral contraceptives. Women who did not indicate oral contraceptive use were randomly selected and individually contacted to be scheduled for a test session. Women using oral contraceptives were contacted by phone to identify the specific oral contraceptive they used (i.e., name and dosage information) to determine eligibility for further testing. Only women using monophasic and triphasic oral contraceptives were eligible for further testing. Biphasic oral contraceptives were not included in this study as the sample of biphasic pill users is much smaller than the samples of monophasic and triphasic users. Thus, there were four groups of participants: monophasic oral contraceptive users, triphasic oral contraceptive users, naturally cycling women, and men.

The four groups of participants constituted a larger research study, of which a subsample of monophasic oral contraceptive users and naturally cycling women were allotted for this study. Monophasic oral contraceptive users were included if their pill contained between 1mg and 1.5mg nortehindrone or norethindrone acetate and 10mcg to 30mcg ethinyl estradiol, in order to ensure a homogeneous hormone profile across all participants. Oral contraceptives containing between 10mcg and 30mcg of ethinyl estradiol were selected because the similar doses are all

considered to be “low dose” compared to oral contraceptives containing 50mcg or more of ethinyl estradiol (Batur, Elder, & Mayer, 2003; Rosenberg, Meyers, & Roy, 1999). A random sample of naturally cycling women was selected to match the number of oral contraceptive users.

The final sample included 80 women: 40 monophasic oral contraceptive users and 40 naturally cycling, non-pill users. All participants were between the ages of 18 and 22 (M = 19.08, SD = 0.926); most were Caucasian (88.8%) and spoke English as a first language (97.5%). Oral contraceptive users (M = 19.04, SD = 0.78) and non-users (M = 19.13, SD = 1.06) did not differ significantly in age; $t(78)= -0.43$, p=.668. Table 1 shows the frequency of oral contraceptive pill brands within the oral contraceptive group, and the corresponding hormones in each pill brand.

A power analysis indicated that a total sample size of 80 would be sufficient to detect an effect (d) of 0.63 on a 2-tailed t-test (Cohen, 1992), with an alpha of 0.05 and a power of 0.80. However, a smaller difference in depression may be likely between monophasic oral contraceptive users and non-users. As discussed below, there is only a small gender difference on the depressive symptoms measure (e.g., d=0.25), and the expected effect between estradiol and depressive symptoms will likely be smaller than the gender difference (e.g., d=0.20), as estradiol may account for part of the gender difference in depression.

Procedures

Participants completed a supervised online survey during a one-hour test session in a research laboratory and received course credit for participation.

Measures

Oral Contraceptive Use

Participants self-reported information on their oral contraceptives. Oral contraceptive name, hormone names and dosages were collected by research assistants during a phone interview, in which participants reported information directly from the oral contraceptive pill packet. Additionally, participants completed a Reproductive Health Questionnaire, which contained items about oral contraceptive use, menstrual cycle regularity, and past or current pregnancy. Participants self-reported patterns of hormonal contraceptive use during the past three months. Women currently using a hormonal contraceptive also indicated the reason for use and the type of contraceptive being used (e.g., oral/pill, patch, intrauterine, etc.), and only oral contraceptive users were included in the final analysis. Oral contraceptive users were asked to indicate the name of their current oral contraceptive, the length of time using that oral contraceptive, and the percentage of time they forget to take their oral contraceptive pill; this information was used to exclude participants from analyses. Self-reported oral contraceptive use is a reliable measure of female adolescent contraceptive use behaviors over a six-month interval prior to testing (Sieving, Hellerstedt, McNeely, Fee, Synder, & Resnik, 2005).

Depressive Symptoms

Depressive symptoms were measured by 26 out of 27 items on the Children's Depression Inventory (CDI), which measures disturbed mood, hedonic capacity, vegetative functions, self-evaluation, and interpersonal behaviors (Kovacs, 1983). One CDI item pertaining to suicidal ideation was not included, consistent with previous research (Obeidallah, McHale, & Silbereisen,

1996; Shanahan, McHale, Crouter, & Osgood, 2008) and due to IRB concerns (i.e., need for clinical intervention if a participant indicated suicidal thoughts). Although the CDI is typically used with children, adult CDI scores correlate strongly ($r = 0.81$) with a measure of depressive symptoms typically used with adults, the Beck Depression Inventory (BDI), which indicates the CDI is a valid measure of depressive symptoms in adults up to age 22 (Weir & Jose, 2007). Additionally, the structural change of 4 to 3 response options and language simplification from the BDI to the CDI is unlikely to lead to substantial differences in the responses of college students (Weir & Jose, 2007). The CDI shows adequate internal consistency (Cronbach's alphas of 0.71 - 0.89) and is a valid measure of depressive symptoms in non-clinical samples (Kovacs, 1992; Masip, Amador-Campos, Gomez-Beniot, & Gandara, 2010; Twenge & Nolen-Hoeksema, 2002). In addition to measuring depressive symptoms in general situations, the CDI assesses mood in the context of school and peer relations; both of which are important factors in a college population (Weir & Jose, 2007). Each of the 26 items was rated on a scale of 0 to 2 (e.g., *0. "I like myself"*, *1. "I do not like myself"*, *2. "I hate myself"*) to describe how the participant had felt during the past two weeks.

There is limited data on the CDI in college samples; however, data from older adolescents on the CDI may generalize to college students. The CDI was used because it was in the larger research study. Research shows a small gender difference on the CDI beginning in adolescence, with female adolescents reporting higher CDI scores than males (Chartier & Lassen, 1994; Masip et al., 2010; Shanahan et al., 2008; Schraedley, Gotlib, & Hayward, 1999; Twenge & Nolen-Hoeksema, 2002). These data includes adolescents through age 18, which may generalize to college students between the ages of 18 and 22. Some research with college students, however, did not find a gender difference on the CDI (Weir & Jose, 2007).

Data Analysis Plan

Depressive symptoms scores for each participant were created by recoding and averaging items so that higher scores reflected more depressive symptoms. CDI scores were then compared in monophasic oral contraceptive users versus naturally cycling, non-pill users with a 2-tailed independent samples t-test with Type 1 error selected to be .05.

Results

No significant difference in CDI scores was found between monophasic oral contraceptive users (M = 0.36, SD = 0.20) and naturally cycling, non-pill users' CDI scores (M = 0.30, SD = 0.22); d = 0.27; $t(78)$=1.21, p=.232, 95% CI [-0.04, 0.15]. The 95% confidence interval indicates that the size of the difference between the group means on the CDI in the population could be between d=.19 and d=.71. Therefore, it is possible that the difference between the two groups on the CDI in the population could be moderate to large, with naturally cycling females reporting fewer depressive symptoms than oral contraceptive users. Figure 1 illustrates the variability and restricted range of CDI scores within by group. Overall CDI scores ranged from 0.00 to 1.04, on a 0-2 scale. The low scores show low levels of depressive symptoms, which are typical in female college (Weir & Jose, 2007).

Discussion

The purpose of this study was to see if depressive symptoms varied with oral contraceptive use. Compared to naturally cycling women, oral contraceptive users should report fewer depressive symptoms if the presence of estradiol *fluctuations* contributes to depression, but more depressive symptoms if low estradiol *levels* are a factor in depression.

No significant difference in total CDI score was found between monophasic oral contraceptive users and naturally cycling women. The lack of significant group differences does not support either the hypothesis that increased estradiol fluctuations are associated with increased depressive symptoms or the hypothesis that decreased estradiol levels are associated with increased depressive symptoms. As illustrated in Figure 1, the lack of group differences may be due in part to the limited range of CDI scores; on a scale of 0 to 2, most participants reported CDI scores within the lowest quarter of the scale. Although there was no significant difference between groups in CDI scores, the direction of the small group difference (d = 0.27) and the possibility that the true difference in the population is moderate to large (95% CI [-0.04, 0.15]) indicates that naturally cycling women could report fewer depressive symptoms. If this trend is evident in a larger sample, it would support the hypothesis that lower estradiol levels are associated with increased depressive symptoms.

These results are inconsistent with some but not all work showing a relation between estrogen and depressive symptoms. Most evidence for the relation between oral contraceptives and depression is inconsistent and varies based on the type of oral contraceptives (e.g., only monophasic, only triphasic, or both) included in analysis (for review, see Oinonen &

Mazmanian, 2002). In contrast to these results, some studies suggest that oral contraceptive users report fewer depressive symptoms than non-users throughout the active phase of the menstrual cycle (Bancroft & Rennie, 1993; Graham & Sherwin, 1987; Young et al., 2007). There is also evidence for the stabilizing effect of oral contraceptives on mood (Ott et al., 2008; Walker & Bancroft, 1990; Warner & Bancroft, 1988; for review, see Oinonen & Mazmanian, 2002). Similar to this study, however, is research showing no difference in depressive symptoms between oral contraceptive users and non-users (Duke, Sibbritt, & Young, 2007; O'Connell, Davis, & Kerns, 2007; Rapkin et al., 2006).

There are several possible explanations for the results. First, the lack of significant findings may be due in part to mood changes across the menstrual cycle in oral contraceptive users. Bancroft & Rennie (1993) found that oral contraceptive users reported more depressive symptoms than non-users during menses. Since menstruation accounts for one quarter of the menstrual cycle in oral contraceptive users, it is plausible that one fourth of the monophasic oral contraceptive users completed the depressive symptoms measure during their menstrual period. If oral contraceptive users in this sample experienced increased depressive symptoms during menstruation, as Bancroft & Rennie (1993) found, the incidence of menses in the two weeks prior to testing (i.e., the amount of time assessed in the CDI) could counteract levels of depressive symptoms during the active pill phase. This means that, even if oral contraceptive users report fewer depressive symptoms during the active pill phase, the presence of depressive symptoms during menstruation could increase the overall CDI scores reported by oral contraceptive users. Future research could separate oral contraceptive users by those who were tested during the active phase versus menses, to determine how depressive symptoms vary during menstruation.

Second, the effects of menstrual cycle phase (i.e., follicular vs. luteal) in naturally cycling women may also explain the lack of significant findings, as the circulating level of estradiol differs between the follicular and luteal phase in naturally cycling women (Punnonen et al., 1975). Estradiol is highest at the end of the follicular phase, about one day before ovulation, and declines throughout the luteal phase. It is possible that the lower level of estradiol during the luteal phase produces more depressive symptoms than naturally cycling women would experience during the follicular phase. This decline in estradiol may account for premenstrual depression (Backstrom et al., 1983; Meaden, Hartlage, & Cook-Karr, 2004; Sanders et al., 1983; for review, see Yonkers, O'Brien, & Eriksson, 2008). Subsequently, if a large subsample of the naturally cycling women were tested mid-cycle, when estradiol levels have been at their peak within the previous two weeks (i.e., the time period assessed by the CDI), these women may report lower levels of depressive symptoms; however, there is only a small chance that a large proportion of the naturally cycling women were tested mid-cycle. If low estradiol levels are responsible for an increase in depressive symptoms, oral contraceptive users would likely report more depressive symptoms. As noted before, oral contraceptives inhibit ovulation by decreasing circulating estradiol; therefore oral contraceptive users have lower levels of circulating estradiol than naturally cycling women (Walker & Bancroft, 1990).

Third, while lower levels of estradiol may be associated with more severe depressive symptoms, increased fluctuations of estradiol may also produce the same effect. Since there was not a significant difference in depressive symptoms between oral contraceptive users and non-users, it is possible that there are two true effects of estradiol on depressive symptoms (e.g., levels and fluctuations) which cancel each other out.

Fourth, any effects of oral contraceptive pills on depressive symptoms cannot be solely attributable to estradiol. Along with estradiol, monophasic oral contraceptives contain exogenous stable doses of a progestin throughout the active pill phase (Batur, Elder, & Mayer, 2003), which may influence mood. Similar to estradiol, exogenous doses of progestin stabilize the amount of the hormone that circulates throughout the menstrual cycle in order to prevent pregnancy. Therefore, any effects seen between oral contraceptive users and non-users may be due to changes in estradiol or progestin.

A particular methodological strength of this study is the confidence in oral contraceptive information obtained from participants. Oral contraceptive information was collected when participants had their pill packets with them and could directly report the information from it. Oral contraceptive name, hormone names, and hormone dosages were reported to research assistants directly from each participant's pill package, which provides significant clarification for and confidence in the collected oral contraceptive information. While it is not guaranteed that participants read the oral contraceptive information from their pill package, we can be fairly confident that participants correctly reported their type of contraceptive (e.g., monophasic); other research studies may not differentiate between the type of oral contraceptive that participants use.

There are some conceptual and methodological limitations to this study. First it is not possible to separate the effects of estradiol fluctuations and levels. The data were not sufficient to distinguish between low and high estradiol in oral contraceptive users, nor were circulating estradiol levels measured in naturally cycling women. A research design accounting for low and high estradiol levels in both monophasic oral contraceptive users and naturally cycling women would be able to distinguish between the effects of estradiol fluctuations and circulating estradiol

level. The lack of a direct measurement of endogenous hormones in both groups also limited the ability to determine the amplitude of the difference in endogenous estradiol levels between oral contraceptive users and non-users.

Second, the methods of this study yielded insufficient power to detect the expected effect. While there was sufficient power (0.8) to detect a moderate effect (*d*) of 0.63, the sample size was not sufficient to detect the expected small effect size. The use of a 2-tailed t-test as compared to a 1-tailed test also reduced the power to detect an effect; however, a 2-tailed test was appropriate for the comparison, as effects were possible in either direction.

Third, a conceptual limitation is the small gender difference in college students. This small gender difference is found in college students, in general, and on the CDI, which may have made it difficult to detect an effect of oral contraceptive use. The gender difference on the CDI for adolescents and young adults is small, ranging from 0 to 0.22 (Chartier & Lassen, 1994; Masip et al., 2010; Shanahan et al., 2008; Schraedley, Gotlib, & Hayward, 1999). Since the difference between monophasic oral contraceptive users and naturally cycling women was expected to be smaller than the gender difference, it is possible that a small gender difference in college students limited the ability to detect the expected effect.

Fourth, the restricted range of CDI scores within the sample – mainly below 0.5 on a 0-2 scale – may also have contributed to the inability to detect a significant group difference, as there was not much depression in these participants. A sample with a wider range of depression may provide the ability to see the effects of monophasic oral contraceptives on depressive symptoms.

Results of this study have implications for future research on estradiol and depressive symptoms. While there was not a significant link between oral contraceptives and depressive symptoms in this study, it is possible that there are two opposing effects of estradiol levels and

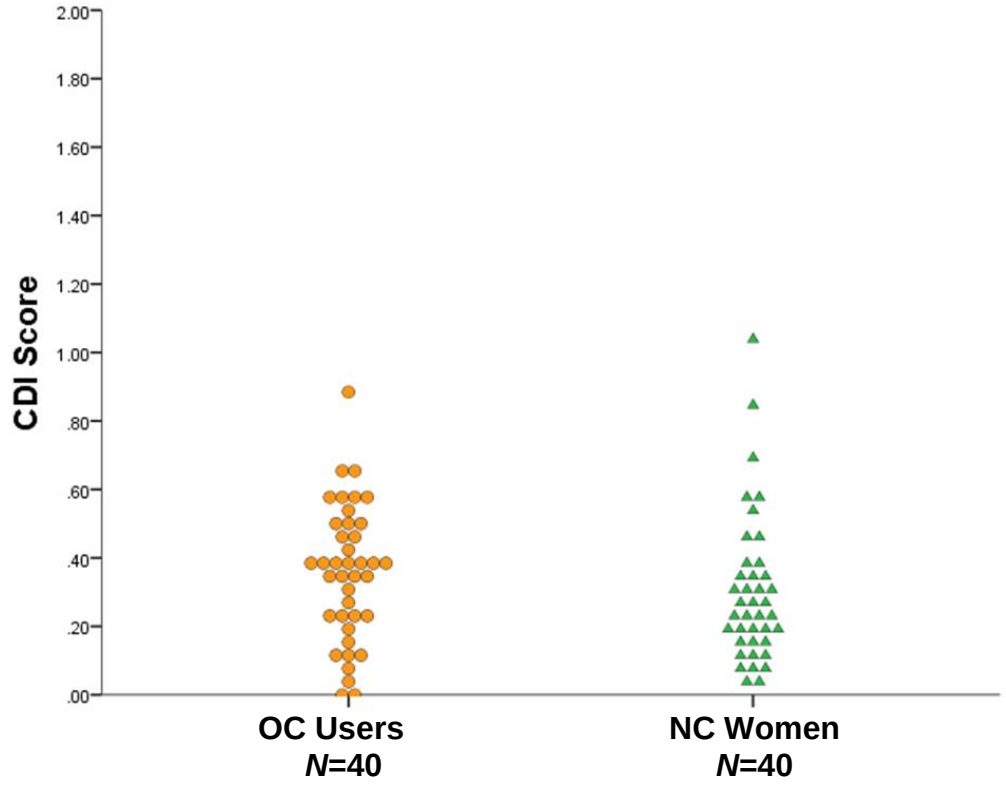
estradiol fluctuations on depressive symptoms. Future research should focus on the separate effects of estradiol fluctuations and level on depression. Research involving monophasic oral contraceptives with a variety of estradiol levels, as well as naturally cycling females who are tested during the late follicular phase, when estradiol levels are high, and late luteal phase, when estradiol levels are low, could distinguish between the effects of estradiol fluctuations and levels on depressive symptoms. Future research could also use a more sensitive measure of depressive symptoms in adults. Although it is possible that a different measure would not be significantly more sensitive at detecting effects of oral contraceptives on depressive symptoms, as the gender difference in college students may be small and depression may be low.

In conclusion, there was not a significant difference in depressive symptoms between monophasic oral contraceptive users and naturally cycling, non-pill users. The lack of significant differences on the CDI may be due to timing of testing in relation to a woman's menses, insufficient power to differentiate between the effects of estradiol fluctuations and overall level of circulating estradiol, or effects of the progestin in the oral contraceptive.

Table 1. *Frequency of Oral Contraceptive Pill Brands and Corresponding Hormones*

Oral Contraceptive	n	Progestin	Estrogen
Gildess FE	4	Norethindrone Acetate	Ethinyl Estradiol
Junel FE	11	Norethindrone	Ethinyl Estradiol
Junel FE 1/20	1	Norethindrone Acetate	Ethinyl Estradiol
Loestrin FE	7	Norethindrone	Ethinyl Estradiol
Microgestin	1	Norethindrone	Ethinyl Estradiol
Microgestin FE	10	Norethindrone Acetate	Ethinyl Estradiol
Minastrin FE	6	Norethindrone Acetate	Ethinyl Estradiol

Figure 1. *CDI Scores of Oral Contraceptive Users and Naturally Cycling Women*



Note. Each circle represents an individual CDI score. OC = Oral Contraceptive; NC = Naturally Cycling

REFERENCES

Angold, A., Costello, E. J., Erkanli, A., & Worthman, C. M. (1999). Pubertal changes in hormone levels and depression in girls. *Psychological Medicine, 29,* 1043-1053.

Angold, A., Costello, E. J., & Worthman, C. M. (1998). Puberty and depression: The roles of age, pubertal status and pubertal timing. *Psychological Medicine, 28,* 51-61.

Angold, A., Erkanli, A., Silberg, J., Eaves, L., & Costello, E. J. (2002). Depression scale scores in 8-17 year olds: Effects of age and gender. *Journal of Child Psychology and Psychiatry, 43*(8), 1053-1063.

Angold, A., & Rutter, M. (1992). Effects of age and pubertal status on depression in a large clinical sample. *Development and Psychopathology, 4,* 5-28.

Angst, J., Gamma, A., Gastpar, M., Lepine, J. P., Mendlewicz, J., & Tylee, A. (2002). Gender differences in depression. *European Archives of Psychiatry and Clinical Neuroscience, 252,* 201-209. doi: 10.1007/s00406-002-0381-6

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. doi: 10.1176/appi.books.9780890425596.dsm04

Avis, N. E., Brambilla, D., McKinlay, S. M., & Vass, K. (1994, May). A longitudinal analysis of the association between menopause and depression: Results from the Massachusetts women's health study. *Annals of Epidemiology, 4*(3), 214-220. Retrieved from http://www.sciencedirect.com.ezaccess.libraries.psu.edu/science/article/pii/1047279794900099X

Avison, W. R., & McAlpine D. D. (1992). Gender differences in symptoms of depression among

adolescents. *Journal of Health and Social Behavior*, *33*(2), 77-96. Retrieved from http://www.jstor.org/stable/2137248

Backstrom, T., Sanders, D., Leask, R., Davidson, D., Warner, P., & Bancroft, J. (1983, December). Mood, sexuality, hormones, and the menstrual cycle. II. Hormone levels and their relationship to the premenstrual syndrome. *Psychosomatic Medicine*, *45*(6), 503-507. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/6686333

Bancroft, J., & Rennie, D. (1993). The impact of oral contraceptives on the experience of perimenstrual mood, clumsiness, food craving and other symptoms. *Journal of Psychosomatic Research, 37*, *2*, 195-202. Retrieved from http://www.sciencedirect.com.ezaccess.libraries.psu.edu/science/article/pii/00223999939 0086U

Bao, A., Ji, Y., Van Someren, E. J. W., Hofman, M. A., Liu, R., & Zhou, J. (2004). Diurnal rhythms of free estradiol and cortisol during the normal menstrual cycle in women with major depression. *Hormones and Behavior*, *45*, 93-102. doi:10.1016/j.yhbeh.2003.09.004

Batur, P., Elder, J., & Mayer, M. (2003). Update on contraception: Benefits and risks of the new formulations. *Cleveland Clinic Journal of Medicine*, *70*(8), 681-696.

Bayer, J., Schultz, H., Gamer, M., & Sommer, T. (2014). Menstrual-cycle dependent fluctuations in ovarian hormones affect emotional memory. *Neurobiology of Learning and Memory*, *110*, 55-63. doi: 10.1016/j.nlm.2014.01.017

Broderick, P. C., & Korteland, C. (2002). Coping style and depression in early adolescence: Relationships to gender, gender role, and implicit beliefs. *Sex Roles*, *46*(7/8), 201-213. Retrieved from http://link.springer.com/article/10.1023%2FA%3A1019946714220

Böttcher, B., Radenbach, K., Wildt, L., & Hinney, B. (2012). Hormonal contraception and

depression: A survey of the present state of knowledge. *Archives of Gynecology and Obstetrics*, *286*, 231-236. doi: 10.1007/s00404-012-2298-2

Cinar, N., Harmanic, A., Demir, B., & Yildiz, B. O. (2012). Effect of an oral contraceptive on emotional distress, anxiety and depression of women with polycystic ovary syndrome: A prospective study. *Human Reproduction, 27*(6), 1840-1845. doi: 10.1093/humrep/des113

Cunningham, J., Yonkers, K. A., O'Brien, S., & Eriksson, E. (2009). Update on research and treatment of premenstrual dysphoric disorder. *Harvard Review of Psychiatry*, *17*(2), 120-137. doi: 10.1080/10673220902891836

Chartier, G. M., & Lassen, M. K. (1994). Adolescent depression: Children's depression inventory norms, suicidal ideation, and (weak) gender effects. *Adolescence*, *29*(116), 859.

Cohen, J. (1992). A power primer. *Psychological Bulletin*, *112*(1), 155-159. doi: 10.1037/0033-2909.112.1.155

Duke, J. M, Sibbritt, D. W., & Young, A. F. (2007). Is there an association between the use of oral contraception and depressive symptoms in young Australian women?. *Contraception*, *75*, 27-31. doi: 10.1016/j.contraception.2006.08.002

Ge, X., Conger, R. D., & Elder, G. H. Jr. (2001). Pubertal transition, stressful life events, and the emergence of gender differences in adolescent depressive symptoms. *Developmental Psychology*, *37*(3), 404-417. doi: 10.1037//0012-1649.37.3.404

Graham, C. A., & Sherwin, B. B. (1987). The relationship between retrospective premenstrual symptom reporting and present oral contraceptive use. *Journal of Psychosomatic Research*, *31*, *1*, 45-53. Retrieved from http://www.sciencedirect.com.ezaccess.libraries.psu.edu/science/article/pii/0022399987900973

Graham, C. A., & Sherwin, B. B. (1992). A prospective treatment study of premenstrual symptoms using a triphasic oral contraceptive. *Journal of Psychosomatic Research*, *36*, *3*, 257-266. Retrieved from http://www.sciencedirect.com.ezaccess.libraries.psu.edu/science/article/pii/0022399992900090O

Halbreich, U., Borenstein, J., Pearlstein, T., & Kahn, L. S. (2003). The prevalence, impairment, impact, and burden of premenstrual dysphoric disorder (PMS/PMDD). *Psychoneuroendocrinology*, *28*, 1-23. doi: 10.1016/S0306-4530(03)00098-2

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*, *107*(1), 128-140.

Jacobs, E., & D'Esposito, M. (2011). Estrogen shapes dopamine-dependent cognitive processes: Implications for women's health. *The Journal of Neuroscience*, *31*(14), 5286-5293. doi: 10.1523/JNEUROSCI.6394-10.2011

Jewell, J. A., & Brown, C. S. (2014). Relations among gender typicality, peer relations, and mental health during early adolescence. *Social Development*, *23*(1), 137-156. doi: 10.1111/sode.12042

Joffe, H., Petrillo, L. F., Viguera, A. C., Gottshcall, H., Soares, C. N., Hall, J. E., & Cohen, L. S. (2007). Treatment of premenstrual worsening of depression with adjunctive oral contraceptive pills: A preliminary report. *The Journal of Clinical Psychiatry, 68*(12), 1954-1962. Retrieved from http://www-ncbi-nlm-nih-

gov.ezaccess.libaries.psu.edu/pubmed/?term=treatment+of+premenstrual+worsening+of+depression+with+adjunctive+oral+contraceptive

Kessler, R. C., McGonagle, K. A., Swartz, M., Blazer, D. G., & Nelson, C. B. (1993). Sex and depression in the National Comorbidity Survey I: Lifetime prevalence, chronicity and recurrence. *Journal of Affective Disorders*, *29*, 85-96. Retrieved from http://www.sciencedirect.com.ezaccess.libraries.psu.edu/science/article/pii/016503279390026G

Keyes, K. M., Cheslack-Postava, K., Westhoff, C., Heim, C. M., Haloossim, M., Walsh, K., & Koenen, K. (2013). Association of hormonal contraceptive use with reduced levels of depressive symptoms: A national study of sexually active women in the United States. *American Journal of Epidemiology*, *178*(9), 1378-1388. doi: 10.1093/aje/kwt188

Kovacs, M. (1983). *Children's Depression Inventory (CDI).* Unpublished Manuscript. University of Pittsburg.

Kovacs, M. (1992). *Children's Depression Inventory (CDI)*. New York: Multi-health Systems, Inc.

Logue, C. M., & Moos, R. H. (1986, July/August). Perimenstrual symptoms: Prevalence and risk factors. *Psychosomatic Medicine*, *48*(6), 388-414. Retrieved from http://www.psychosomaticmedicine.org/content/48/6/388.short

Masip, A. F., Amador-Campos, J. A., Gomez-Benito, J., & Gandara, V. D. B. (2010). Psychometric properties of the Children's Depression Inventory in Community and Clinical Samples. *The Spanish Journal of Psychology*, *13*(2), 990-999.

Mayo Clinic. (2013). Perimenopause. *Diseases and Conditions*. Retrieved from

http://www.mayoclinic.org/diseases-conditions/perimenopause/basics/definition/con-20029473

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. doi: 10.1176/appi.books.9780890425596.dsm04

Meaden, P. M., Hartlage, S. A., & Cook-Karr, J. (2004). Timing and severity of symptoms associated with the menstrual cycle in a community-based sample in the Midwestern United States. *Psychiatry Research*, *134*(1), 27-36. doi: 10.1016/j.psychres.2005.01.003

Nobel, R. E. (2005). Depression in women. *Metabolism Clinical and Experimental*, *54*, *1*, 49-52. doi:10.1016/ j.metabol.2005.01.014

Nolen-Hoeksema, S., Larson, J., & Grayson, C. (1999). Explaining the gender difference in depressive symptoms. *Journal of Personality and Social Psychology*, *77*(5), 1061-1072.

Nyberg, S. (2013). Mood and physical symptoms improve in women with severe cyclical changes by taking an oral contraceptive containing 250-mcg norgestimate and 35-mcg ethinyl estradiol. *Contraception, 87,* 773-781. doi: 10.1016/j.contraception.2012.09.024

Obeidallah, D. A., McHale, S. M., & Silbereisen, R. K. (1996). Gender role socialization and adolescents' reports of depression: Why some girls and not others?. *Journal of Youth and Adolescence*, *25*(6), 775-785.

O'Connell, K. O., Davis, A. R., & Kerns, J. (2007). Oral contraceptives: Side effects and depression in adolescent girls. *Contraception*, *75*, 299-304. doi: 10.1016/j.contraception.2006.09.008

O'Hara, M. W., & Swain, A. M. (1996). Rates and risk of postpartum depression – a meta-analysis. *International Review of Psychiatry*, *8*(1), 37. doi: 10.3109/09540269609037816

Oinonen, K. A., & Mazmanian, D. (2002). To what extent do oral contraceptives influence mood

and affect?. *Journal of Affective Disorders, 70,* 229-240. Retrieved from http://www.sciencedirect.com.ezaccess.libraries.psu.edu/science/article/pii/S0165032701003561

Ott, M. A., Shew, M. L, Ofner, S., Tu, W., & Fortenberry, J. D. (2008). The influence of hormonal contraception on mood and sexual interest among adolescents. *Archives of Sexual Behavior, 37,* 605-613. doi: 10.1007/s10508-007-9302-0

Payne, J. L. (2003, August). The role of estrogen in mood disorders in women. *International Review of Psychiatry, 15,* 280-290. doi:10.1080/0954026031000136893

Priess, H. A., Lindberg, S. M., & Hyde, J. S. (2009). Adolescent gender-role identity and mental health: Gender intensification revisited. *Child Devlopment*, *80*(5), 1531-1544. doi: 10.1111/j.1467-8624.2009.01349.x

Punnonen, R., Nummi, S., Ylikorkala, O., Alapiessa, U., Karvonen, P., & Viinikka, L. (1975). A composite picture of the normal menstrual cycle. *Acta Obstetricia et Gynecologica Scandinavica*, *54*(51), 64-70. doi: 10.3109/00016347509156434

Rapkin, A. J., Morgan, M., Sogliano, C., Biggio, G., & Concas, A. (2006). Decreased neuroactive steroids induced by combined oral contraceptive pills are not associated with mood changes. *Fertility and Sterility*, *85*(5), 1371-1378. doi: 10.1016/j.fertnstert.2005.10.031

Reed, S. C., Levin, F. R., & Evans, S. M. (2008). Changes in mood, cognitive performance and appetite in the late luteal and follicular phases of the menstrual cycle in women with and without PMDD (Premenstrual Dysphoric Disorder). *Hormones and Behavior, 54*(1), 185-193.

Rosenberg, M. J., Meyers, A, & Roy, V. (1999, December). Efficacy, cycle control, and side

effects of low- and lower-dose oral contraceptives: A randomized trial of 20mg and 35mg estrogen preparations. *Contraception*, *60*(6), 321-329. Retrieved from http://www.sciencedirect.com.ezaccess.libraries.psu.edu/science/article/pii/S0010782499001092

Sanders, D., Warner, P., Backstrom, T., & Bancroft, J. (1983, December). Mood, sexuality, hormones and the menstrual cycle. I. Changes in mood and physical state: Description of subjects and method. *Psychosomatic Medicine*, *45*(6), 487-501. Retrieved from http://www.psychosomaticmedicine.org/content/45/6/487.short

Schmidt, P. J. (2005). Mood, depression, and reproductive hormones in the menopausal transition. *The American Journal of Medicine*, *188*(12B), 54S-58S. doi:10.1016/j.amjmed.2005.09.033

Schiller, C. E., O'Hara, M. W., Rubinow, D. R., & Johnson, A. K. (2013). Estradiol modulates anhedonia and behavior despair in rats and negative affect in a subgroup of women at high risk for postpartum depression. *Physiology & Behavior*, *119*, 137-144. doi: 10.1016/j.physbeh.2013.06.009

Schraedley, P. K., Gotlib, I. H., & Hayward, C. (1999). Gender differences in correlates of depressive symptoms in adolescents. *Journal of Adolescent Health*, *25*, 98-108.

Shanahan, L., McHale, S. M., Crouter, A. C., & Osgood, D. W. (2008). Linkages between parents' differential treatment, youth depressive symptoms, and sibling relationships. *Journal of Marriage and Family*, *70*, 480-494.

Shors, T. J., & Leuner, B. (2003). Estrogen-mediated effects on depression and memory formation in females. *Journal of Affective Disorders*, *74*, 85-96. doi:10.1016/S0165-0327(02)00428-7

Sieving, R., Hellerstedt, W., McNeely, C., Fee, R., Synder, J., & Resnik, M. (2005). Reliability of self-reported contraceptive use and sexual behaviors among adolescent girls. *The Journal of Sex Research*, *42*(2), 159-166. doi:10.1080/00224490509552269

Spona, J., Feichtinger, W., Kindermann, C., Wunsch, C., & Brill, K. (1996). Inhibition of ovulation by an oral contraceptive containing 100 micrograms levonorgestrel in combination with 20 micrograms ethinylestradiol. *Contraception, 54*(5), 299-304. doi: 10.1016/S0010-7824(96)00183-7

Toffol, E., Heikinheimo, O., Koponen, P., Luoto, R., & Partonen, T. (2012). Further evidence for lack of negative associations between hormonal contraception and mental health. *Contraception*, *86*, 470-480. doi: 10.1016/j.contraception.2012.02.014

Twenge, J. M., & Nolen-Hoeksema, S. (2002). Age, gender, race, socioeconomic status, and birth cohort differences on the Children's Depression Inventory: A meta-analysis. *Journal of Abnormal Psychology*, *111*(4), 578-588. doi:10.1037//0021-843X.111.4.578

Walker, A., & Bancroft, J. (1990). Relationship between premenstrual symptoms and oral contraceptive use: A controlled study. *Psychosomatic Medicine, 52*, 86-96. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/2305025

Warner, P., & Bancroft, J. (1988). Mood, sexuality, oral contraceptives and the menstrual cycle. *Journal of Psychosomatic Research, 32*, *4/5*, 417-427. Retrieved from http://www.sciencedirect.com.ezaccess.libraries.psu.edu/science/article/pii/0022399988900256

Weir, K. F., & Jose, P. E. (2007). Can child self-report measures of depression and anxiety be

used in college samples? *Psychological Reports, 100,* 827-837. doi: 10.2466/PR0.100.3 .827-837

Whitley, B. E. Jr.. (1984). Sex-role orientation and psychological well-being: Two meta-analyses. *Sex Roles, 12*(1/2), 207-225. Retrieved from http://link.springer.com/article/10.1007%2FBF00288048

Wichstrøm, L. (1999). The emergence of gender difference in depressed mood during adolescence: The role of intensified gender socialization. *Developmental Psychology, 35*(1), 232-245. doi: 10.1037/0012-1649.35.1.232

Wisner, K. L., Parry, B. L., Piontek, C. M. (2002). Postpartum depression. *New England Journal of Medicine, 347*(3), 194-199. doi: 10.1056/NEJMcp011542

Wisner, K. L., Sit, D. K. Y., McShea, M. C., Rizzo, D. M., Zoretich, R. A., Hughes, C. L., … Hanusa, B. H. (2013). Onset timing, thoughts of self-harm, and diagnoses in postpartum women with screen-positive depression findings. *Psychiatry, 70*(5), 490-498. doi: 10.1001/jamapsychiatry.2013.87

Yonkers, K. A., O'Brien, S., & Eriksson, E. (2008). Premenstrual syndrome. *Lancet, 371*(9619), 1200-1210. doi: 10.1016/S0140-6736(08)60527-9

Young, E. A., Kornstein, S. G., Harvey, A. T., Wisniewski, S. R., Barkin, J., Fava, M., … Rush, A. J. (2007). Influences of hormone-based contraception on depressive symptoms in premenopausal women with major depression. *Psychoneuroendocrinology, 32,* 843-853. doi: 10.1016/j.psyneuen.2007.05.013

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Jersey Optional Gymnastics Association Scholarship Recipient 2011

RELEVANT EXPERIENCE

Intern, MidStep Centers for Child Development , State College, PA — May 2014 - Present
- Administered intake screenings
- Responsible for scoring behavioral tests and additional office duties

Co-leader of a Social Skills Therapy Group, State College, PA — Fall 2014
- Facilitated a weekly group for 3rd-6th graders to develop social skills
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Teaching Assistant in Adolescent Psychology, University Park, PA — Fall 2013
- Prepared and instructed review sessions
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Tutor, Morristown, NJ and University Park, PA — 2010 - 2013
- Assisted students in the process of learning psychology and math

Assistant Counselor, Camp Brody, Morristown, NJ — 2009 - 2010
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RESEARCH EXPERIENCE

Research Assistant, Developmental Lab, University Park, PA — January 2013 - Present
- Prepared testing materials, collected, and analyzed data

LEADERSHIP EXPERIENCE

Vice President, Helping Across the Community Organization, University Park, PA — 2011 - 2014
- Developed and facilitated community service events
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Assistant Coach, Rettig's Gymnastics, Cedar Knolls, NJ — 2008 - 2011
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