J Youth Adolescence (2012) 41:1325–1338
DOI 10.1007/s10964-011-9722-y

EMPIRICAL RESEARCH

Developmental Associations Between Adolescent Change in Depressive Symptoms and Menstrual-Cycle-Phase-Specific Negative Affect During Early Adulthood

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Received: 13 August 2011 / Accepted: 7 October 2011 / Published online: 16 October 2011
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Abstract The causal factors associated with increases in depressive symptoms among adolescent girls remain an area of theoretical debate, and the limited research considering a hormonal influence has provided mixed results. The goal of the present study was to test a set of longitudinal associations, that, if found, would provide support for a hormonal contribution to these changes. Specifically, this study tested the hypotheses that changes in depressive symptoms among adolescent girls would be associated with phase-specific symptoms of the menstrual cycle during early adulthood; that these associations would differ across three phases of the menstrual cycle; and that the pattern of associations would differ for changes in depressive symptoms during early- and late-adolescence. The sample consisted of 47 women with longitudinal data from 12 to 21 years old (approximately 91% European Canadian, 4% Middle Eastern Canadian, 2% Haitian Canadian, and 2% Asian Canadian). Consistent with expectations, results showed that early-adolescent increases in depressive symptoms were negatively associated with menstrual-phase negative affect, and positively associated with mid-cycle negative affect, but not associated with premenstrual negative affect; whereas late-adolescent change in depressive symptoms was only associated with depressive symptoms at 20–21 years. Thus, early-adolescent changes in depressive symptoms are longitudinally associated with later mood change across the menstrual cycle, suggesting a common underlying cause, which is hypothesized to be hormonal. Moreover, results suggest that, with respect to variables that are involved in affective development, important differences exist between early- and late-adolescence. The discussion considers menstrual-cycle-related symptoms (e.g., dysmenorrhea) during adolescence, and the need to study their effects on development. It is suggested that focused intervention and prevention efforts may be indicated to interrupt negative developmental outcomes.

Keywords Depressive symptoms · Adolescence · Menstrual cycle · Development

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Introduction

Research unambiguously has established that adolescent girls and adult women demonstrate higher levels of depression than adolescent boys and adult men (Angold and Rutter 1992; Lewinsohn et al. 1993). Past research suggests that this difference first emerges between 13 and 15 years of age (Hankin et al. 1998), and is evident considering both depressive symptoms (Angold and Rutter 1992; Ge et al. 2001) and diagnoses (Angold and Rutter 1992; Hankin et al. 1998). Moreover, this gender difference appears to be primarily attributable to an increase in depression among adolescent girls, which is then sustained throughout adulthood (Angold and Rutter 1992; Hankin et al. 1998). Despite a wide range of theories, focusing on variables such as cognitive bias (Hankin and Abramson 2001), body image dissatisfaction (Brooks-Gunn 1988), sexual abuse (Schraedley et al. 1999), and reproductive steroids (Halbreich and Kahn 2001), a clear understanding of the causes and developmental course of this increase in depression among adolescent girls has not been reached.

Although past research has not provided strong evidence supporting the theoretical position that reproductive steroids play an important role in the affective changes observed among adolescent girls (see below), research *has* demonstrated the importance of reproductive steroids in affective changes associated with other reproductive-life-cycle events, including the menstrual cycle, the post-partum, and menopause (see Steiner et al. 2003, for a review). Moreover, individual affective responses to these reproductive-life-cycle events are associated with each other. For example, women who experience PMS are more likely to experience post-partum depression (Bloch et al. 2005), and menopausal depression (Freeman et al. 2004), and women who experience post-partum depression are more likely to experience menopausal depression (Stewart and Boydell 1993). These established associations between affective response to different reproductive-life-cycle events provide the theoretical basis for the present study. Specifically, the present study tests whether changes in girls' depressive symptoms during adolescence are associated with negative affect across different phases of the menstrual cycle, during early adulthood. Because the proposed link between early changes in depressive symptoms and menstrual-cycle-related negative affect depends on putative effects of reproductive steroids, the introduction will briefly discuss the relevant literature regarding the associations between reproductive steroids and affective symptoms, and the important individual differences in these associations.

During adolescence there are significant changes in reproductive steroids (e.g., progesterone and estrogen), with an obvious and well-established differentiation between males and females regarding levels and fluctuations. Following adolescence, women continue to experience large cyclical changes in these steroids, synchronous with the menstrual cycle. Considering that these steroids have been linked with depression (see for reviews, Halbreich and Kahn 2001; Rubinow and Schmidt 2006), as well as specific modulatory effects on neurotransmitter systems that are associated with depression, such as GABA (Epperson et al. 2002; Majewska et al. 1986) and serotonin (Dubrovsky 2005), these steroids would appear to be prime candidates for better understanding the increase of depressive symptoms among adolescent girls.

Past reviews of the literature within psychology, however, have concluded that there is little support for the hypothesis that female reproductive steroids contribute to the increase in depressive symptoms among adolescent girls (Hankin and Abramson 2001; Nolen-Hoeksema and Girgus 1994). These conclusions are based on evidence showing that levels of reproductive steroids among adolescent girls are either weakly associated with depressive symptoms (Brooks-Gunn and Warren 1989; Paikoff et al. 1991) or not at all associated with these symptoms (Susman et al. 1987; Susman et al. 1991). However, a more recent study, considering a sample of $n = 465$ girls (9–15 year olds), found strong effects of both estrogen and testosterone on depression (Angold et al. 1999). Therefore, conclusions of null effects are far from being established.

More important than the mixed results across these studies is recent work showing that individual differences in *sensitivity* to reproductive steroids (e.g., strength and valence of response), rather than their serum levels, are responsible for the affective changes associated with them (see Rubinow and Schmidt 2006, for discussion). These individual differences in sensitivity appear to vary as a function of both *strength* of response, as well as the *valence* of that response, and are highlighted by studies showing idiosyncratic responses to hormonal interventions used in clinical treatment of Premenstrual Syndrome/Premenstrual Dysphoric Disorder (PMS/PMDD). For example, the pharmacological suppression of ovarian activity results in symptom relief for about 50% of PMS/PMDD patients, but a "non-response" (Schmidt et al. 1998) or a worsening of symptoms (Bancroft et al. 1987) for the other 50% of patients. Moreover, those who improved following ovarian suppression demonstrated a return of symptoms when either progesterone *or* estrogen was pharmacologically reintroduced, and women without PMS showed no changes in symptoms during ovarian suppression or hormonal replacement (Schmidt et al. 1998). Although these studies on pharmacological interventions with PMS/PMDD patients may not seem relevant for the present study, the results are very important because they show that some women respond positively, others negatively, and others show no change, to the same hormonal alterations.

Research also has demonstrated the importance of studying individual differences in psychological changes that are synchronous with the menstrual-cycle. For example, although physical and psychological symptoms of the menstrual cycle are correlated (Kiesner 2009), some women demonstrate negative psychological changes (e.g., negative mood), others positive psychological changes (e.g., positive mood), and others no change, in relationship to the same physical symptoms of the menstrual cycle (Kiesner and Pastore 2010). Moreover, it recently was shown that, whereas 61% of a sample of 213 female university students demonstrated a premenstrual increase and a mid-cycle decrease in depression/anxiety (classic PMS pattern), about 13% of the sample demonstrated the exact opposite pattern, with a mid-cycle high and premenstrual low in symptoms, and 26% showed no cyclical variation (Kiesner 2011). These important individual differences may help explain why some past studies have not found

main effects for menstrual-cycle phase on symptoms of anxiety and depression (see, for example, Golub and Harrington 1981).

In addition to demonstrating the existence of very heterogeneous symptom-response profiles across individual women, these findings clearly indicate that simply testing for main effects of steroids and menstrual-cycle phase is not adequate, or appropriate, for understanding their potential contributions to psychological wellness. Therefore, rather than test for associations between affect and pubertal stage, or affect and menstrual phase, in the present study we test for a correlation between change in affect across puberty and change in affect across different phases of the menstrual cycle. This approach is conceptually analogous to research testing for associations between individual affective response to different reproductive-life-cycle events (as discussed above). Importantly, consistent with findings discussed above (Kiesner, 2011), this approach does not assume that all women respond in the same way to the same hormonal changes.

Present Study

In the present study, it is hypothesized that change in depressive symptoms across adolescence will be correlated with change in affect across the menstrual cycle in young adulthood. However, finding an association between affective change during adolescence and later affective change across the menstrual cycle, would not itself provide strong evidence in support of a hormonal link between these temporally distal changes. Therefore, a very specific set of hypotheses are tested, that, if confirmed, would make alternative explanations very unlikely.

These specific hypotheses derive from the general hypothesis that, for individual women, similar affective changes will be observed in moments of similar hormonal change. The hormonal changes that are relevant to the present study are those associated with (a) different developmental periods across adolescence, and (b) different phases of the menstrual cycle. For example, early pubertal maturation for girls is associated with a significant increase in reproductive steroids, which then show significant increases and decreases across the menstrual cycle, throughout adulthood. By considering different moments of pubertal development, and different phases of the menstrual cycle, it is possible to test a very specific set of hypotheses. These hypotheses are: (a) that changes in depressive symptoms during adolescence will be associated with phase-specific affective symptoms of the menstrual cycle during early adulthood; (b) these associations will differ across three phases of the menstrual cycle; and (c) the pattern of effects will differ for changes in depressive symptoms during early- and late-adolescence. Two important design aspects of the present study make possible these hypothesis tests.

The first important design aspect of the present study is that *individual response* to three phases of the menstrual cycle are measured. This is important because these separate phases are characterized by very different hormonal milieus. For example, whereas the *menstrual phase* (early follicular) is characterized by very low levels of both progesterone and estrogen, the *mid-cycle phase* (ovulatory) is associated with a spike in estrogen, but low levels of progesterone, and the *premenstrual phase* (late luteal) is characterized by high levels of progesterone, and moderate levels of estrogen. If adolescent changes in depressive symptoms (increase or decrease) are caused by changing steroid levels (developmental increases that start before menarche and continue after menarche, Dorn et al. 1999), then we should expect that those early changes in depressive symptoms would show phase-specific associations with individual response to the three menstrual-cycle phases. Specifically, it is hypothesized that increases in depressive symptoms during adolescence will be correlated positively with negative affect during the phases of the menstrual cycle that are characterized by high or increasing levels of steroids (mid-cycle and premenstrual), and a negative correlation with negative affect in the phase of the cycle that is characterized by low levels of steroids (menstrual phase). Thus, by measuring affective response to these specific menstrual-cycle phases, it is possible to test the hypothesis that associations between prior changes in depressive symptoms and later response to the menstrual cycle vary across the different menstrual-cycle phases.

The second important design aspect of the present study is that depressive symptoms are measured across adolescence, allowing us to examine changes in these symptoms during early adolescence as well as late adolescence. Specifically, in the present study we examine changes in depressive symptoms from 12–13 to 14–15 years old, and changes from 14–15 to 16–17 years old (see methods section for explanation of age groupings). Because the average age of menarche is approximately 12.5 years old, and is associated with important developmental changes in the Hypothalamic-Pituitary–Gonadal (HPG) axis (the endocrine system that governs the menstrual cycle), changes in depressive symptoms during early adolescence are temporally closer to the maturation of the HPG axis, as compared to changes in depressive symptoms that occur later in adolescence. Therefore, it could be hypothesized that changes in depression during early adolescence are more strongly linked with the changing milieu of hormones associated with the developmental maturation of the HPG axis. For example, past research has shown that adolescent girls are more vulnerable to depression,

obsessive–compulsive disorder, and eating disorders during the first year post-menarche (Bisaga et al. 2002). Therefore, it is hypothesized that early-adolescent change in depressive symptoms will be associated with later response to the menstrual cycle, and late-adolescent changes in depressive symptoms will be less strongly associated with later response to the menstrual cycle.

Therefore, the following three hypotheses are tested in this study. First, it is hypothesized that changes in depressive symptoms during adolescence will be associated with symptoms of the menstrual cycle during young adulthood. Second, it is hypothesized that these relationships will differ significantly for the three phases of the menstrual cycle, with effects differing not only in magnitude, but also in direction (increases in depressive symptoms during adolescence will be positively correlated with negative affect during the mid-cycle and premenstrual phases, and negatively correlated with negative affect during the menstrual phase). Finally, it is hypothesized that the pattern of these effects will differ for changes in depressive symptoms during early adolescence (from 12–13 to 14–15 years old) and changes in depressive symptoms during later adolescence (from 14–15 to 16–17 years old). Specifically, it is expected that the associations between changes in depressive symptoms and later menstrual cycle symptoms will be stronger when considering change in depressive symptoms across early adolescence, as compared to later adolescence.

Method

Participants

This longitudinal study began with 390 (226 females and 164 males) Grade 6 students (mean age = 12.38 years; SD = 0.42) who were enrolled in eight elementary schools from a large French-speaking school district in Canada. The Internal Review Board for Ethics in Research with Humans, at the second author's University, approved this study, and parents provided written consent for their child's participation. Approximately 75% of the available student population participated in this study. The overall sample was 90% European Canadian, 3% Haitian Canadian, 3% Middle Eastern Canadian, 2% Asian Canadian, and 2% Latino Canadian. Seventy-two percent of the participants lived with both biological parents. The sample was largely middle class, with a mean family income between $45,000 and $55,000 (CAN). Mothers and fathers had completed an average of 13.10 (SD = 2.68) and 13.20 (SD = 3.20) years of schooling, respectively. Of the original sample, 79% (186 females and 121 males) were still involved in the study 10 years later.

Although data were collected annually from 12 to 21 years old, in the present study we examine data from 12 to 17 and from 20 to 21 years old, thus excluding data from 18 and 19 years old. This was done for two reasons. First, the 12–18-year-old age range is a period during which we should expect a rapid change in girls' depressive symptoms (Hankin et al. 1998). Second, because different measures of depressive symptoms were used prior to and after 17 years old (see below) we could not have defined a variable of change that included measures across the ages of 17 years old to 18 and 19 years old.

In this study, only women who reported not taking hormonal contraception (e.g., oral contraception) at age 21 were retained in the final sample. Based on self-reported use of hormonal contraception, $n = 51$ could be included in this study, $n = 20$ women had missing data on this question, and $n = 115$ were using hormonal contraception. Of the 51 women who were eligible, 2 were excluded for missing data on the questionnaire regarding menstrual symptoms, and 2 were excluded because they reported not having a menstrual cycle. Therefore, the final sample included $n = 47$ participants.

At wave 1, the mean age for these $n = 47$ participants was $M = 12.42$ years, with a range from 11.7 to 13.6 years. Because age is an important construct when considering adolescent change in depressive symptoms, and because the wide age-range at T1 could add uncontrolled variance to the data, analyses will be conducted with all $n = 47$ participants, as well as with a reduced sample excluding participants who were $\geq$13 years old at time 1. Thus, the primary research questions will also be tested using a more homogeneous sample with regards to age. The specific age cutoff of 13 years was used because the expected age at time 1 was 12 years old.

Comparisons of women who were included in the present study ($n = 47$) and those who were not included (e.g., those who were using hormonal contraception) are presented in the end of the methods section.

Procedures

At wave 1 (Grade 6; age 12), questionnaires were completed in the classroom. Graduate research assistants were in charge of the questionnaire administration. At waves 2–7 (Grade 7–11; ages 13–17), similar procedures were followed within the high-school context, with questionnaires being completed in the school setting under the supervision of research assistants. Participants were enrolled in over 30 high-schools, however, and some assessments had to be conducted individually at the participant's home (approximately 10 cases per year) or the questionnaires had to be sent by mail (approximately 5 cases per year). After high school (ages 18 onward), assessments were conducted

individually. In most cases, the interviews took place at the participant's home. In some cases, questionnaires were sent by mail. From Grade 9 onward (age 15), participants received a gift certificate for their participation at each time point (to a movie theater, music store, or sports store).

Depressive Symptoms (ages 12–17)

A French version of the Children's Depression Inventory (CDI; Kovacs 1980; see Boivin et al. 1994, for French translation) was employed to measure depressive symptoms during the early age period. The CDI is a self-administered questionnaire assessing the severity of affective, behavioural and cognitive symptoms of depression among youth. The questionnaire includes a total of 27 items. In the present study, the suicidal ideation item was eliminated for ethical reasons. For each item, participants are asked to choose one of three statements that best describes how they have felt over the last 2 weeks (e.g., (a) "I am tired sometimes"; (b) "I am tired often"; (c) "I am tired all the time"). Individual item scores range from 0 to 2, with higher ratings indicating more severe symptoms. A sum-score across all items was calculated for each participant. Thus, the possible range of the sum-score could vary from 0 to 52. The CDI has demonstrated good reliability and has been validated using normative and clinic-referred samples (Finch et al. 1985; Fundulis et al. 1991). Internal consistency was high in this sample (average α across all ages was $\alpha = .83$).

Depressive Symptoms (ages 20 and 21)

A French version of the Center for Epidemiological Studies–Depression Scale (CES-D; Radloff 1977; see Führer and Rouillon 1989, for French translation) was used to measure depressive symptoms at the later age period. The CES-D is a self-administered questionnaire assessing the severity of depressive symptoms among youth. The questionnaire includes a total of 20 items (e.g., "I felt depressed", "I did not feel like eating; my appetite was poor"). Participants are asked to respond using a 4-point Likert scale, considering the past week, with higher ratings indicating more severe symptoms. A sum-score was calculated for each participant. Thus, the possible range of the sum-score could vary from 20 to 80. The CES-D is considered to be a good screening instrument for depression in adolescent populations (Roberts et al. 1991). Cronbach's alpha at 20 years old was $\alpha = .88$, and at 21 years old was $\alpha = .91$.

Menstrual Cycle Symptoms

Affective symptoms associated with three different phases of the menstrual cycle (premenstrual, menstrual, and mid-cycle) were measured during young adulthood (age 21) at a random moment during their menstrual cycle. These symptoms were measured using 9 items from a self-report questionnaire asking about anxiety (2 items: anxious, tense), depression (3 items: depressed, down, sad), and irritability (4 items: mood swings, irritable, nervous, aggressive). Specifically, participants were asked to use a 3-point scale (0 = not at all, 1 = somewhat, and 2 = very much) to indicate how much they usually feel each emotional state, during the days just prior to menstruation, during the days of menstruation, and during the days in the middle of their cycle. The raw item scores were combined to create the three scale scores, and the three scale scores were then combined into a single negative-affect score for each menstrual-cycle phase. Correlations among the three scale scores ranged from $r = .35$ to $r = .66$ for the premenstrual phase, from $r = .37$ to $r = .60$ for the menstrual phase, and from $r = .31$ to $r = .69$ for the mid-cycle phase (based on $n = 47$ participants included in primary analyses).

Although retrospective reports have methodological limitations, the strategy of asking subjects about symptoms across different menstrual-cycle phases has been used in past research, and has shown good reliability and validity (Hart et al. 1987; Moos 1968; Woods et al. 1982). For example, Hart et al. (1987) found that retrospective reports of *usual* premenstrual symptoms explained 21% of the variance in prospectively measured symptoms of the following menstrual cycle; whereas prospective symptom reports from one menstrual cycle explained only 14% of the variance in prospectively measured symptoms of the following menstrual cycle. Thus, retrospective accounts may be better at measuring average or general tendencies, and less influenced by idiosyncrasies of specific cycles. Also relevant, is that past research has shown that retrospective reports of premenstrual and menstrual symptoms are not significantly influenced by the specific menstrual-cycle phase in which the questionnaire was completed (Rouse 1978).

Age of Menarche and Past Hormonal Contraceptive Use

At 13 years old, participants were asked whether they had had their first menstruation (menarche), and if so at what age. This is the only age at which this variable was measured, so it only provides data on those participants who had had menarche by 13 years old. Of the $n = 47$ participants included in the analyses, $n = 36$ had had their menarche by 13 years old, with a mean age of 11.49 years. Although this provides an underestimate of the *average* age of menarche for all of the 47 participants included in the analyses, it clearly shows that this sample is within the expected range for age of menarche.

Starting at 16 years old, participants who reported being sexually active were asked whether they were using hormonal contraception. Thus, availability of these data are confounded with being sexually active at the same data collection interval. However, at 21 years old, all participants were asked whether they had ever, in the past, used hormonal contraception. Thus, responses to this question are independent of whether the individual was, or had been, sexually active. These data are used in follow-up analyses, in which hormonal contraceptive use at 16 and 17 years old, and past use at any age, are used as control variables. At both 16 and 17 years old $n = 14$ of the 47 included participants reported using hormonal contraception (although these were not the same 14 participants across both ages), and at 21 years old $n = 34$ reported that they had, at some point in the past, used hormonal contraception.

Missing Data

Although retention rates were very high across the entire study, some participants were missing data from multiple data-collection waves. For example, excluding all participants with any missing data on the variables used in these analyses resulted in a sample size of $n = 31$. Therefore, two steps were taken to address the issue of missing data. First, for the measures of depressive symptoms, data were averaged across 2-year intervals, resulting in depressive symptom scores for 12–13 years old, 14–15 years old, 16–17 years old, and 20–21 years old. In doing so, if a participant was missing data from 1 year, the score was simply the score for the available year. This helped to reduce the amount of missing data, and the number of variables and analyses. Combining depressive symptom scores across 2-year intervals resulted in a sample size of $n = 40$ (i.e., 7 participants still had missing data on at least one of the variables).

The second step used to reduce missing data was to impute missing values using the multivariate function in JMP 8.0 (SAS Institute 2010), which imputes missing data based on the full pattern of correlations present in a multivariate correlation matrix including all available data. This allowed the remaining 7 participants to also be included in the analyses.

Group Comparisons: Included vs. Excluded Participants

To compare the participants who were included in the primary analyses ($n = 47$) with those who were not included, we conducted a series of t tests using the four depressive symptom scores, and the three menstrual-cycle symptom scores, as the dependent variables. These groups are primarily differentiated by whether or not they were using hormonal contraception, although some participants were also not included in the primary analyses because of too much missing data. All results are presented in Table 1. Although, a significant difference was found for only one measurement of depressive symptoms (16–17 years old), the trend across all fours waves was that the women who were included in the analyses had slightly higher levels of depressive symptoms than those who were excluded from the analyses. No differences were found on the menstrual-cycle symptoms.

Data Analysis

The primary hypotheses, that adolescent change in depressive symptoms would be associated with later menstrual-cycle-phase-specific negative affect, are tested with Structural Equation Models (SEM). The specific structure and technical aspects of these analyses are explained in the results section, following a presentation of the bivariate correlations among all of the variables.

In addition to the SEM analyses, two sets of follow-up analyses are also conducted. The first set of follow-up analyses were conducted without imputing missing data, using simple multiple regression analyses, and excluding participants who were $\geq$13 years old at time 1. These analyses were conducted because (1) with a small sample size, concerns could be raised with regards to both imputing missing data, and using SEM analyses with a fairly high number of parameters as compared to the number of participants; and (2) to have a more

Table 1 Means, standard deviations (in parentheses), and t tests comparing women included in the analyses and those excluded

	Included	Not included	t (df)	p
Dep. symptoms 12–13	11.62 (6.4)	10.12 (6.3)	1.37 (154)	.17
Dep. symptoms 14–15	11.51 (6.7)	9.26 (6.5)	1.84 (137)	.07
Dep. symptoms 16–17	11.14 (5.8)	8.68 (5.9)	2.28 (143)	.02
Dep. symptoms 20–21	30.11 (7.6)	28.42 (6.6)	1.39 (154)	.17
Premenstrual	.66 (.49)	.67 (.51)	.18 (149)	.86
Menstrual	.46 (.35)	.56 (.45)	1.38 (149)	.17
Mid-cycle	.30 (.32)	.25 (.34)	.87 (149)	.39

"Dep. symptoms" = depressive symptoms

homogeneous sample with regards to age. Thus, these analyses provide a simplified replication using a more homogeneous sample.

The second set of follow-up analyses were conducted controlling for past use of hormonal contraception. In the primary analyses, participants who were using hormonal contraceptives at age 21 were excluded. This was done because the primary variables of interest at this age were individual affective change across the different phases of the menstrual cycle, and current hormonal contraceptive use would essentially eliminate these different menstrual-cycle phases. Similarly, past use of hormonal contraceptives could be related to changes in depressive symptoms across adolescence, thus creating a potential confound when studying this change. Therefore, follow-up analyses were also conducted testing the main hypotheses, but controlling for past use of hormonal contraceptives.

Results

Correlations

Correlations between the measures of depressive symptoms and the measures of negative affect associated with the premenstrual, menstrual, and mid-cycle phases of the menstrual cycle are presented in Table 2 ($n = 47$). Three general conclusions can be drawn from these correlations. First, measures of depressive symptoms that were temporally close to each other were strongly correlated, and the more temporally distant the measurements were made from each other, the weaker those correlations became. It should be noted, however, that different measures of depressive symptoms were used during adolescence and early adulthood, which likely contributed to differences in correlations observed across age periods. Second, all three measures of negative affect associated with menstrual-cycle phase were moderately to strongly correlated with each other, but the strongest was between menstrual and mid-cycle negative affect. Finally, the correlations between adolescent depressive symptoms (12–17 years old) and negative affect associated with the menstrual cycle, were all non-significant, although depressive symptoms at 20–21 years old were significantly and moderately correlated with negative affect during all three phases of the menstrual cycle.

Tests of Longitudinal Associations

To test the hypotheses that change in depressive symptoms during adolescence would be associated with individual response to the menstrual cycle, and to later depressive symptoms, two Structural Equation Models (Lisrel 8.7; Jöreskog and Sörbom 1996) were conducted. In the first model, change in depressive symptoms from 12–13 to 14–15 years old (T2–T1) was used to predict depressive symptoms at 20–21 years old, and negative affect associated with the three phases of the menstrual cycle. In the second model, change in depressive symptoms from 14–15 to 16–17 years old (T3–T2) was used to predict the same set of outcome variables. Thus, the only difference across the two models was the age at which earlier change in depressive symptoms was measured (see Fig. 1a, b).

In addition to these longitudinal effects, we also included the path from negative premenstrual affect to negative menstrual affect, and from negative menstrual affect to negative mid-cycle affect. These paths were included for two reasons. First, the questions used to measure negative affect associated with different phases of the menstrual cycle were part of the same questionnaire and there may have been carryover effects from one question to the next, resulting in a response bias that could have influenced these

Table 2 Correlations among measures of depression and negative affect associated with phases of the menstrual cycle

	Dep. symptoms 12–13	Dep. symptoms 14–15	Dep. symptoms 16–17	Dep. symptoms 20–21	Pre-menstrual	Menstrual	Mid-cycle
Dep. symptoms 12–13	1.00						
Dep. symptoms 14–15	.70***	1.00					
Dep. symptoms 16–17	.44**	.71***	1.00				
Dep. symptoms 20–21	.10	−.04	.27	1.00			
Premenstrual	.10	.16	.21	.39**	1.00		
Menstrual	.13	−.03	.06	.48***	.45**	1.00	
Mid-cycle	−.07	−.05	.09	.49***	.50***	.78***	1.00

** $p < .01$; *** $p < .001$, $n = 47$, "Dep. symptoms" = depressive symptoms

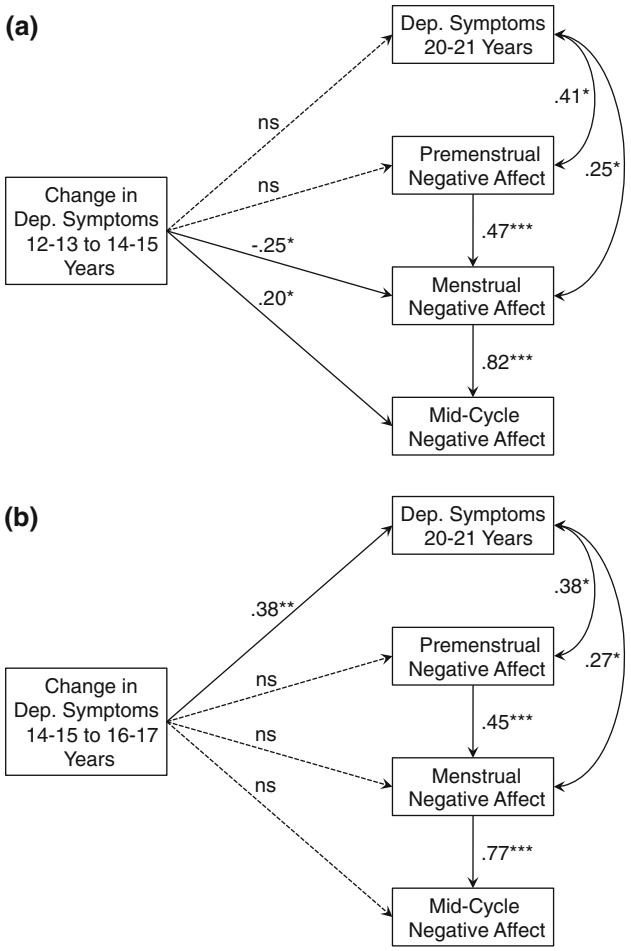

Fig. 1 Structural equation models testing for longitudinal associations between change in depression during adolescence and negative affect associated with the menstrual cycle during young adulthood (all coefficients are standardized). "Dep. symptoms" = depressive symptoms

scores. Second, there may be real affective carryover effects from one menstrual-cycle phase to another. Therefore, by including these paths, we were able to control for these potential influences, whether they be the result of response bias or actual carryover effects of mood.

Finally, we allowed the residuals of depressive symptoms at 20–21 years old to correlate with the residuals of negative premenstrual and menstrual affect. Although, depressive symptoms and negative affect associated with the menstrual cycle are conceptually different, there are obvious reasons to expect them to be correlated. Therefore, we initially tested the correlations between residuals of depressive symptoms at 20–21 years old and the residuals of all three menstrual-affect measures. The residuals of depressive symptoms and the residuals of negative mid-cycle affect were not significantly correlated, therefore, this correlation was not included in the final model.

Although RMSEA is a common fit index presented with SEM models, it is not considered a good index when *df*s and sampled sizes are small. Therefore, because in the present analyses both the *df*s and sampled sizes were small, the RMSEA is not presented.

The first model fit the data well ($\chi^2 = 3.91$, $df = 2$, $p = .14$; SRMR = .04; CFI = .97; GFI = .97). As is evidenced in Fig. 1a, change in depressive symptoms from 12–13 to 14–15 years old had no effect on depressive symptoms at 20–21 years old, or on premenstrual negative affect. However, early change in depressive symptoms did significantly predict menstrual and mid-cycle negative affect at 21 years old. More importantly, as was predicted, these two effects were in opposite directions: Whereas the path going to negative menstrual affect was negative, the path going to negative mid-cycle affect was positive. Thus, increases in depressive symptoms during early adolescence are associated with higher levels of negative affect during the mid-cycle phase and lower levels of negative affect during the menstrual phase. Both correlated residuals and both carryover effects were also statistically significant.

The second model also fit the data well ($\chi^2 = 3.85$, $df = 2$, $p = .15$; SRMR = .05; CFI = .97; GFI = .97). Moreover, as was expected, the pattern of results was very different for this model, as compared to the first model. As is evidenced in Fig. 1b, change in depressive symptoms from 14–15 to 16–17 years old was not associated with negative affect during any of the menstrual-cycle phases. However, it was positively associated with depressive symptoms at 20–21 years old. Thus, increases in depressive symptoms during late adolescence is only associated with later depressive symptoms that are not associated with the menstrual cycle. Both correlated residuals and both carryover effects were again statistically significant.

Follow-up Analyses

Because the sample size was small, concerns could be raised with regards to (1) imputing missing data, and (2) using SEM analyses with a fairly high number of parameters as compared to the number of participants. Therefore, further analyses were conducted without imputing missing data, and using simple multiple regression analyses. Moreover, as stated in the methods section, because the time-1 age-range was fairly large (11.7–13.6 years), potentially biasing or attenuating the estimated effects, those participants who were ≥13 years old at time 1 ($n = 7$) were excluded from these analyses. Finally, to make these analyses comparable across the two dependent variables, only participants who could be included in both analyses were included. The resulting sample for the present analyses was $n = 33$.

In these analyses, change in depressive symptoms during adolescence was used as the dependent variable, with depressive symptoms at 20–21 years old, and negative affect associated with the premenstrual, menstrual, and mid-cycle phases of the menstrual cycle, used as predictor variables. Although it is counterintuitive to use an earlier change score as the dependent/outcome variable, and the later measures from young adulthood as the predictors, these analyses provide valid statistical tests of the same basic associations that were tested in the SEM models. That is to say, these analyses test for associations between changes in depressive symptoms during adolescence and later phase-specific menstrual-cycle symptoms, while controlling for the associations among the menstrual-cycle symptoms. Thus, these analyses provide a simplified replication of the original analyses, without imputing missing data. It should be noted that, the theoretical basis of this study is not that early depression causes later menstrual-cycle symptoms, or that later menstrual-cycle symptoms cause earlier depression, but that both sets of symptoms share an underlying causal mechanism (sensitivity to steroid fluctuations). Therefore, it is not necessary, or even expected, that the analyses provide significant effects in one direction, but not the other.

In the first model, using change in depressive symptoms from 12–13 to 14–15 years old as the dependent variable, the overall model was significant ($F(4, 28) = 2.93$, $p < .03$, $R^2 = .29$), and the pattern of effects and significance tests replicated the findings of the SEM model. Specifically, whereas menstrual and mid-cycle negative affect showed significant associations with early-adolescent change in depressive symptoms that were in the same directions as those observed in the SEM model ($\beta_{\text{menstrual}} = -.80$, $p = .009$; $\beta_{\text{mid-cycle}} = .65$, $p = .02$), depressive symptoms at 20–21 years old and premenstrual negative affect showed no significant associations.

In the second model, using change in depressive symptoms from 14–15 to 16–17 years old as the dependent variable, although the overall model was not significant ($F(4, 28) = 1.94$, $p = .13$, $R^2 = .22$), the pattern of effects and significance tests exactly replicated the findings of the SEM model. Specifically, depressive symptoms at 20–21 years old was the only variable to show a significant association with late-adolescent change in depressive symptoms ($\beta_{\text{Dep 20–21}} = .50$, $p = .01$). None of the menstrual-cycle negative-affect variables showed a significant association.

These simplified analyses with no imputed data, and excluding participants who were older than 13 years old at time 1, replicated the findings that were observed in the SEM analyses, and lead to the same conclusions. The only apparent difference was in the magnitude of the regression/path coefficients, which were of larger magnitude in the multiple regression models.

Finally, although women who were using hormonal contraception at 21 years old were excluded from all of the above analyses, *past* use of hormonal contraception was not controlled for in these analyses. Therefore, further multiple regression analyses (similar to those used above) were conducted including the three measures of past hormonal contraception use (at 16, 17 years old, and ever used in the past). Thus, these analyses test for replication of the initial findings while controlling for possible effects of past use of hormonal contraception. The inclusion of these three variables (in three separate sets of analyses) did not change the results either with regards to significance tests or magnitude of effects. Moreover, no main effects of past hormonal contraceptive use, or interactions with any of the other variables, were found on either of the depressive-symptoms change scores. Therefore, past use of hormonal contraceptives did not bias or affect the pattern of results found in the primary analyses.

Discussion

Past research on changes in depressive symptoms among adolescent girls has given mixed results regarding the potential role of reproductive steroids (Angold et al. 1999; Brooks-Gunn and Warren 1989; Paikoff et al. 1991; Susman et al. 1987, 1991). Other research, however, showing associations between affective response to different reproductive-life-cycle events (Bloch et al. 2005; Freeman et al. 2004; Stewart and Boydell 1993), provided the theoretical basis for hypothesizing that changes in depressive symptoms across early adolescence, but not late adolescence, would be associated to menstrual-cycle symptoms in early adulthood. Results supported this hypothesis, thus providing indirect support for a putative role of reproductive steroids on changes in depressive symptoms among adolescent girls.

More specifically, the present study tested the hypotheses that changes in depressive symptoms during adolescence would be associated with affective symptoms of the menstrual cycle during adulthood; and that these associations would differ across three phases of the menstrual cycle; and that the pattern of effects would differ for changes in depressive symptoms across early- and late-adolescence. Results confirmed all three hypotheses, showing that early-adolescent increases in depressive symptoms were associated with later menstrual-cycle-phase-specific negative affect, whereas late-adolescent change in depressive symptoms was associated only with later depressive symptoms.

Similar to previous studies showing associations between individual affective response to diverse reproductive-life-cycle events (menstrual cycle, post-partum, menopause; Bloch et al. 2005; Freeman et al. 2004; Steiner et al. 2003; Stewart and Boydell 1993), the present study has demonstrated an association between affective change during early adolescence (pubertal maturation) and phase-specific symptoms of the menstrual cycle. Thus, it is argued that these findings provide support for a hypothesized effect of reproductive steroids on changes in depressive symptoms among adolescent girls. Specifically, because past research has demonstrated that hormonal changes play an important role in menstrual-cycle related affective changes, and because both the menstrual cycle and early adolescence are associated with significant hormonal changes, these hormonal changes provide a possible explanation for the observed association between adolescent change in depression and menstrual-cycle related change in affect. Thus, this follows the same logic as inferring hormonal involvement in explaining the associations, for example, between post-partum depression and menopausal depression. Although this support is indirect, the specificity of the hypotheses, and the close fit between the hypotheses and results, further support this interpretation. An alternative explanation would need to account for this specificity in the pattern of results, which will be discussed below.

One important aspect of this specificity regards the associations between early adolescent changes in depressive symptoms and negative affect during the *menstrual* and *mid-cycle* phases. As expected, an increase in depressive symptoms from 12–13 to 14–15 years old (a developmental period associated with increases in reproductive steroids) was associated with more negative affect during the *mid-cycle* phase (when estrogen is increasing) and lower levels of negative affect during the *menstrual* phase (when all reproductive steroids are at their lowest levels). Thus, girls who experience an increase in depressive symptoms during *early adolescence* are also observed to show high levels of negative affect during the specific phase of the menstrual cycle that is associated with an increase in estrogen, and low levels of negative affect during the menstrual-cycle phase associated with low levels of both estrogen and progesterone.

Although early adolescent changes in depressive symptoms were associated with later *menstrual* and *mid-cycle* negative affect, these early changes in depressive symptoms were not associated with later *premenstrual* affect. Because the current theoretical postulation would have predicted this association, this null association needs to be discussed. One possible explanation is that, because premenstrual affective changes (depression, anxiety) are common and openly discussed, they may be considered normative, as compared to menstrual and mid-cycle symptoms. As a result, there may be a response bias to conform to expectations, or an attentional/memory bias affecting the measure of premenstrual symptoms. Although research has shown that manipulations to increase saliency and expectations of premenstrual symptoms has little effect on reports of these symptoms (Gallant et al. 1992; Olasov and Jackson 1987), these biases may be present at baseline. For example, research also has shown that retrospective accounts of premenstrual symptoms likely overestimate the extent of premenstrual symptoms, at least prior to completing a daily symptom questionnaire for one entire menstrual cycle (Endicott and Halbreich 1982), after which these overestimates disappear. Such biases may create an additional source of variance that could wash out actual associations between premenstrual affect and earlier changes in depressive symptoms. Further research will be needed to better understand whether or not premenstrual symptoms are associated with changes in depressive symptoms during early adolescence.

A second aspect of the specificity of these results, in relationship to the theory-driven expectations, regards the distinction between changes in depressive symptoms during early *vs.* late adolescence. In addition to the associations discussed above, that were specific to early adolescence, it also was found that changes in depressive symptoms *only during late adolescence* were associated with early-adult depressive symptoms. Although temporal proximity may explain part of this difference, it is unlikely to explain all of it. First, the strength of association between *change* in depressive symptoms from 14–15 to 16–17 years old and depressive symptoms at 20–21 years old ($\beta_{SEM\ Model} = .38$; $\beta_{Multiple\ Regression} = .50$) was substantially larger than the bivariate correlation between depressive symptoms at 16–17 years old and depressive symptoms at 20–21 years old ($r = .27$). Thus, proximity of measurement cannot explain this difference. Second, and possibly more importantly, if temporal proximity were the primary cause for this difference, it should have created a similar bias on the results regarding the prediction of menstrual-cycle symptoms—which it clearly did not.

The overall pattern of these findings supports the hypothesis that changes in depressive symptoms during early adolescence and late adolescence may be associated with different causal factors. Specifically, as we have hypothesized, changes in depressive symptoms during early adolescence may be more strongly associated with hormonal fluctuations, and changes in depressive symptoms during later adolescence may be more strongly associated with other developmental challenges across domains of the individual's life (e.g., social relationships, social roles, cognitive processes, negative life events). Although this hypothesis is speculative, future research and theory should consider this as a possibility.

Although this study provides indirect evidence for involvement of reproductive steroids in the development of depressive symptoms among adolescent girls, the mechanism of such effects are not addressed in the present study. One possible mechanism is that reproductive steroids have direct effects on the affective centers of the brain, and that these effects are relatively stable over time. If so, the changes in depressive symptoms during adolescence, and later mood fluctuations associated with the menstrual cycle, may be indications of a similar response profile to the general case of changing steroid levels. In one case these changes are across an extended developmental period (adolescence), and in the other case these changes are rapid cyclical fluctuations associated with the menstrual cycle. This hypothesis is made plausible by the known neuroactive effects of these steroids (Dubrovsky 2005).

Other causal mechanisms, however, could be postulated focusing on individual response to the menstrual cycle during adolescence. For example, one possible mechanism is that some adolescent girls may experience higher levels of depressive symptoms as a result of negative physical symptoms associated with the menstrual cycle (e.g., cramps, headaches). These effects may then be cyclically replicated in early adulthood. Thus, the proximate cause of change in depressive symptoms, both during adolescence and early adulthood, would be the physical symptoms associated with the menstrual cycle. Although possible, past research has not supported this *physical distress* hypothesis regarding psychological symptoms of the menstrual cycle during early adulthood (Kiesner and Pastore 2010).

A third possible mechanism involves changes in school performance and/or social interactions, which may fluctuate across the menstrual cycle. Changes in either of these areas may lead to frustration, disappointment, and misattributions regarding individual ability and or likability, thus leading to depressive symptoms. These early changes in depressive symptoms could then also be associated with later menstrual-cycle related symptoms. The limited research on this area has focused on the associations between dysmenorrhea (painful symptoms such as cramps, nausea, and headaches during menstruation) and school and social variables. For example, Klein and Litt (1981) analyzed data from a national probability sample ($n = 2{,}699$; 12–17 years old), and found that 14% of the sample reported missing school because of dysmenorrhea symptoms. In another study, including $n = 384$ menstruating 15–17 year olds, 80% reported dysmenorrhea symptoms, of whom 45% reported that their school activities were limited by their dysmenorrhea, 48% reported that their sporting activities were limited by their dysmenorrhea, and 46% reported that their social life was limited (Hillen et al. 1999). Research has also shown that many adolescent girls experience cyclical changes in mood and physical symptoms, similar to adult women (Derman et al. 2004; Dorn et al. 2009; Vichnin et al. 2006). Although nothing is known about the developmental consequences of these experiences, it is easy to imagine that such significant levels of distress would have long-term developmental consequences. It is surprising that more research has not focused specifically on *menstrual-cycle related symptoms* in relationship to development of social, physical and psychological wellness during adolescence.

A promising aspect of understanding the associations between long-term developmental outcomes and physical and psychological distress associated with puberty and the menstrual-cycle, is the potential for developing intervention and prevention programs focused on these specific developmental challenges. For example, programs could focus on inaccurate attributional biases regarding menstrual-cycle-related changes in individual performance and mood, which, if unaddressed, may lead to self-blame and low self-esteem. However, before attempting such programs, much more research is needed to understand the presence of symptoms, the longitudinal effects across adolescence, the underlying mechanisms, and the wide range of individual differences in these processes.

Although the paths controlling for carryover effects and correlated residuals were not central to our hypotheses, comment should be made regarding the magnitude of these associations. Specifically, whereas 3 of the 4 associations were of moderate strength (.25 to .47), the path from menstrual affect to mid-cycle affect was much stronger (.77 and .82). Two explanations could be considered for the magnitude of this association. First, women may have difficulty *recalling* differences between these two phases of the menstrual cycle. Second, although the mid-cycle is associated with a rapid increase and decrease in estrogen, this peak in estrogen may not last long enough to result in significant affective change—thus resulting in affective *stability* across these two phases. For example, research on migraines has suggested that, in order for estrogen withdrawal to trigger menstrual-cycle related migraines, high levels of estrogen must be sustained for several days (Somerville 1975). However, although menstrual-phase negative affect and mid-cycle negative affect were strongly correlated, these variables did show very different associations (opposite valence) with early-adolescent change in depressive symptoms, as expected. Thus, although strongly correlated, important differences were found between menstrual and mid-cycle affect, in their associations with early adolescent change in depressive symptoms.

Five limitations of this study should be considered. The first limitation is that no overall increase in depressive symptoms across adolescence was found among this sample of girls, as would have been expected. Thus, this

sample may have experienced lower levels of change in depressive symptoms as compared to the general population of adolescent girls, possibly resulting in attenuated effect sizes. As a result, the generalizability of the present findings is somewhat limited, and replication is needed. It should be noted, however, that the hypothesized effects were observed, thus showing that individual change did occur and was associated with the expected variables. Therefore, the lack of group-level change in depressive symptoms did not limit our ability to test our hypotheses.

A second limitation is the lack of hormonal measures in a study that postulates hormonal mechanisms. However, although measuring steroids would add information, and allow the verification of ovulatory cycles and changes in steroids during adolescence, main effects of hormone levels should not be expected. For example, as noted in the introduction, some women respond positively, some negatively, and some experience no affective change to the same hormonal fluctuations. Thus, a direct test of the present theoretical position would require repeated measures of hormones over many occasions, allowing for reliable estimates of the association between hormone level and individual affective change—within each individual participant. By using inexpensive and non-invasive measures of steroids (e.g., saliva), such an approach is becoming more feasible, but remains prohibitively expensive when frequent repeated measures are used with adequately large samples.

A third limitation is that the $n = 47$ participants included in these analyses were selected because they were not using hormonal contraception, whereas a majority of the sample was using hormonal contraception ($n = 115$). Although differences were tested for on depressive symptoms and menstrual-cycle related affective change, other differences may exist that could not be tested for in the present study. For example, there are many reasons for using hormonal contraceptives, including birth control, managing physical and psychological symptoms of the menstrual cycle, and suppressing the natural menstrual cycle for other reasons (e.g., management of anemia, endometriosis, competitive sports). Similarly, there are various reasons for not using hormonal contraceptives, including cost, a preference for "safer" methods of contraception (hormonal contraceptives only protect against pregnancy and provide no protection against sexually transmitted diseases), and the occurrence of PMS types of physical and psychological symptoms resulting from the hormonal contraceptives themselves. Knowing the motivations for using or not using hormonal contraceptives may provide a better understanding of the possible differences between these groups. Future research should attempt to obtain this information.

The fourth limitation regards group differences between the included and excluded participants on depressive symptoms during adolescence. Specifically, it was found that the included participants, as compared to the excluded participants, tended to show higher levels of depressive symptoms across adolescence, although this difference was significant only at one age period (16–17 years old). However, because these differences were small and mostly non-significant, it seems unlikely that either the internal validity or the external validity of this study is significantly threatened by these small group differences.

A final limitation is that the number of participants included in the theoretical analyses was fairly small ($n = 47$), which may result in low statistical power and limited generalizability. However, because most of the expected effects were statistically significant, the issue of low statistical power does not appear to have created problems. Moreover, because many young women use hormonal contraceptives, and because response to the menstrual cycle can only be studied in the absence of these forms of contraception, having longitudinal data on large samples of women who do not use hormonal contraceptives will be challenging. Nonetheless, to have stronger conclusions that can be generalized with more confidence, these results should be replicated with a larger sample.

Although the above limitations need to be acknowledged, the strengths of this study also need to be highlighted. First, this study is based on longitudinal data from the ages of 12–21 years old. Few studies follow participants for such an extended period, and these data provided a unique opportunity to examine changes in depressive symptoms across adolescence in relationship to menstrual-cycle response in young adulthood. Second, this study used a novel approach by examining the associations between adolescent changes in depressive symptoms and later menstrual-cycle-phase-specific changes in affect. These methodological and design characteristics allowed us to test for associations linking biological/hormonal processes with developmental changes in affect, in a way that has never before been considered. The dearth of research on this topic, and the promising results of the present study, underline the importance of pursuing these associations in future research.

In conclusion, the present study provides evidence that change in depressive symptoms among adolescent girls is associated with individual response to the menstrual cycle. These findings are important because past research only sporadically has examined the possible contribution of reproductive steroids to the increases in depressive symptoms among adolescent girls, and has provided mixed results. In the present study, rather than examining the main effects of steroids or the menstrual cycle on mood, we examined affective change across adolescence and across three different phases of the menstrual cycle. This provides a novel and theoretically justified strategy for examining

how short-term changes (e.g., menstrual cycle) and long-term changes (e.g., pubertal maturation) in reproductive steroids may contribute to individual wellness among adolescent girls and young adult women. These findings suggest that future research should consider carefully how increasing and fluctuating levels of reproductive steroids influence the developing wellness of adolescent girls. A better understanding of these developmental effects may provide opportunities for intervention and prevention programs that focus on correct attributions regarding emotional, behavioral and relationship difficulties that may be caused by individual response to hormonal changes.

Acknowledgments This study was supported by research grants from the Social Sciences and Humanities Research Council of Canada and from the Fonds Québécois pour la Recherche sur la Société et la Culture.

Conflict of interest The authors declare that they have no conflicts of interest.

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