Bidirectional relationship between intraindividual changes in behavioral activation and intraindividual changes in postpartum depressive symptoms: A random intercept cross-lagged panel model

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Abstract

According to Lewisohn's model of depression, decreases in behavioral activation (BA) occurring after facing a vital stressor may increase the risk of depression. Transition to parenthood is a potentially stressful life event that increases the risks of postpartum depression. We aimed to (1) describe the changes in BA and depressive symptomatology between the prepartum period, one month, three months, and six months postpartum and (2) evaluate the bi-directionality of the relationship between intraindividual changes in BA and intraindividual changes in depressive symptoms longitudinally. Chilean pregnant women (*N* = 503) completed a battery of questionnaires when they were between 32 and 37 weeks of gestation, and one, three, and six months after delivery. A repeated measures ANOVA showed that BA significantly decreased from the prepartum to one month postpartum. A Random Intercept Cross-lagged Panel Model (RI-CLPM) supported the bidirectional inverse relationship between intraindividual changes in BA and intraindividual changes in depressive symptoms. The effect sizes of these associations were large (*ß*s ranging from -.141 to -.243) according to Orth et al. (2022)'s recommendations for cross-lagged effect benchmarks. This relationship showed robustness when multi-group RI-CLPMs were conducted to adjust for several covariates (i.e., marital status, the type of health insurance, type of delivery, primiparous versus multiparous participants, and pregnancy or delivery complication or newborn health problem). Nonetheless, reporting a previous history of major depression moderated this relationship so that intra-individual decreases in BA more likely led to intra-individual increases in depressive symptoms in people with a history of depression than in people without such a history. We discuss implications for behavioral models of depression.

Keywords: postpartum, depression, behavioral activation, RI-CLPM.

General Scientific Summary

Models of depression conceptualize behavioral activation as a key concept, but its role in postpartum depressive symptoms has not been fully investigated. This study analyzes the cross-lagged effect of behavioral activation and depressive symptoms in a sample of post-partum women. Results of the study support the cross-lagged, bidirectional inverse relation between depression and behavioral activation.

Bidirectional relationship between intraindividual changes in behavioral activation and intraindividual changes in postpartum depressive symptoms: A random intercept cross-lagged panel model

Postpartum depression (PPD) is a common complication of childbearing that is of major public health concern. PPD is impactful due to the potential long-term effects on the mother, her child, and their relationship. Women with PPD may encounter difficulties coping with daily life and parenting tasks, which may result in long-term persistent depressive symptoms (Mendoza & Saldivia, 2015). These complications can lead to troubled mother-child relations that may prove to be detrimental to the child's emotional, behavioral, and cognitive development (Mendoza & Saldivia, 2015). Untreated maternal depression also puts mothers at a higher risk for smoking, alcohol or substance abuse, and physical, emotional, or sexual abuse when compared to non-depressed mothers (Fitelson et al., 2010). Therefore, attempting to better comprehend the factors that potentially play a role in the development and maintenance of PPD is fundamental. One of these potential factors is behavioral activation (BA) (Dimidjian et al., 2017).

Researchers have undertaken a multitude of randomized controlled trials that show that BA and depression are linked. Some of the most compelling pieces of evidence that advocate for the strong relationship between BA and depressive symptoms are the abundant studies showing the effectiveness of Behavioral Activation Therapy (Cuijpers et al., 2007; Ekers et al., 2014). Preliminary research suggests that BA mediates the effect of behavioral activation therapy on depressive symptoms (Dimidjian et al., 2017; Nasrin et al., 2017; Santos et al., 2019). For example, Nasrin et al. (2017) investigated the impact of a single session of behavioral activation therapy (n =22) on depressive symptomatology compared with a waiting list control (n = 24). They found that self-reported change in BA significantly mediated the effect of the treatment on symptoms. Similarly, Dimidjiam et al. (2017) conducted a randomized clinical trial with pregnant women assigned to a BA intervention *(n* = 86) or treatment as usual (*n* = 77). They found that early engagement in BA (from baseline to ten weeks) mediated subsequent depression symptoms severity improvement (from 10 weeks to three months postpartum). Finally, Santos et al. (2019) investigated BA as a potential mechanism of change in the context of a BA-focused residential treatment intervention for mood problems using repeated measures of self-reported BA and depression from a large comorbid sample (*N* = 578). Using growth curve modeling, they observed that both linear and quadratic changes in BA were associated with the linear and quadratic rate of change of depression. The results of these studies suggest that BA and depressive symptoms may be causally linked. However, these studies have some limitations, including their exclusive focus on treatment samples, and more research about the causal link between BA and depression is still needed.

According to Lewisohn's model of depression, BA decreases after a vital stressor. These decreases in BA theoretically lead to an array of changes that culminate in an increase in depressive symptoms which, in turn, further disrupt the individual's behavioral and emotional response, leading to a bidirectional association over time (Dimidjian et al., 2011; Lewinsohn & Shaffer, 1971). Only two studies have explored the association between BA and depressive symptoms outside the context of therapy (Shudo et al., 2017; Wagener et al., 2016). Wagener et al. (2016) used a correlational design to study the influence of BA on different depressive symptoms in a sample of adults (*N* = 1169) recruited from adult communities or mental health care facilities. In line with Lewisohn's theory, their findings suggest that BA is negatively correlated with most depressive symptoms present in both genders, with slight differences in magnitude according to their gender. Nonetheless, the cross-sectional design of this study does not allow us to infer a temporal relation between these variables. Shudo et al. (2017) longitudinally assessed self-reported BA level and depressive symptoms at two different times (T1, T2) over an 8-week time interval in a sample of undergraduate students (*N*=129). After adjusting for previous levels of depressive symptoms as well as previous and current avoidance, BA at T1 was negatively associated with depressive symptoms at T1, but it was not a predictor of depressive symptoms at T2. Shudo et al. (2017) interpreted these findings as evidence that an individual's reported level of BA may alleviate current symptoms of depression but does not buffer against the future occurrence of depression. According to this view, non-intentional changes in BA occurring outside of a treatment context would not be causally related to changes in depressive symptoms. Nonetheless, the study by Shudo et al. (2017) had some important limitations. First, the sample selected was a group of undergraduate students without a standardized and uniform stressor, a key concept of behavioral models of depression (Dimidjian et al., 2011). Another limitation is the lack of extended follow-up, as data was only collected twice. To evaluate the form of change of variables, current methodological guidelines for longitudinal research suggest a minimum of three measurements (Kehr & Kowatsch, 2015). In sum, to our knowledge, only the two previously mentioned studies have examined the association between BA and depressive symptoms outside of the context of treatment, but in none of these studies, participants were assessed after a vital stressor, suggesting the need to further investigate the relationship between BA and depressive symptoms in longitudinal samples with baseline exposure to vital stressors.

Accordingly, the main goal of this study was to examine the relationship between BA and PPD in women going through pregnancy and postpartum. We chose to perform our study with this sample because the transition to parenthood is a stressful life event (Barimaniet et al., 2017; Saxbe et al., 2018; Simpson et al., 2003) characterized by both personal and familial changes that require an important adjustment. Taking care of a baby can result in profound changes in an individual's lifestyle, generating changes in recreational time, sleep patterns, relationships, and even identity (Epifanio et al., 2015), intimately affecting women's patterns of behavior and increasing the risks of PPD. BA involves engaging in activities that have meaning (that are tied to one´s life values) or provide enjoyment or a sense of competency. Transitions to motherhood may produce changes in the type of behaviors women perform. For some women, these changes may represent a reduction in BA because they involve abandoning, at least temporarily, some important and valued activities (i.e., sleeping, hobbies, job activities, friendships, etc.) to focus on the newborn. This decrease in BA may put them at risk for postpartum depression. Despite these hypotheses being in line with current behavioral theories of depression, to our knowledge, they have never been empirically tested. Therefore, we first aimed to separately describe the changes in BA and depressive symptomatology between the prepartum period, one month, three months, and six months postpartum in a sample of Chilean women. We expected a significant decrease in BA levels between the prepartum and postpartum periods, as well as a significant increase in depressive symptomatology. Secondly, we evaluated the bi-directionality of the relationship between intraindividual changes in BA and intraindividual changes in PPD symptoms longitudinally. We expected to observe that when women have lower than their expected individual level of BA, they will experience a subsequent increase relative to their expected individual level of depressive symptoms; and vice versa, that when women present higher than their expected individual level of depressive symptoms they will experience a subsequent decrease relative to their expected individual level of BA. In other words, we hypothesized a prospective negative effect of temporary deviations in BA on the temporal deviations from expected depressive symptoms and a prospective negative effect of temporary deviations from the expected level of depressive symptoms on the temporal deviations from the expected level in BA.

Given that childbirth is a relatively common experience and that postpartum depressive symptoms have numerous aversive effects for both mother and child, investigating the relationship between BA and postpartum depressive symptoms, including subclinical postpartum depressive symptoms, is important and informative. The present study contributes to understanding whether decreases in BA after exposure to a potentially stressful event are a risk factor for depression, providing a strong test of current behavioral theories of depression outside the context of therapy.

Method

Participants

This study used data collected in a prospective pregnancy-pain-related longitudinal study. Women (*N*=504) who were 32 to 37 weeks pregnant were recruited from obstetrics and gynecology services, at a health network service in the Santiago Metropolitan area in Chile. Exclusion criteria included being younger than 18 years old, having trouble speaking Spanish, presence of chronic pain before pregnancy, and non-viability of the fetus. The data of one of the participants was excluded from the database because her baby passed away during the postpartum period; therefore, analyses were conducted with a sample size of 503.

Recruitment and procedure

Research assistants approached and invited women to participate while they waited to be attended to by their physicians. Alternatively, they were introduced to the study by a nurse or doctor or volunteered to participate in the study after encountering advertisements posted in the waiting rooms. To avoid participants feeling pressured to take part in the study, a structured verbatim speech was designed.

The design consisted of four interviews: an initial face-to-face interview at 32 to 37 weeks of pregnancy, and three follow-up phone calls at one month, three months, and six months postpartum (see Figure 1). Once the individual decided to participate, a more detailed explanation of the study was provided, and a consent form was signed. The first interview was conducted immediately after consent was given and it took 40 to 60 minutes to answer, whereas the follow-ups took 25 minutes to answer approximately. Research assistants, all of whom were trained master-level psychologists, took care of explaining the study to the participants, oversaw the informed consent process, and led both the initial interview and the follow-ups. Finally, participants were given gift cards after the completion of each interview, 10.000 CLP (approximately US$12) after the first assessment, and 5.000 CLP for each telephone follow-up (approximately US$6). This study was approved by the Scientific Ethics Committee of Health Sciences of the Pontificia Universidad Católica de Chile (CEC-MedUC, protocol number 160826002), as well as the funding agency.

[Please, insert here Figure 1]

Questionnaires

Edinburgh Postnatal Depression Scale (EPDS)

To assess prenatal and postpartum depressive symptoms, we used the validated Chilean version of the Edinburgh Postnatal Depression Scale (Jadresic et al., 1995). This questionnaire asks participants to report the depressive symptoms experienced in the last seven days. Despite this scale being designed to measure PPD, there is evidence supporting its use during pregnancy as well (Vega-Dienstmaier et al., 1997). The questionnaire contains 10 items rated on a four-point scale, with scores ranging from 0 to 3, with a total potential score ranging from 0 to 30 points, where the higher the score indicates the greater the severity of depressive symptoms. The Chilean Health Ministry (Ministerio de Salud, 2014), as well as other Chilean researchers (Mendoza & Saldivia, 2015), recommend using the EPDS to detect depression during pregnancy and postpartum. For Chilean women, a cut-off score of 13 or more is recommended during pregnancy, whereas a cut-off score of 10 is recommended during postpartum (Ministerio de Salud, 2014). The internal consistency for the EPDS in our sample was estimated by Cronbach's α = .84.

Behavioral Activation for Depression Scale—Activation Subscale (BADS-A)

To assess BA, we used the subscale "A" of the validated Spanish version of the Behavioral Activation for Depression Scale (Barraca et al., 2011). Originally, the BADS is a 25-item scale consisting of four subscales that assess activation, avoidance/rumination, work/school impairment, and social impairment "during the last week including today" (Kanter et al., 2007). Subscale "A" contains seven items rated on a seven-point scale ranging from zero to six and a total score ranging from 0 to 42. Higher scores suggest higher levels of activation. The internal consistency for the BADS-A in our sample was estimated by Cronbach's α = .85.

Demographical and clinical variables.

Demographical and clinical variables were collected during the first interview. To determine the history of major depression, participants were "Have you ever suffered from major depression?", during the first interview.

Data Analysis

To accomplish our first aim, we estimated repeated measure ANOVAs to test longitudinal mean differences in BA and depressive symptoms scores throughout the prepartum and postpartum period. These analyses were conducted using multiple imputations with the mice package (v3.13.0; van Buuren & Groothuis-Oudshoorn, 2011) in R Version 4.3.1 (R Core Team, 2019) to handle missing data. For the second aim, we estimated a random-intercept cross-lagged panel model (RI-CLPM). RI-CLPM is a relatively new model that overcomes the limitations of simple cross-lagged panel models (Hamaker et al., 2015). It aims to analyze bidirectional relationships at a within-person level while accounting for time-invariant trait-like differences at the between-person level (Hamaker et al., 2015). The RI-CLPM model separates the variance of each measured construct into two parts. First, a between-subject component is estimated as the random intercept, with a grand mean representing the stable, individual, trait-like dimension, and a variance that allows to freely estimate the grand-mean variability between subjects. Second, a within-subject component, which represents the grand mean at each specific point and a variance that represents the individual variation around that mean. Thus, unlike traditional CLPM models, in RI-CLPM models autoregressive and cross-lagged coefficients represent within-subjects, not between-subject effects (Orth et al., 2021). Missing data was handled using full-information maximum likelihood, considering all available data points. These analyses were conducted in Mplus version 8.10 (Muthen & Muthen, 2017). In order, to interpret cross-lagged effect sizes (standardized *ß*) we used the benchmarks proposed by Orth et al. (2022) for this kind of model, that is, .03 (small effect), .07 (medium effect), and .12 (large effect).

Finally, we conducted several robustness analyses. Namely, we repeated the RI-CLPM analyses with and without participants who were in treatment for depression. Additionally, we run a series of multi-group RI-CLPM considering several relevant covariates: marital status, type of health insurance (a proxy for socioeconomic status), type of delivery (vaginal vs cesarean), primiparous versus multiparous participants, pregnancy or delivery complication or newborn health problems, and reporting a history of major depression.

Transparency and Openness Promotion-related statements.

Data, materials, and analysis codes are available upon request from Lydia Gómez-Perez. The present research was not preregistered. The authors report no conflict of interest.

Results

Descriptive Statistics

Descriptive statistics are presented in Table 1. Participants were on average 31.19 (*SD*=5.3) years old, with over half possessing university degrees. The participants in our study were fairly representative when compared to pregnant women from the metropolitan area of Santiago except for some differences in educational level and marital status, where our sample presented higher educational level and were married in a greater proportion (Instituto Nacional de Estadísticas de Chile, 2019). The percentage of women having their first child was 52%. A small percentage (1.66; *n* = 7) of the sample declared to have birthed twins at this time.

[Please, insert here Table 1]

Clinical characteristics measured longitudinally at the prepartum, one-month postpartum, three-month postpartum, and six-month postpartum phases are summarized in Table 2. Reported BA scores were relatively high, while depressive symptoms were moderately low throughout the prepartum and postpartum periods

[Please, insert here Table 2]

We conducted ANOVA or t-test to examine differences in BA and depressive symptoms as a function of marital status, occupational status, educational level, type of health insurance (a proxy of socioeconomic level), reporting a history of major depression, being under psychotherapy at enrollment, and being under pharmacological treatment at enrollment (see Tables S1 and S2). There were no differences in BA or depressive symptoms according to marital status, occupational status, or educational level in any of the assessments. Participants with public health insurance (indicative of lower socioeconomic status) presented lower depressive symptom scores at one month and at three months postpartum than participants with private health insurance. Participants who reported a history of major depression presented higher depressive symptoms scores across all waves than participants without a history of major depression. In addition, participants who reported a history of major depression presented lower BA scores at one, three and six months postpartum than participants without a history of major depression. Participants who were under psychotherapy at enrollment presented higher depressive symptoms during pregnancy and at one and three months postpartum than those who were not under psychotherapy. In addition, participants who were under psychotherapy at enrollment presented lower BA scores at one month postpartum than participants not receiving psychotherapy Participants who were under pharmacological treatment at enrollment presented higher depressive symptoms prepartum and at one and three months postpartum than participants who were not under pharmacological treatment. Neither the correlations between age and depressive symptoms, nor age and BA were statistically significant.

Changes in BA and depressive symptoms

The results of the repeated measures ANOVAs pooled over 100 imputed datasets showed that there were statistically significant differences in BA means between one-month postpartum and prepartum. All other time-point comparisons in BA were not significantly different (see Figure 2). Additionally, there were no significant differences in mean depressive symptoms scores between waves (p > .0.05) (see Figure 3).

[Please, insert Figures 2 and 3 near here]

Relationship between depressive symptoms and BA scores

To examine the directionality of the relationship between depressive symptoms and BA scores, the data were analyzed utilizing RI-CLPM. Nested models were tested, where the autoregressive parameters for both BA and PDD symptoms as well as the cross-lagged parameters were restricted to be equal across waves; because according to Orth et al. (2021), "*Researchers should use cross-wave equality constraints on structural coefficients (e.g., autoregressive and cross-lagged effects) unless there is a clear rationale against doing so*" (p. 1030). It is worth mentioning that, although time intervals between waves were not exactly the same (e.g., not equal space intervals), the differences were rather small (three months between wave one and wave two versus two months between wave two and wave three). Furthermore, in the end, the equality constraints imposed provide a strict test for the equivalence of the regression coefficients. The more restrictive and parsimonious model was chosen, as chi-square comparisons established that it was not different from the unrestricted model ($\Delta\chi^2$ = 15.209, Δdf = 10, p = .125). The model fit to the data was good; $\chi^2(19)$ =40.721, p < .01, CFI=0.972, TLI=0.959, RMSEA = 0.048. Cross-lagged and autoregressive parameter estimates are presented in Figure 4. All coefficients are presented in the supplemental material in Table S1. Cross-lagged parameters indicated an inverse bidirectional relationship, where within-person deviations from the expected level of BA scores predicted within-person deviations from the expected level of depressive symptoms, and within-person deviations from the expected level of depressive symptoms predicted within-person deviations from the expected level of BA scores. The magnitude of the standardized β was higher for depressive symptoms to BA (ranging from -.216 to -.243) than for BA to depressive symptoms (ranging from -.141 to -.198) (See Figure 4 and Table S3). Given this, we decided to run a second RI-CLPM where we restricted the model even further by fixing the cross-lagged coefficients to be equal. In this way, a significant decrease in the goodness of fit of the model would imply the parameters differ significantly. The chi-square comparison revealed a statistically significant difference ($\Delta\chi2$ = 34,656, Δdf = 11, p = .0038), indicating that the effect of intra-individual change of depression symptoms on BA was greater than vice versa. Nonetheless, it should be noted that this test assumes equal variances across waves and constructs, so it should be interpreted with caution.

Both, the autoregressive parameters for and depressive symptoms were statistically significant (See Figure 4 and Table S1). Standardized autoregressive coefficients ranged from .137 to .221 for BA and from .245 to 254 for depressive symptoms. These results indicate that after accounting for trait-level BA and depressive symptoms, both BA and depression showed significant within-person stability across waves.

It is worth mentioning that, in support of the type of analysis conducted, most of the item-level variance was within rather than between individuals. Specifically, for depressive symptoms, on average, 61% of the item-level variance (range 46 to 75%) was within individuals and 39% between individuals, whereas for BA 80% of the item-level variance (range 71 to 86%) was within individuals and 20% between individuals.

Lastly, there was a large inverse correlation r = -.522, p <.001, *95% CI* [-.750, -.293] between the random intercepts of BA and depressive symptoms. The random intercept factors provide information only about correlational associations between the constructs, similar to cross-sectional correlations, with the difference that the intercept factors are based on information from several waves (Orth et al., 2021).

Robustness analyses

We examined whether our results were robust when excluding from the RI-CLPM 46 women who were under treatment for depression at enrollment (N = 457). As in the model with the entire sample, we chose the more parsimonious model, in which we restricted the autoregressive parameters for both BA and PDD symptoms as well as the cross-lagged parameters to be equal across waves, as chi-square comparisons established that it was not different from the unrestricted model ($\Delta\chi2$ = 14.20, Δdf = 10, p =.164). The fit of this model was very good; $\chi 2(15)$ =38.434, p < .001, CFI=0.970, TLI=0.961, RMSEA = 0.047. The results were similar to those found in the model with the general sample, except that the cross-lagged parameters from BA to depressive symptoms and the autoregressive parameters for BA were not significant. Nonetheless, despite the lack of significance, the effect sizes of the cross-lagged parameters were large according to Orth et al. (2022). See Figure 5 and Table S4.

[Please, Insert Figures 4 and 5 near here]

We conducted again our main RI-CLPM including age as a covariable. As recommended by Mulder and Hamacker (2021), we compared models with age as a predictor of the observed variables versus the random intercepts. The comparison was not statistically significant ($\Delta\chi2$ =

1,82, *Δdf* = 2, *p* = 998), which indicated that the more parsimonious model in which age predicted the random intercepts was preferable. In this model, age was not significantly associated with any of the intercepts.

No differences were found in autoregressive and cross-lagged parameters on the separate multi-group analyses by marital status ($\Delta\chi 2$ = 3,42, Δ*df* = 7, *p* = .970), type of health insurance ($\Delta\chi 2$ = 11.42, *Δdf* = 7, *p* = .121), type of delivery ($\Delta\chi 2$ = 12.52, *Δdf* = 7, *p* = .08), primiparous versus multiparous participants ($\Delta\chi 2$ = 5.26, *Δdf* = 7, *p* = .628), and pregnancy or delivery complication or newborn health problem ($\Delta\chi 2$ = 10.87, *Δdf* = 7, *p* = .144), indicating the robustness of the model across these populations. Nevertheless, we found that there were differences in the model as a function of reporting a history of major depression ($\Delta\chi 2$ = 15,76; *Δdf* = 7, *p* = .03). Namely, the cross-lagged effects from BA to depressive symptoms were significant only for those individuals who reported a history of major depression (with standardized coefficient ranging from -.276 to -.312) but not for those without such a history. (See Figure 6 and Tables S5 and S6). Nonetheless, despite the lack of significance in these cross-lagged effects for the model of women without a history of major depression**,** the magnitude of the effect sizes for this model ranged from moderate (*ß* = -.099) to large 148 according to Orth et al. (2022)'s recommendations for cross-lagged effect benchmarks. The magnitude of the cross-lagged effect from depressive symptoms to BA was larger for those individuals reporting a history of major depression than for those without such a history (ranging from -.304 to -.317 and from -.166 to .195, respectively for each group). Autoregressive coefficients were significant only for those individuals without a history of major depression, but not for those reporting a history of major depression. Despite this, in the case of the autoregressive effects for depressive symptoms, the effect sizes were similar in both groups. Specifically, autoregressive standardized coefficients for depressive symptoms ranged from .204 to .237 for the group with a history of major depression and from .203 to .223 for the group without a history of major depression. Auto-regressive standardized coefficients for BA ranged from .113 to .141 for the group with a history of major depression and from .137 to .251 for the group without a history of major depression.

Discussion

The current study aimed to describe changes in BA levels and depressive symptoms throughout the prepartum and postpartum period, and to examine the bidirectional relationship between BA levels and PDD symptoms. For our first aim, we expected a significant decrease in BA levels and an increase in depressive symptoms between the prepartum and postpartum period, as all our participants were subjected to a life stressor. Results are partially in line with our hypothesis because, as we expected, BA scores dropped at one month postpartum. As for depressive symptoms, no significant change was observed between waves. These observations may be attributed to the fact that our sample was overall non-clinical. As for our second aim, we expected to observe an inverse bidirectional relationship between intraindividual changes in BA scores and intraindividual changes in depressive symptoms, indicating that when women have lower than expected individual-level of BA, they experience a subsequent increase in depressive symptoms; and when women present higher than expected individual-level of depressive symptoms they experience a subsequent decrease in BA. Our findings are in support of our hypothesis, as BA and depressive symptoms showed significant cross-lagged associations. The effect sizes of these associations were large according to Orth et al.´s benchmarks (2022), with standardized *ßs* ranging from -.141 to -.198 in the case of the association between intraindividual changes in BA and subsequent intraindividual changes in depressive symptoms; and ranging from -.216 to -.249 in the case of the association the other way around. These findings are in line with Lewisohn's model of depression which states that both emotional and behavioral factors (such as depressive symptoms and the reduction in BA, respectively) interact across time and can lead to depressed states that trap people in a vicious circle of unhealthy behaviors and mood (Dimidjian et al., 2011). Our findings are also in line with results obtained by Wagener et al., (2016) who found an inverse relationship between these two variables in a sample of adults. Nonetheless, our results contrast with Shudos et al.'s (2017), as they found that BA did not predict depressive symptoms eight weeks after in a sample of undergraduate students. Some design and methodological strengths in our study may account for these discrepancies in the results. Firstly, unlike Shudo et al., (2017) we did not assess avoidance. Future longitudinal studies must examine the relationship between BA and depressive symptoms adjusting for this variable. Secondly, unlike in Shudo et al.'s, all our participants were exposed to a vital stressor. Thirdly, we used RI-CLPM, a type of analysis that allows us to assess whether BA predicts intraindividual changes in depressive symptoms, and vice versa while adjusting for trait-like stable invariant levels of both constructs. Finally, our sample size was much larger, contributing to more power to test the expected effects.

We found that an asymmetrical model in which the cross-lagged coefficients were higher for depressive symptoms to BA than for BA to depressive symptoms had a significantly better fit than a model in which these coefficients were equal. This suggests that the relationship between intraindividual changes in depression and future intraindividual changes in BA is stronger than the opposite relationship. It may be easier or more commonplace to experience a decrease in BA when depressed, but harder to develop depressive symptoms when circumstantial life events alter BA levels. Resuming activities after a vital stressor may be harder for women who already have some depressive symptomatology than for those who are free from these symptoms. Despite the statistical significance of this result, the magnitude of the difference in the beta coefficient *(Δß)* was modest, ranging from .045 to .075. Furthermore, this result must be taken with caution as the chi-square comparison requires an equal variance across time and constructs. Finally, the trait-like relationship between both constructs was large with 27% of shared variance between these constructs, indicating that people who tend to have low levels of BA tend to present depressive symptoms. In line with previous literature, the results of the present study support the use of BA to improve current (Shudo et al., 2017; Wagener et al., 2017) and future symptoms of depression (Cuijpers et al 2007; Dimidjian et al., 2017; Ekers et al., 2014; Nasrin et al., 2017; Santo et al., 2019).

The findings regarding the bidirectional association between intraindividual changes in BA and intraindividual changes in depressive symptoms showed robustness when multi-group RI-CLPM were conducted to adjust for several covariates (i.e., marital status, the type of health insurance, type of delivery, primiparous versus multiparous participants, and pregnancy or delivery complication or newborn health problem). Nonetheless, reporting a previous history of major depression moderated the influence of intraindividual changes in BA and intraindividual changes in depressive symptoms. Specifically, this relationship was significant among women with a history of major depression, but not among women without a history of major depression. The large differences in the magnitude of the effect sizes suggest that intra-individual decreases in BA were more likely to lead to intra-individual increases in depressive symptoms in people with a history of depression than in people without such a history. Unlike women with a history of major depression, women without a history of depression may have protective factors that safeguard them from depressive symptoms even when their BA decreases. Conversely, women with a history of major depression may present other vulnerability factors that amplify the detrimental effect of their decreases in BA. Nonetheless, these results must be taken with caution, because reports of lifetime major depression usually have low reliability (Stuart et al., 2014; Wells & Horwood, 2004). Similarly, when participants who were under treatment for depression during pregnancy were removed, the prospective effect of temporary deviations from the trait level in BA on changes in the temporal deviations from the trait level in depressive symptoms was no longer statistically significant (p = .055). Differences in the magnitude of the effect sizes (*Δß)* between the model with and without these participants were relatively small, ranging from .034 to .059. It is worth mentioning that although the pathways from BA to depressive symptoms were not significant neither in the group of participants without a history of major depression nor when participants under treatment for depression at enrollment were removed, the effect sizes ranged from moderate to large. Future longitudinal studies regarding the relationship between intraindividual changes in BA and depressive symptoms among participants without a history of depression and other mental health problems, in which these variables are assessed in a more reliable way (e.g., using mental health clinical records), are needed.

We found that previous deviations of depressive symptoms from expected scores systematically predicted future within-women deviation from expected depression scores. This coincides with the abundant existing literature on the important predictive power of prior depression over future depressive symptoms (Tram & Cole, 2006), which has also been observed to be true in the postpartum context (Guintivano et al., 2018). These results were maintained in all of our robustness analyses, except for the multigroup RI-CLPM controlling for previous history of major depression. Nonetheless, though the auto-regressive effect for depressive symptoms was significant only for those individuals with a history of major depression (but not for those without it), the effect size of this relationship was similar in both groups. Our study is the first one to show that previous deviations of BA from expected scores systematically predict future within-women deviations from expected BA scores. This result was also maintained in all of our robustness analyses, except for the model conducted without participants under treatment for depression and the group of participants who had a history of major depression. A higher instability in their depressive symptoms and BA scores could explain why the auto-regressive effects among women with a history of major depression were not significant. However, this is just a speculation, and future research is needed.

Some limitations of the present study include the fact that avoidance and response-contingent positive reinforcements were not considered even though these constructs are heavily targeted by Behavioral Activation Therapy. Control variables such as social support, disrupted sleep, and parental stress were also not contemplated, and the absence of these relevant covariates is reflected in the relatively large within-wave error correlations, which suggest that the association between BA and PPD is influenced by other non-measured variables. It is possible that reductions in BA that occur after an event, such as having a baby, lead to depression to a higher degree in women who are experiencing other risk factors (i.e., not desire pregnancy, other simultaneous stressors such as couple problems, previous mental health problems) or who present higher levels of other variables (i. e., parenting stress, lack of social support). In other words, there are factors other than BA that may be explaining intraindividual changes in depressive symptoms, and vice versa. In fact, other variables such as avoidance, response-contingent positive reinforcements (Santos et al., 2021), and reward network modulation (Nagy et al., 2020) have been proposed to mediate the relationship between these two variables. In this line, we found that intraindividual changes in BA had a stronger association with intra-individual changes in depressive symptoms in individuals with a previous history of major depression. A better understanding of the mechanisms that might be playing a role in the development, maintenance, and treatment of PPD is key to continuing to improve both preventive and therapeutic alternatives to PPD symptoms. Future studies should consider accounting for avoidance, response-contingent positive reinforcements, as well as common psychosocial factors that affect PPD symptomatology; and examine more complex models including potential mediators and moderators of the relationship between changes in BA and depressive symptoms after facing a vital stressor. Another limitation was that the sample was not entirely representative of pregnant women in Santiago, therefore it would be important to replicate this study with a more representative sample or a contrasting population such as pregnant women with lower income and educational level. Future studies should also examine different trajectories of BA and depressive symptoms, as not all women may present the same trajectory of change in these variables after delivery. Additionally, parenthood is undoubtedly one of the most meaningful and significant events in life, representing a new challenge to which parents have to adjust and providing a source of stress (i.e., having to adjust to a new lifestyle, new routines, caring for another, changes in identity, the decline in partner relationship quality and satisfaction, etc.) (Corner et al., 2023; Song-Choi & Woodin, 2022). Furthermore, giving birth is experienced by between 14% and 35% of women as traumatic (Türkmenet al., 2021). Despite this, we understand that testing these effects in other samples, with higher levels of stress, might lead to different results. Studies examining the relationship between BA and depressive symptoms in a sample exposed to traumatic events (e.g., natural disasters or car accidents) need to be conducted. Finally, although we consider that the changes in activities that occur in the transitions to motherhood may represent a reduction in BA because it is likely that most women have to abandon valued activities to focus on the newborn; for other women, the new activities that they must perform to take care of the newborn may represent an increase in BA, because they can be highly tied to their values. The value given to maternity, the sense of competency in the care of the newborn, and the value given to the activities that women have to give up (at least temporarily) may play a role in BA. Further studies need to be performed to address the role of these factors.

Alternatively, one of the strengths of the present study is that it is the first study to explore the bidirectional relationship between activation levels and depressive symptoms longitudinally. While other studies have considered a relationship between BA and depressive symptoms, none of the previous studies had assessed the bidirectional relationship between them. Furthermore, it is the first study to examine the association between BA and depressive symptoms after a vital stressor (i.e., giving birth) and outside the context of therapy. It is also the first study investigating this relationship in the perinatal context, a relevant population to address given the consequences of PPD on both women and their offspring. Additionally, in a more practical sense, this study further supports the early screening protocol for PPD, already available in our healthcare system (Ministerio de Salud, 2014). It also supports modifications of BA as a mechanism of change for PPD, an idea also supported by the effectiveness of BAT in this context (Dimidjian et al., 2017). The study also contributes additional evidence for the theoretical behavioral model of depression proposed by Lewinsohn. Furthermore, our results suggest that a previous history of major depression may be an important indicator of risk for the development of postpartum depression, with the potential for informing preventive intervention efforts among pregnant women. Finally, several limitations of previous studies were overcome by including a stressor, adding longitudinal waves, and selecting a more current and robust statistical analysis. Overall, the findings of this study contribute to the growing literature surrounding behavioral models of depression, providing support for a bidirectional relationship between depressive symptoms and BA in the peripartum period.

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Table 1

Demographic and Clinical Characteristics of the Sample.

Characteristic	*N* = 503
Age, years, mean (SD)	31.19 (5.3)
Marital Status, *n* (%)	
Single	128 (25.45%)
Cohabitation with partner	191 (37.97%)
Married	175 (34.79%)
Divorced	5 (0.99%)
Missing data	4 (0.80%)
Occupational Status, *n* (%)	
Student	37 (7.36%)
Full-time employee	54 (10.74%)
Part-time employee	23 (4.57%)
Unemployed	29 (5.77%)
Maternity leave	287 (57.06%)
Sabbatical	6 (1.19%)
Housewife	49 (9.74%)
Other	17 (3.38%)
Missing data	1 (0.20%)
Education Level, *n* (%)	
< High school	8 (1.59%)
High school	54 (10.74%)
Technical degree	115 (22.86%)
University	267 (53.08%)
Postgraduate	57 (11.33%)
Missing data	2 (0.40%)
Type of Health Insurance, *n* (%)	
Public	276 (54.87%)
Private	223 (44.33%)
Missing data	4 (0.80%)
History of major depression, *n* (%)	134 (26.64%)
Treatment for emotional or psychological problem at enrollment, *n* (%)	
Psychotherapy	34 (6.76%)
Pharmacological	19 (3.78%)

Table 2

Mean and standard deviation of Behavioral Activation and Depressive Symptoms based on 100 imputed datasets N=503.

Measure	Prepartum (N = 503)	One month postpartum (N = 419)	Three months postpartum (N = 437)	Six months postpartum (N = 418)
BADS-A, mean *(SD)*	34.64 (6.39)	30.56 (8.71)	33.52 (8.33)	32.77 (9.04)
EPDS, mean *(SD)*	5.70 (4.49)	5.68 (4.62)	5.53 (4.98)	5.77 (5.33)
Depression, *n (%)*	42 (8.33%)	85(20.29%)	86(19.68%)	79(18.90%)

Note: Percentages of depression are estimated based on observed data (*n* in parenthesis) and according to the clinical cutoff score on the Edinburgh Postnatal Depression Scale (EPDS).

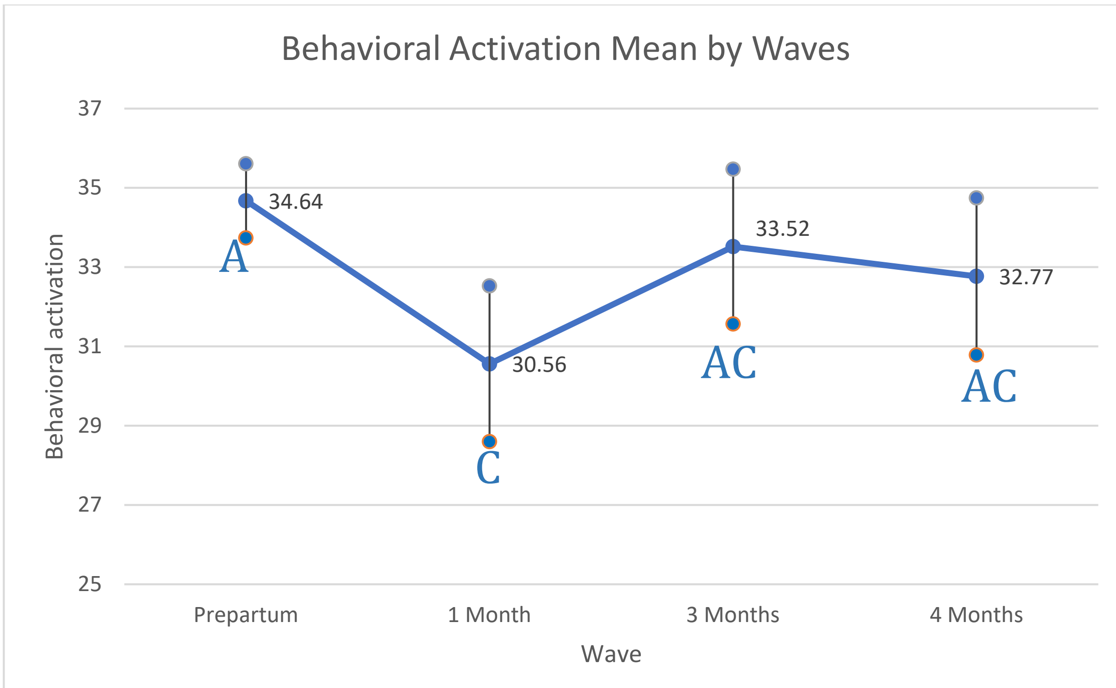
Figure 1

Temporal Flowchart of the Study's Design Procedure

Time 1		Time 2	Time 3	Time 4
N = 503		*N* = 419	*N* = 437	*N* = 418
Prepartum at 32 to 36 weeks of pregnancy	CHILDBIRTH	one-month postpartum	three-months postpartum	six-months postpartum
Face-to-face		Phone call	Phone call	Phone call
40 to 60 min assessment		25 min assessment	25 min assessment	25 min assessment

Figure 2

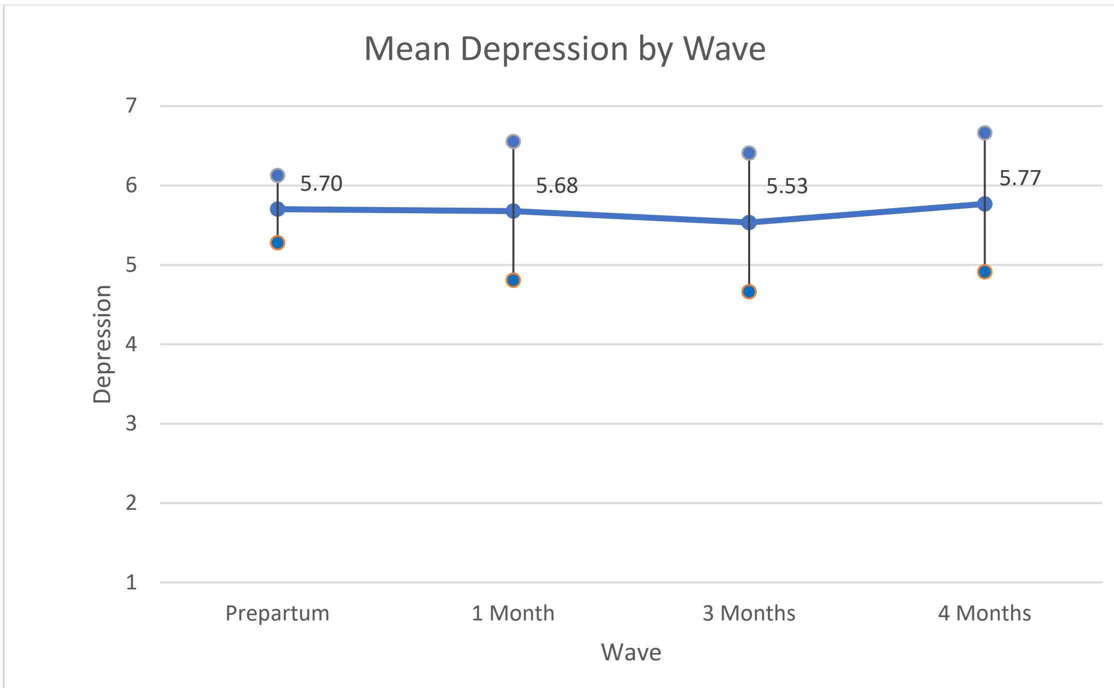
Line graph with 95 confidence intervals of behavioral activation scores between prepartum, one-month postpartum, three-months postpartum, and six-months postpartum



Note. Estimates are based on 100 imputed data sets, n=503. Matching letters indicate no significant differences between those periods were found, *p <.01*. Bars represent 95% confidence intervals around the means. The original scale ranges from 0 to 42 points. The ranges of BA scores in this sample were 11-42 in the prepartum and 0-42 in one-month, three-month, and six-month postpartum.

Figure 3

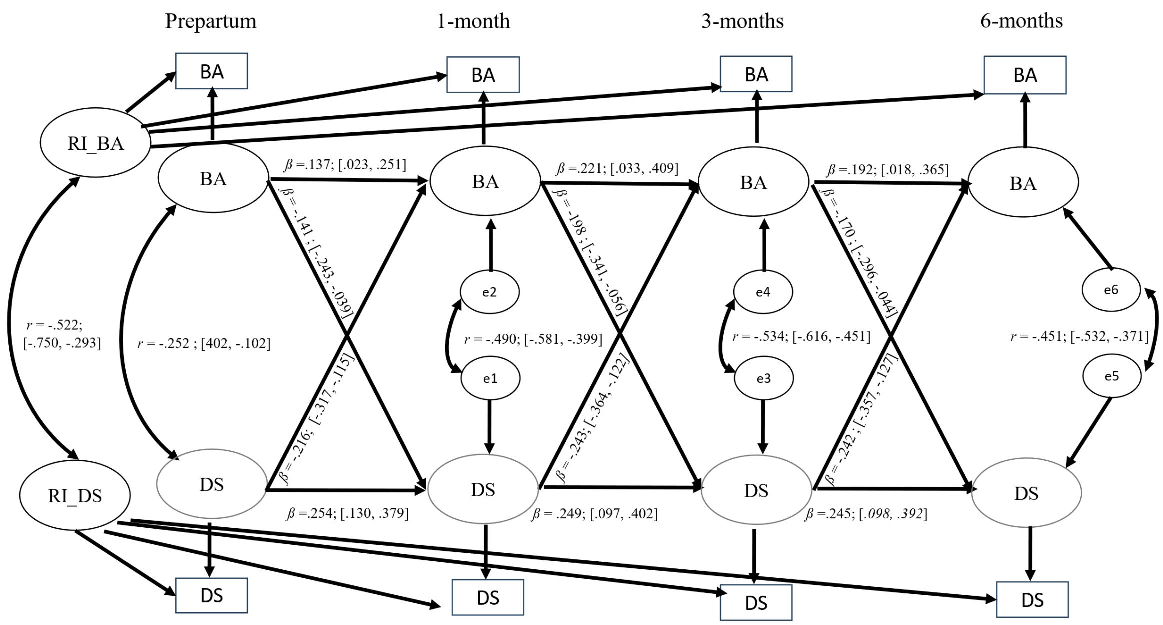
Line graph with 95 confidence intervals of mean depressive symptoms scores between prepartum, 1-month postpartum, 3-months postpartum, and 6-months postpartum



Note. Estimates are based on 100 imputed data sets, *N*=503. No significant mean differences at $p < .01$. Bars represent 95% confidence intervals around the means. The original scale ranges from 0 to 30 points. The international cutoff score is 12 points. The national cutoff scores are 13 or more points on the EPDS scale at the prepartum and ten or more points at the postpartum. The ranges of depressive symptoms scores in this sample were 0-27 in the prepartum and at one month postpartum, and 0-26 at three-months and six months postpartum.

Figure 4

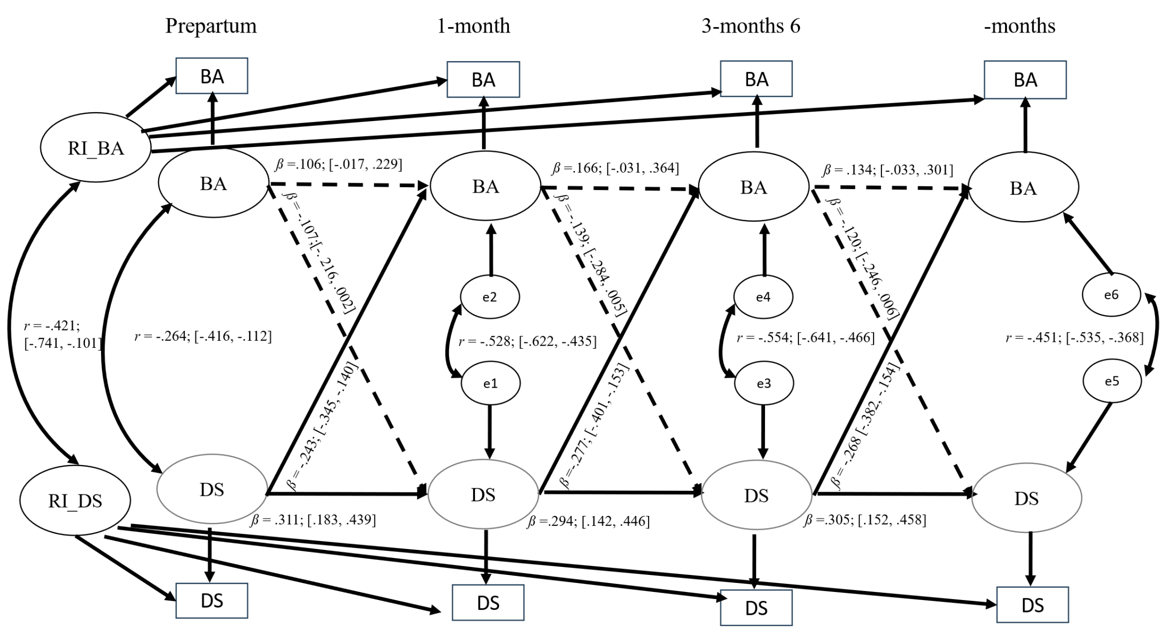
Random Intercept Cross-Lagged Panel Model of Behavioral Activation (BA) and Depressive Symptoms (DS) calculated with the entire sample (N = 503).



Note. Standardized coefficients are presented. All coefficients shown are significant $p < .05$. 95% CI in brackets. The estimated correlation between residuals of variables at the within-person level is represented as the relationship between e1 with e2, e3 with e4, and e5 with e6.

Figure 5

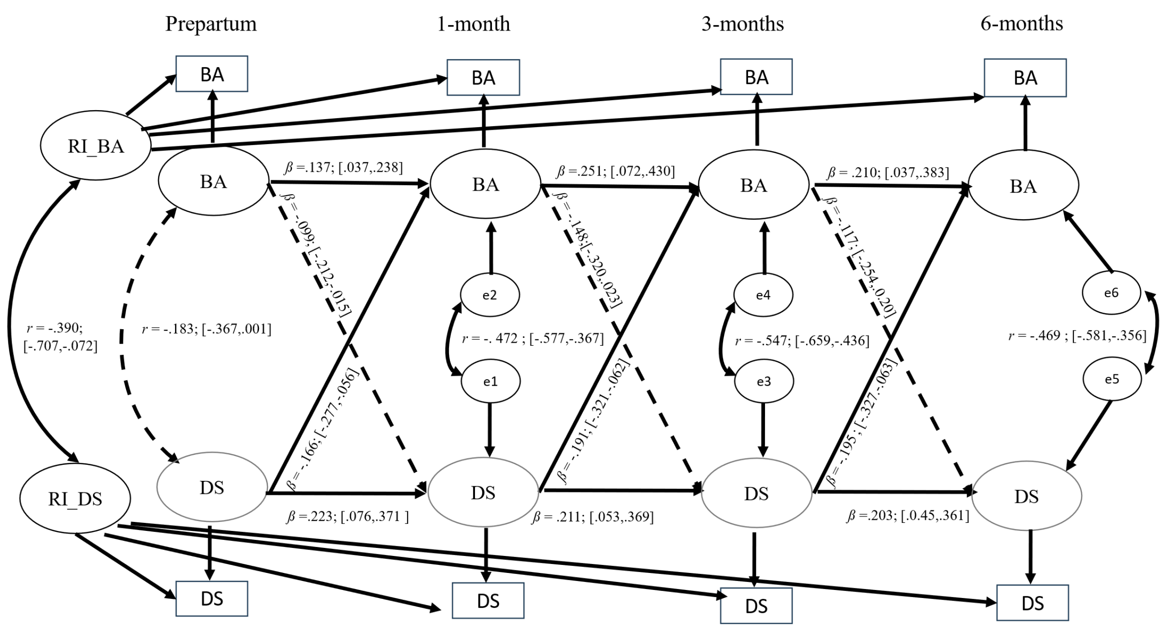
Random Intercept Cross-Lagged Panel Model of Behavioral Activation (BA) and Depressive Symptoms (DS) calculated excluding 46 women who were under treatment for depression at enrollment (N = 457).



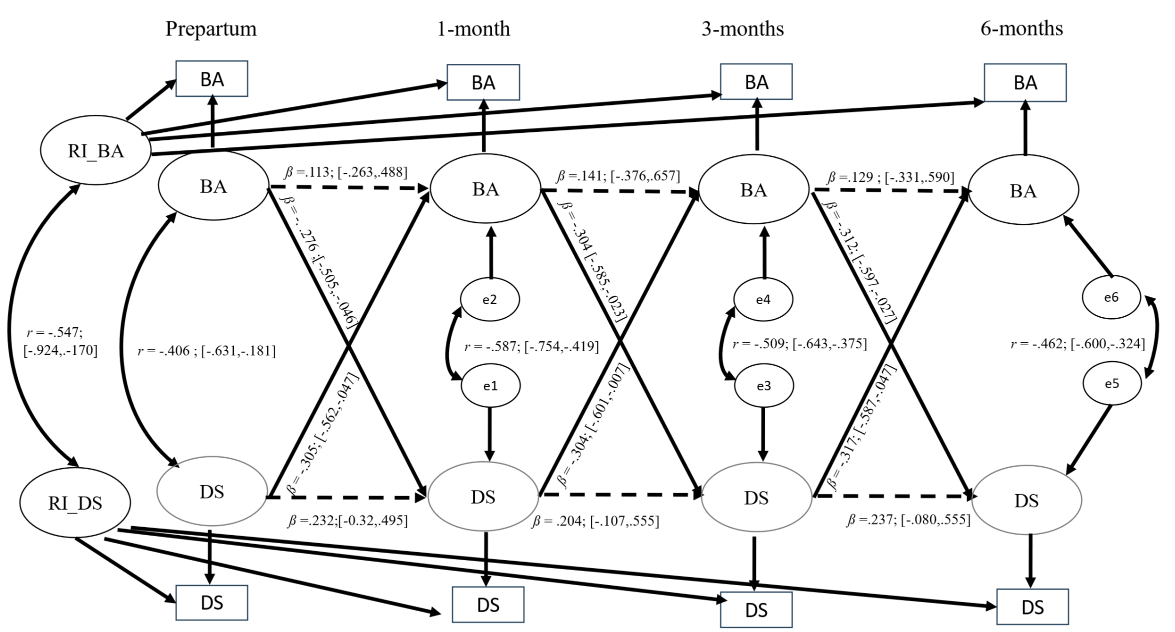
Note. Standardized coefficients are presented. Dashed lines indicate non-significant coefficients. All coefficients shown are significant *p* < .05. 95% CI in brackets. The estimated correlation between residuals of variables at the within-person level is represented as the relationship between e1 with e2, e3 with e4, and e5 with e6.

Figure 6. Multigroup *Random Intercept Cross-Lagged Panel Model of Behavioral Activation (BA) and Depressive Symptoms (DS)* considering as a covariate reporting a history of major depression.

A.



B.



Note. A. Model for women without a history of major depression. B. Model for women with a history of major depression. Standardized coefficients are presented. Dashed lines indicate non-significant coefficients. All coefficients shown are significant p < .05. 95% CI in brackets. The estimated correlation between residuals of variables at the within-person level is represented as the relationship between e1 with e2, e3 with e4, and e5 with e6.