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A longitudinal model of rejection sensitivity and internalizing symptoms: Testing emotion regulation deficits as a mechanism and outcome of symptoms

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Abstract

Objective: Individuals who experience heightened rejection sensitivity (RS) are at greater risk of increased internalizing symptoms over time. This is especially so for adolescents and young adults, as this is a time of many social transitions and an average increase in such symptoms. Yet, little longitudinal research has explored specific mechanisms that may help explain how RS lends itself to increased symptomology during adolescence and young adulthood. In this study, we tested the summative effect of emotion dysregulation, expressive suppression, and social avoidance (i.e., *ER-deficits*) as mechanisms. Moreover, we estimated bidirectional temporal associations between ER-deficits and symptoms.

Method: Participants included 402 adolescents and young adults aged 17 to 27 years ($M = 19.9$ years, 66% female) who completed two assessments over a 1-year period.

Results: In a path model, participants who reported more RS increased in anxious symptoms, and RS was indirectly associated with increased anxious and depressive symptoms via the three ER-deficits. Additionally, cross-lagged panel analyses showed that dysregulation and suppression predicted increased symptoms over time, while anxious symptoms predicted increased social avoidance over time.

Conclusion: These findings expand understanding of the role of RS in young people's increasing internalizing symptoms, implicating ER-deficits in these processes.

KEYWORDS

anxiety, depression, emotion regulation, rejection sensitivity, social avoidance

1 | INTRODUCTION

Rejection sensitivity (RS) is a cognitive-affective bias where one readily perceives, anxiously expects, and negatively overreacts to both ambiguous and overt cues of rejection (Downey & Feldman, 1996). It was originally conceptualized as a personality disposition that would explain why some individuals appear to be more likely to perceive rejection by others and to experience greater emotional and interpersonal difficulties when rejection is perceived. In developing RS theory to simultaneously account for the negative perceptions of others and the self, as well as the negatively biased expectations and detrimental reactions that follow from RS, Downey and colleagues (Downey & Feldman, 1996; Levy, Ayduk, & Downey, 2001) developed a comprehensive RS model. In this model, RS is proposed as an outcome of interpersonal rejection experiences, and, once developed, RS is expected to impel increasingly negative emotional and behavioral reactions to events that can induce further rejection. Experimental and correlational research have empirically supported these negative effects from RS

during childhood and adulthood. Individuals higher in RS report more maladaptive expectations of, and reactions to, interpersonal rejection (and other similar stressors such as victimization) (Marston, Hare, & Allen, 2010; Pearson, Watkins, & Mullan, 2011; Zimmer-Gembeck & Nesdale, 2013; Zimmer-Gembeck, Trevaskis, Nesdale, & Downey, 2014). Furthermore, in studies expanding on this theme, RS models have sought to identify how maladaptive expectations and reactions related to RS predict internalizing symptoms, such as depression and anxiety (Gardner & Zimmer-Gembeck, 2018; Peters, Smart, & Baer, 2015; Watson & Nesdale, 2012).

An important aspect of the RS model is the elucidation of transactional associations, such that individuals high in RS experience negative emotional and behavioral reactions in what has been termed a self-fulfilling prophecy (Levy et al., 2001; Zimmer-Gembeck, 2016). That is, as a result of heightened negative emotional and behavioral reactions among individuals high in RS, rejection by others appears more likely to occur into the future, which in turn, then perpetuates RS. Given that these emotional and behavioral reactions are key mechanisms that appear to heighten the risk of psychopathology and future rejection experiences (Levy et al., 2001; Zimmer-Gembeck, 2016), research is needed to unpack which reactions may mediate associations between RS and internalizing symptoms over time.

Beyond exploring mechanisms of effect between RS and internalizing symptoms, related questions emerge in relation to other points within this process. Specifically, there is a need to consider and explicitly examine potential bidirectional effects. For example, increased symptoms may be an outgrowth of the negative emotional and behavioral reactions that are elevated when RS is present, but increased symptoms may also predict an escalation in these emotional and behavioral responses over time. Consequently, bringing to bear a more expansive view of RS effects on internalizing symptoms also intimates a need to explore reciprocal links between emotional experiences and symptoms. With these two main aims in mind (i.e., mediational processes and bidirectional effects), we focus here on late adolescents and young adults as a developmental period that is highly relevant to understanding RS. Adolescents and young adults are forming new social relationships, and deepening intimacy in preexisting friendships and romantic partnerships (Marston et al., 2010; O'Rourke, Halpern, & Vaysman, 2018; Zimmer-Gembeck, 2002). Each of these experiences carry with them the potential for rejection. In addition, emerging evidence indicates that young adults may show increases in symptoms of anxiety and depression, beyond what is reported when they were adolescents (e.g., Hankin et al., 2015). As such, RS and symptom development during this period are especially salient.

1.1 | A longitudinal RS model: Emotion regulation deficits as mediators

Identifying negative emotional and behavioral reactions that stem from heightened RS, and flow-on to internalizing symptoms, is essential for informing prevention and intervention efforts that aim to specifically reduce the factors that may heighten young people's risk for psychopathology. Fortunately, the cognitive and emotional responses identified as relevant for explaining psychopathology in the RS model have been unpacked in some past research. For example, studies testing the RS model in adolescents and young adults support poor coping and emotion regulation (ER)-deficits, considered here to be negative emotional and behavioral reactions or strategies that increase risk for psychopathology, that appear elevated in individuals who report more RS. In cross-sectional and longitudinal studies, individuals who report higher RS have been found to report greater rumination about negative events (Pearson et al., 2011), show greater social avoidance (Watson & Nesdale, 2012), more emotional dysregulation and suppression (Gardner & Zimmer-Gembeck, 2018), and more self- and other-blame for rejection (Zimmer-Gembeck, Nesdale, Webb, Khatibi, & Downey, 2016). Furthermore, several of these vulnerabilities contribute to the direct links between RS and internalizing symptoms in late adolescence and young adulthood. For example, findings from two cross-sectional studies (Gardner & Zimmer-Gembeck, 2018; Peters et al., 2015) have supported the notion that young adults' greater emotionality, measured as emotional dysregulation, is a vulnerability helping to account for why heightened RS is related to elevated internalizing symptoms. In the first study, RS had an indirect impact on affective instability (a component of Borderline Personality Disorder) via multiple measures of ER-deficits, when deficits were measured as emotion dysregulation, impulse control difficulties, anger rumination, and difficulties engaging in goal-oriented behavior (Peters et al., 2015). In the second cross-sectional study, RS had an indirect association with psychopathology symptoms via emotion dysregulation, suppression, and social avoidance (Gardner & Zimmer-Gembeck, 2018). While these findings implicate maladaptive emotional and behavioral reactions as important mechanisms, they are limited by their cross-sectional design. Thus, there is a need to better identify the roles of these ER-deficits in RS processes over time.

Despite some attention to coping and ER in the RS model, some responses that have been emerging as quite relevant for understanding interpersonal problems have been overlooked in longitudinal research. For instance in cross-sectional and longitudinal studies with young adolescents (about age 10 to 14 years), heightened sadness and anger and greater social withdrawal in response to scenarios that imply peer rejection (along with other maladaptive responses such as rumination

and self-blame) have been articulated as key mediators that help to better explain the reciprocal associations between RS (or rejection experiences), emotional and behavioral coping, and symptoms (Zimmer-Gembeck, 2015; Zimmer-Gembeck et al., 2016; Zimmer-Gembeck & Skinner, 2015). As a result, it may be the case that high intensity negative emotions and difficulties downregulating negative emotions, often described as emotion dysregulation (e.g., Perry-Parrish & Zeman, 2011; Peters et al., 2015), is uniquely related to increased symptoms.

Although high intensity negative emotion and difficulties downregulating emotion is likely implicated in the association between RS and symptoms, there may be two other ER-deficits that can play a role. First, evidence suggests that attempts to suppress or minimize emotional experience may prolong the feeling of distress when rejection experiences (or other relational schemas) are perceived or activated (Gardner & Zimmer-Gembeck, 2018; Levy et al., 2001). Thus, emotional suppression may also be an ER-deficit accounting for when RS leads to increased internalizing symptoms. Second, in both cross-sectional (e.g., Watson & Nesdale, 2012; Zimmer-Gembeck & Skinner, 2015) and longitudinal (e.g., Zimmer-Gembeck, 2015; Zimmer-Gembeck et al., 2016) studies of adolescents and young adults, social avoidance (i.e., a disengagement coping strategy), which is a more common response to managing emotion following perceived rejection among individuals higher in RS, appears to be uniquely relevant for prolonged internalizing symptoms over time. When the evidence across these studies is considered, it suggests that emotion dysregulation, emotional suppression and social avoidance in response to perceived rejection may act as three mediators linking RS to increasing internalizing symptoms over time. Yet, few longitudinal studies have explicitly tested the summative effect of all three as potential mediators within a single model with the aim of accounting for the effect of RS on increasing internalizing symptoms over time among older adolescents and young adults. Thus, we test a more explicit longitudinal model, which aligns with, and builds upon, original RS research. Here, our longitudinal model proposes that RS prospectively predicts increased internalizing symptoms over time via ER-deficits of dysregulation, suppression, and social avoidance, which were considered as three separate mediators within a single path model (see Figure 1).

1.2 | Bidirectional associations: Emotion regulation (ER)-deficits and internalizing symptoms

In exploring a mediating process in which RS exerts flow-on effects to internalising symptoms via ER-deficits, critical additional questions come to light. That is, within this RS-internalizing dynamic, bidirectional effects are possible, and indeed likely, as suggested by previous research (Compas et al., 2017; De France, Lennarz, Kindt, & Hollenstein, 2019; Masters, Zimmer-Gembeck, & Farrell, 2019). Specifically, ER-deficits and increased internalizing symptoms likely reciprocally influence each other over time, and so a full exploration of a longitudinal RS model includes testing these reciprocal effects. This examination is also relevant as constantly changing affective and interpersonal experiences during adolescence and young adulthood can give rise to heightened risk for emotional symptoms (De France et al., 2019; Larsen et al., 2013; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011; Perry-Parrish & Zeman, 2011). These symptoms are typically shown as outgrowths of deficits in ER, defined as the processes involved in monitoring, evaluating, and modulating one's emotional

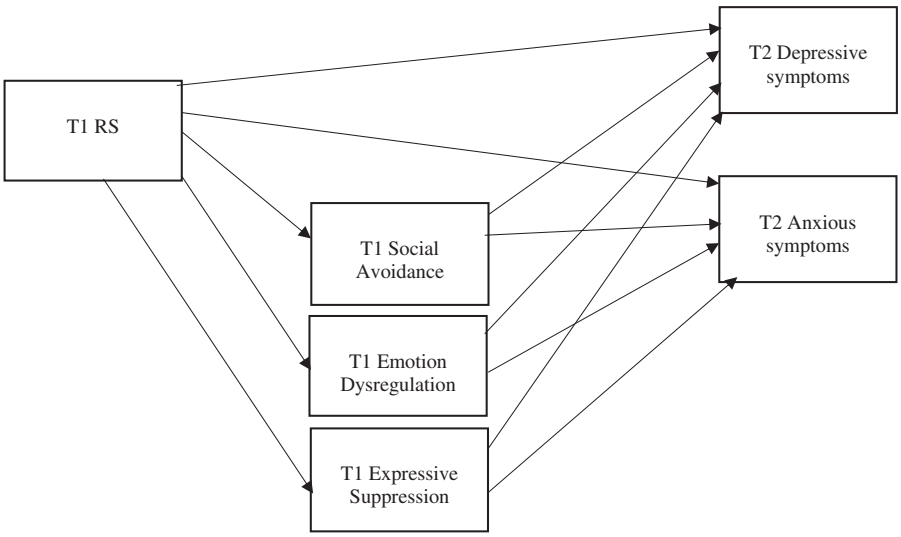

FIGURE 1 Hypothesized longitudinal model of RS and internalizing symptoms via emotion regulation deficits. T1 = Time 1; T2 = Time 2. For results of testing this path model, see Table 3

reactions, including their temporal and intensive features, to accomplish one's goal (Thompson, 1994). At the same time, complex neurological systems responsible for organizing and coordinating the emotional and cognitive processes involved in ER do not fully develop until the late 20s (Compas et al., 2017; Nolen-Hoeksema & Aldao, 2011; Zimmer-Gembeck & Skinner, 2016). Yet, these systems are relied upon to assist youth to respond adaptively to internal and environmental changes as they move into new social settings and become more autonomous and self-reliant (O'Rourke et al., 2018; Zimmer-Gembeck & Skinner, 2016). Thus, ER-deficits are likely mechanisms that act as risk for increased symptomology during this period.

Beyond a role of ER-deficits in exacerbating risk for internalizing symptoms, ER envelopes an ongoing sequence of reacting to events, so that internalizing symptoms could potentially impact subsequent ER. However, much of the literature focused on child, adolescent and young adult ER and adjustment specifies this relationship as unidirectional (i.e., an ER-deficit direction of effects), indicating deficits in the habitual use of adaptive ER strategies, and overuse of maladaptive strategies, as risk factors for emotional maladjustment (Compas et al., 2017; Nolen-Hoeksema & Aldao, 2011; Perry-Parrish & Zeman, 2011; Thompson, 1994). That said, more recent longitudinal models underscore that the links between ER strategies and emotional maladjustment are likely to be bidirectional (De France et al., 2019; Larsen et al., 2013; McLaughlin et al., 2011), though a clear direction of effect remains unclear. For example, among one study assessing early adolescents over the course of 1 year (Larsen et al., 2013), and another over 2 years (De France et al., 2019), both found that depressive symptoms significantly predicted increases in expressive suppression over time, but not vice versa. In another study examining early to mid-adolescents, emotion dysregulation predicted increases in symptoms of anxiety, aggression, and eating pathology over the course of 7 months, with no support found for converse associations (McLaughlin et al., 2011). Finally, in a later adolescent to young adulthood sample, assessed over 3 years, symptoms of depression and anxiety at T1 did not predict self-regulation at T2, whereas self-regulation at T2 predicted increases in depression and anxiety at T3 (Coyne, Stockdale, & Summers, 2019). This suggests that at least as individuals mature, ER-deficits contribute to internalizing symptoms, but potentially not vice versa.

1.3 | The current study

In summary, heightened RS appears to serve as a critical risk factor for increased symptoms over time (Gao, Assink, Cipriani, & Lin, 2017; Marston et al., 2010; Zimmer-Gembeck et al., 2016). Building on additional research of RS, this link between RS and internalizing symptoms likely occurs through an unfolding process. Here, heightened-RS leads to maladaptive ER in response to stressors, including events which bring the possibility of rejection, which then flows on to increased symptoms. In particular, we measured these ER-deficits as emotional dysregulation, suppression, and social avoidance. Therefore, we first test the prospective associations of late adolescents' and young adults' RS with symptoms across 1 year, exploring how these three responses might mediate these links. Here, we tested symptoms of depression and anxiety as separate measured variables, guided by past research in young adult populations that have done so and found significant but slightly differing effects between the two outcomes (e.g., Gao et al., 2017; Marston et al., 2010). More specifically, one longitudinal study found RS to be more consistently associated with anxiety symptoms across three consecutive years of follow-up assessment (with $\beta's$ ranging between .10 and .22) than depressive symptoms (with $\beta's$ ranging between .10 and .16) (Marston et al., 2010), while a meta-analytic study found that after adjusting for the possibility of bias, there were slightly different effect sizes for the longitudinal associations of RS with anxiety ($r = .30$) than with depression ($r = .21$). We hypothesized then that RS would be positively linked to emotion dysregulation, expressive suppression, social avoidance, and depressive and anxious symptoms both concurrently and longitudinally (Hypothesis 1). We also hypothesized that RS would be a risk factor for increased symptoms over time, and this risk would be transmitted via emotion dysregulation, expressive suppression, and social avoidance within a single path model (Hypothesis 2; see Figure 1).

Building on this longitudinal RS model, our third aim was to more specifically focus attention on bidirectional associations. Thus, we narrowed our attention on how these emotional and behavioral responses and symptoms reciprocally unfold over the 1-year of this study. Here, we hypothesized reciprocal relations over time, where emotion dysregulation, suppression, and social avoidance would predict increases in symptoms (i.e., an ER-deficit model), at the same time that symptoms would predict increases in these three responses over the 1-year period (i.e., a vulnerability model) (Hypothesis 3). Additionally, we examined differences by gender in Hypotheses 2 and 3, given previous research has indicated that adolescent girls and young women tend to report heightened emotional responses to stress as well as heightened emotional maladjustment over the course of adolescence and young adulthood (De France et al., 2019; Marston et al., 2010; Nolen-Hoeksema & Aldao, 2011; Zimmer-Gembeck & Skinner, 2015). Finally, we estimated an alternate longitudinal RS model, consistent with our assertions that high-RS individuals may experience heightened symptoms whenever this bias is activated, making it more difficult to regulate the emotional effects of RS. Here,

ER-deficits were examined as the outcome and internalizing symptoms as the mediator to determine if this direction of effects appeared as viable as the longitudinal RS model with ER-deficits as mediators.

2 | METHOD

2.1 | Participants and procedure

The participants were 402 adolescents and young adults aged 17 to 27 years ($M = 19.9$, $SD = 2.8$, 66.2% female) who completed two assessments over a 1-year period. Seventy-nine percent identified as white Australians with European descent, while 10.0% identified as Asian, 2.5% as Australian First Peoples or Pacific Islander, and 8.5% as other (inclusive of African, Egyptian, Bosnian etc.). Most participants were domestic university students (69.1%), with 53.5% currently living with their parents. A further 6.0% of participants reported living alone and 26.9% reported living in a shared accommodation. About one third of participants reported that the university level education was the highest level of education for their mother (41.4%) as well as their father (32.1%). Finally, 50.3% of the participants reported their parents were married or living together, with 37.9% identifying their parents as either divorced or separated. The original sample was 661 young people at the time 1 (T1) assessment (retention rate of 61%). We ran sensitivity checks (i.e., independent samples *t* tests) and found no systematic difference in any of the measures of interest or the demographic measures in those who were lost to follow-up and those who were not (*p's* ranged from .08 to .99).

Approval for a longitudinal study was received from the university Human Research Ethics Committee (HREC). Participants were recruited to participate using convenience sampling during the orientation week (the week before the start) of the first trimester of the school year. Students were approached by a researcher in common areas and asked to participate in the study by completing a paper-and-pencil survey. These participants received a chocolate bar or entered a prize draw for gift cards. Once the first trimester started, the first-year psychology subject research pool was also used for recruitment where participants applied for participation in the study and completed an online version of the survey. Upon completion of the survey, these 163 participants (23%) received partial psychology course credit (.5% credit for the course). Independent samples *t* tests revealed no significant differences amongst any of the variables of interest based on recruitment strategy (*p's* ranged from .06 to .99). At T2 (1 year later), participants were contacted by email or telephone and invited to complete an online version of the survey. Following completion of the T2 questionnaire, each participant received a small dollar value gift voucher.

2.2 | Measures

2.2.1 | Depressive symptoms

At T1 and T2, the 10-item Centre for Epidemiologic Studies for Depression Scale—Short form (Radloff, 1977) was used to assess depressive symptoms. Participants rated each statement from 1 (rarely or none of the time—less than 1 day) to 4 (most or all of the time—5 to 7 days). Averaging responses created composite scores, with higher scores indicating more symptoms. Cronbach's alpha was .83 at T1 and .83 at T2.

2.2.2 | Anxiety symptoms

At T1 and T2, the 20-item trait composite of the State-Trait Anxiety Inventory (Speilberger, Gorsuch, & Lushene, 1970) was used to measure anxiety symptoms. Participants rated each statement from 1 (rarely or none of the time—less than 1 day) to 4 (most or all of the time—5 to 7 days). Averaging responses created composite scores, with higher scores indicating more symptoms. Cronbach's alpha was.92 at T1 and .93 at T2.

2.2.3 | Rejection sensitivity

At T1, RS was measured using the RS Questionnaire for University Students (Downey & Feldman, 1996). The RS-US consisted of eight items that assessed participants' rejection expectations and anxiety about rejection. Each item begins with a hypothetical situation in which rejection by a significant other (i.e., parents, friends, and romantic partner) is possible (e.g., "You ask a friend to do you a big favor"). For each situation, participants first indicated the degree of concern or anxiety about the outcome of the situation on a 6-point scale from 1 (very unconcerned) to 6 (very concerned), and then indicated the likelihood that the person would respond in an accepting manner on a 6-point scale from 1 (very unlikely) to 6 (very likely). After multiplying the level of rejection concern by the reverse-scored likelihood levels to produce a RS score for each question, RS scores were averaged to obtain the total RS score, where higher responses indicated greater sensitivity to rejection. Possible scores ranged from a minimum of 1 to a maximum of 36. Cronbach's alpha was .73.

2.2.4 | Deficits in emotion regulation

Three emotion regulation deficits were measured at T1 and T2. First, emotion dysregulation was measured using the six-item composite (e.g., "usually, if I get a feeling of sadness/worry, it paralyses me") of the Emotion Regulation Inventory

(Roth, Assor, Niemiec, Ryan, & Deci, 2009). Second, expressive suppression was measured by the four-item composite (e.g., "I control my emotions by not expressing them") of the Emotion Regulation Questionnaire (Gross & John, 2003). Participants responded on a range from 1 (strongly disagree) to 5 (strongly agree), where items were averaged to form composites, and higher scores on each represented greater use of the relevant strategy. Cronbach's alpha for dysregulation was .87 at T1 and .85 at T2; for suppression was .75 at T1 and .78 at T2.

Third, social avoidance was measured using the Reactions to Implied Rejection Scale: University Student Version (Zimmer-Gembeck & Nesdale, 2013) to assess withdrawal in response to potential rejection experiences. Participants were presented with three scenarios (e.g., "You hear that someone you know is throwing a big birthday party on the beach. Most of your group of friends expect to go. You hear that some of your friends have received their invitations and are excited about the event. You still have not received your invitation and the party is not far off. How would you feel?"), followed by nine items across the three vignettes assessing social avoidance (e.g., "try to avoid situations where you have to mix with others"). Response options ranged from 1 (strongly disagree) to 5 (strongly agree). Averaging items across the three scenarios formed the total score, so that a higher score represented more social avoidance. Cronbach's alpha was .88 at T1 and .89 at T2.

2.3 | Overview of the statistical analyses

Of the final 402 participants, there was less than 2% missing data on all the constructs, and no individual was missing more than two items on any measure. Thus, composite scores were formed based on the completed items. Means (*M*s), standard deviations (*SD*s), and Pearson's correlations between all variables were calculated at T1 and T2, along with identifying differences between young men and women in all the study variables using independent samples *t*-tests. In addition to examining preliminary associations among our variables of interest, we examined the effects of age and gender using these analyses. Here, participants' gender ("what is your sex?," 0: *male*, 1: *female*) and age ("how old are you?") were used. Path analyses using full-information maximum likelihood estimation (FIML) within AMOS software (IBM Corporation) were used for the primary analyses. To further test Hypotheses 1 and 2, we examined direct and indirect effects in a longitudinal RS model, freeing pathways from T1 RS (i.e., predictor) to T2 depressive and anxious symptoms (i.e., outcomes) via T1 emotion dysregulation, expressive suppression, and social avoidance (i.e., mediators) in a single path model, controlling for gender and age effects based on their significant effects from the preliminary analyses (i.e., *t* tests and correlations). In this single model, all paths were freed as hypothesized in Figure 1. To test hypotheses regarding indirect pathways from T1 RS to T2 depressive and T2 anxious symptoms separately within the single path model, bootstrapping, using 200 samples, was used to estimate standard errors and 95% bias-corrected confidence intervals for all effects.

A second path model was then tested to examine Hypothesis 3. Here, bidirectional effects between the three measured ER-deficits and the two measured depressive and anxious symptoms at T1 and T2 were tested by specifying a single cross-lagged panel model. To evaluate model fit for both the longitudinal and bidirectional models, goodness-of-fit indices (χ^2, χ^2 relative to sample size, goodness of fit index-GFI, comparative fit index-CFI, and root mean square error of approximation-RMSEA) and parameter estimates for model paths were considered. Acceptable model fitness was determined by the following parameters: $\chi^2/df \leq 3$, GFI and CFI values $\geq .95$ and a RMSEA value $\leq .05$ as suggested by recommendations in Byrne (2016). Following on from the above analyses, multi-group analyses were conducted for both the longitudinal and bidirectional models to test whether model paths in each were invariant by gender. Finally, we fit an alternative path model with internalizing symptoms as the mediator. In this model, the longitudinal associations of T1 RS with T2 emotion dysregulation, expressive suppression, and social avoidance as the outcomes were estimated when T1 depressive and anxious symptoms were specified as the mediator.

3 | RESULTS

3.1 | Means, standard deviations, gender differences, and zero-order correlations

Table 1 presents the *Ms, SDs*, and gender differences of all the measures. Young women, compared to young men, reported higher emotion dysregulation and anticipated using more social avoidance in response to rejection vignettes. Young women also reported less expressive suppression at both T1 and T2, relative to young men.

As shown in Table 2, T1 RS was associated with endorsing more symptoms and greater dysregulation, suppression, and social avoidance at both T1 and T2, except for T2 expressive suppression. T1 expressive suppression was associated with greater T1 and T2 symptoms, along with greater dysregulation at T2. Both T1 and T2 emotion dysregulation and social avoidance were positively interrelated and associated with more symptoms within and across waves. There was also moderate stability evidenced between all variables across waves. Finally, age was negatively associated with T1 and T2 anxious symptoms, T1 and T2 expressive

TABLE 1 Means (M) and standard deviations (*SD*) for all participants, for males and females, and tests of gender differences ($N = 402$)

Measure	Overall, *M* (*SD*)	Young men, *M* (*SD*) $n = 136$	Young women, *M* (*SD*) $n = 266$	*t*(1,400)
T1 Depressive symptoms	1.99 (.60)	1.96 (.61)	2.02 (.59)	−.98
T1 Anxiety symptoms	2.13 (.60)	2.07 (.57)	2.16 (.61)	−.46
T1 Expressive suppression	2.85 (.80)	3.08 (.77)	2.74 (.80)	4.06**
T1 Emotion dysregulation	3.00 (.96)	2.78 (.99)	3.11 (.92)	−3.31*
T1 Social avoidance	2.98 (.89)	2.82 (.83)	3.06 (.91)	−2.48*
T1 Rejection sensitivity	9.14 (4.08)	8.89 (3.54)	9.27 (4.33)	−.87
T2 Depressive symptoms	2.00 (.58)	1.98 (.51)	2.01 (.61)	−.48
T2 Anxiety symptoms	2.18 (.60)	2.14 (.54)	2.21 (.63)	−1.05
T2 Expressive suppression	2.73 (.85)	2.95 (.87)	2.62 (.82)	3.70**
T2 Emotion dysregulation	2.99 (.86)	2.84 (.90)	3.07 (.84)	−2.40*
T2 Social avoidance	3.20 (.91)	2.98 (.86)	3.31 (.92)	−3.50*

Abbreviations: T1, time 1; T2, time 2.

*$p < .05$; **$p < .01$.

TABLE 2 Zero-order correlations between all measures at T1 and T2 ($N = 402$)

Measure	1	2	3	4	5	6	7	8	9	10	11
1. T1 Depressive symptoms	–										
2. T1 Anxiety symptoms	.84**	–									
3. T1 Expressive suppression	.20**	.22**	–								
4. T1 Emotion dysregulation	.50**	.53**	.03	–							
5. T1 Social avoidance	.31**	.38**	.06	.35**	–						
6. T1 Rejection sensitivity	.35**	.43**	.22**	.30**	.34**	–					
7. T2 Depressive symptoms	.45**	.43**	.16**	.34**	.22**	.25**	–				
8. T2 Anxiety symptoms	.45**	.51**	.14**	.37**	.28**	.31**	.86**	–			
9. T2 Expressive suppression	.08	.09	.44**	−.00	.06	.06	.19**	.22**	–		
10. T2 Emotion dysregulation	.36**	.38**	−.01*	.57**	.27**	.19**	.49**	.54**	.07	–	
11. T2 Social avoidance	.20**	.28**	.03	.27**	.46**	.23**	.41**	.46**	.18**	.39**	–
12. Age	−.09	−.10*	−.23**	−.10	.00	−.10	−.11	−.14**	−.20**	−.10*	−.07

Abbreviations: T1, time 1; T2, time 2.

*$p < .05$; **$p < .01$.

suppression, and T2 emotion dysregulation. Given the significant associations of gender and age with the main variables under investigation, we included gender and age as covariates in our longitudinal RS model of ER-deficits and internalizing symptoms below.

3.2 | A longitudinal RS model of ER-deficits and internalizing symptoms

3.2.1 | Structural model

In the first model, all paths were freed from T1 RS to T2 depressive and anxious symptoms; from T1 RS to T1 emotion dysregulation, suppression, and social avoidance; and from T1 emotion dysregulation, suppression, and social avoidance to T2 depressive and anxious symptoms (as shown in Figure 1). Gender, age, and T1 depressive and anxious symptoms were also considered as controls. After freeing some error variances that were theoretically viable, the model demonstrated an acceptable fit to the data [χ^2 (15) = 24.63, $p = .06$, $\chi^2/df = 1.64$, GFI = .99, CFI = .99, RMSEA = .04 (.000–.067), $p = .69$]. The overall model accounted for 21% of the variance in T2 depressive symptoms, and 26% of the variance in T2 anxious symptoms. Table 3 presents the path estimates, standard errors, and 95% CIs. As can be seen, there were several direct effects in the model, with small to moderate effect sizes ranging from .10 to .33. T1 RS was associated with higher T1 dysregulation, suppression, social avoidance, and increased

TABLE 3 Direct and indirect associations of rejection sensitivity with internalizing symptoms via ER-deficits ($N = 402$)

	Standardized estimates			Unstandardized estimates		
	B (*SE B*)	Lower 95% CI	Upper 95% CI	*B* (*SE B*)	Lower 95% CIs	Upper 95% CIs
Direct effects						
T1 RS → T2 Depressive symptoms	.07 (.05)	.01	.16	.01 (.01)	.00	.02
T1 RS → T2 Anxious symptoms	.10* (.05)	.03	.17	.01* (.01)	.00	.03
T1 RS → T1 Dysregulation	.30** (.05)	.21	.37	.07** (.01)	.05	.09
T1 RS → T1 Suppression	.21** (.05)	.13	.30	.04** (.01)	.03	.06
T1 RS → T1 Social avoidance	.33** (.05)	.25	.41	.07**(.01)	.05	.09
T1 Dysregulation → T2 Depressive symptoms	.16* (.05)	.08	.24	.09* (.03)	.05	.14
T1 Dysregulation → T2 Anxious symptoms	.16* (.05)	.07	.25	.10* (.03)	.05	.16
T1 Suppression → T2 Depressive symptoms	.08 (.05)	.01	.16	.06 (.03)	.00	.11
T1 Suppression → T2 Anxious symptoms	.05 (.04)	−.03	.12	.03 (.03)	−.02	.09
T1 Social avoidance → T2 Depressive symptoms	.06 (.06)	−.03	.15	.04 (.03)	−.02	.10
T1 Social avoidance → T2 Anxious symptoms	.07 (.05)	−.02	.16	.05 (.03)	−.01	.10
T1 Depressive symptoms → T2 Depressive symptoms	.28** (.04)	.22	.35	.26** (.04)	.20	.33
T1 Anxious symptoms → T2 Anxious symptoms	.31** (.05)	.24	.40	.31** (.04)	.23	.38
Indirect effects						
T1 RS → T2 Depressive symptoms	.08* (.03)	.05	.13	.01* (.00)	.01	.02
T1 RS → T2 Anxious symptoms	.08* (.03)	.04	.13	.01* (.00)	.01	.02

Note: The indirect effects from T1 RS to T2 depressive and anxious symptoms occurs via the summative effect of the three ER-deficits.

Abbreviations: RS, rejection sensitivity; T1, time 1; T2, time 2.

*$p < .05$; **$p < .01$.

anxious symptoms at T2 relative to T1. T1 dysregulation also predicted increased depressive and anxious symptoms at T2 relative to T1. There were also indirect effects to note. T1 RS had positive indirect associations with increased depressive and anxious symptoms at T2 relative to T1 via the summed effect of the three T1 ER-deficits.

When all the paths in the model were freed to differ for young men and women, the fit of the two-group model was good [χ^2 (20) = 42.441, $p < .05$, CFI = .99, RMSEA = .05 (.031–.075), $p = .38$]. When all model paths were fixed to equality for both men and women, the resulting χ^2 value of 85.6267 with 43 degrees of freedom significantly differed from that of the two-group model fit: χ^2_{diff} (23) = 42.83, $p < .05$. Upon further examination, one path significantly differed between men and women, from T1 RS to T1 emotion dysregulation. The association was significant for both young men and women, but significantly stronger for men ($\beta = .45$, $p < .01$) than women ($\beta = .23$, $p < .01$).

3.3 | Bidirectional associations between ER-deficits and internalizing symptoms

The results of the cross-lagged path model are shown in Figure 2, with standardized estimates shown. Not displayed on the figure, all covariances among the measures assessed at the same time were freed, and all were significant, except one (between expressive suppression and social avoidance at T1; $p = .31$). All paths from T1 to T2 measures were also freed. The model demonstrated good fit to the data [χ^2 (10) = 19.86, $p < .05$, $\chi^2/df = 1.99$, GFI = .99, CFI = .99, RMSEA = .05 (.015–.081), $p = .46$]. Furthermore, as seen in Figure 2, the model accounted for between 19% (suppression) and 31% (dysregulation) of the variance in the T2 outcomes.

As seen in Figure 2, five of the prospective paths were significant, with small effect sizes ranging from .09 to .22. In support of an ER-deficit hypothesis, T1 emotion dysregulation and suppression predicted increased depressive and anxious symptoms at T2 relative to T1. Additionally, there was support for adjustment as a precursor to increasing ER-deficits, where T1 anxious symptoms were associated with increased social avoidance at T2 relative to T1. When all the paths in the model were freed to differ for young men and women, the fit of the two-group model was excellent [χ^2 (20) = 35.106, $p < .05$, CFI = .99, RMSEA = .04 (.017–.067), $p = .65$]. When all model paths were then fixed to equality for both men and women, the resulting χ^2 value of 69.690 with 55 degrees of freedom did not significantly differ from that of the two-group model fit: χ^2_{diff} (35) = 34.58, $p > .05$; indicating that the model was invariant by gender.

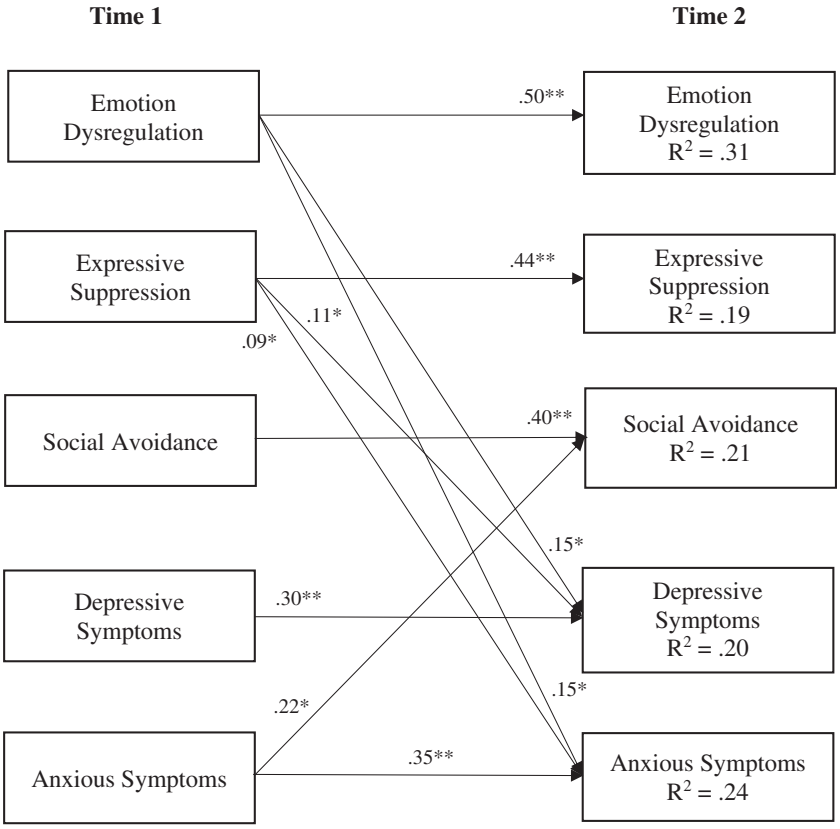

FIGURE 2 Results of the bi-directional associations over time. All cross-lag paths were freed in the model, but only the significant standardized associations are shown here ($N = 402$). *$p < .05$. **$p < .01$

3.4 | An alternate longitudinal RS model

An alternate model was fit to test if RS was prospectively associated with increases in ER-deficits at T2 via T1 symptoms as mediators. Paths were freed from T1 RS to T1 depressive and anxious symptoms, from T1 RS to T2 emotion dysregulation, suppression, and social avoidance, and from T1 depressive and anxious symptoms to T2 emotion dysregulation, suppression, and social avoidance. Furthermore, the model controlled for the effects of gender, age, and T1 emotion dysregulation, suppression, and social withdrawal. The model demonstrated adequate fit to the data [χ^2 (15) = 25.49, $p < .05$, $\chi^2/df = 1.70$, GFI = .99, CFI = .99, RMSEA = .04 (.007–.069), $p = .66$]. However, in the model, there were fewer associations over time relative to our hypothesized model with ER-deficits as mediators; the only significant effect across time was a small effect of T1 anxious symptoms on T2 social avoidance ($\beta = .02$, $p = .01$).

4 | DISCUSSION

The transition to adulthood offers unique opportunities for exploring new social settings and affirming one's identity, yet can also present challenges based on manifold possibilities for entering and developing new close relationships, or renegotiating and deepening intimacy in preexisting relationships (Coyne et al., 2019; O'Rourke et al., 2018; Zimmer-Gembeck, 2002). For adolescents and young adults struggling with RS, such encounters can significantly threaten their well-being (Downey & Feldman, 1996; Gao et al., 2017; Levy et al., 2001; Marston et al., 2010). Guided by RS theory (Downey & Feldman, 1996), we explored this process longitudinally, and examined ER-deficits as potential mediators of the RS-internalizing symptoms relations. Findings indicated that indeed those individuals who reported heightened RS also reported increased symptoms, and that these associations were partially indirect, vis-à-vis all three ER-deficits of emotion dysregulation, expressive suppression and social avoidance. Moreover, in a follow-up model exploring bidirectional effects of ER-deficits and internalizing symptoms, ER-deficits were associated with youth's increased symptoms over time, rather than the converse. Thus, deficits in ER (as opposed to vulnerabilities stemming from symptoms) were supported from our findings. Finally, there were gender differences in each of the ER-deficits, but when gender was tested as a moderator of model paths, only one association, which was between T1 RS and T1 emotion dysregulation, was found to differ; the strength of this association was stronger for young men compared to young women. Overall, these findings further demonstrate the detrimental effects of RS for young people's adjustment, while also identifying

potential pathways via which RS may lead to greater symptoms over time.

4.1 | Longitudinal RS-internalizing model: ER-deficits as mediators

Consistent with previous empirical work on RS and associated symptoms or disorders (Gao et al., 2017; Gardner & Zimmer-Gembeck, 2018; Marston et al., 2010; Watson & Nesdale, 2012; Zimmer-Gembeck et al., 2016), and supporting Hypothesis 1, results from the multivariate path model revealed that young people with heightened RS reported more ER-deficits and, in turn, ER-deficits, specifically emotion dysregulation, predicted increased internalizing symptoms. However, the results from the path model of RS, ER-deficits, and internalizing symptoms revealed that, unlike the finding of both direct and indirect effects of RS on anxious symptoms, RS only had an indirect temporal effect on depressive symptoms via the ER-deficits. Thus, partially supporting Hypothesis 2, this finding suggests that elevated RS is directly related to increasing anxious symptoms over time, but that RS has a role in increasing depressive symptoms only when RS leads to the ER-deficits. This pattern of findings may be attributable to RS being conceptualized as an anxious social-information bias, thus appearing to be targeted, and possibly more directly relevant to symptoms of anxiety than to depressive symptoms. As such, whether directly or indirectly through ER-deficits, high-RS has consistently been found to place young people at heightened risk of more elevated internalizing symptoms.

Notably, these three ER-deficits tapped two aspects of emotion-specific ER (emotion dysregulation and expressive suppression of sad and worry emotions), as well as a specific reaction to implied rejection vignettes (social avoidance) known to be related to internalizing symptoms and RS. With this in mind, the findings suggest that, both in and outside of situations where expectations of rejection may occur, adolescents and young adults high in RS also have more difficulties with ER. That is, they reported feeling heightened emotionality when stressed, are more likely to engage in attempts to suppress emotions, and are more likely to rely on social withdrawal in response to potentially rejecting circumstances. In other words, young people high in RS may miss out on opportunities to find support within relationships, which themselves could provide a better sense of acceptance and belonging, thereby confirming their fears of rejection (Levy et al., 2001; Watson & Nesdale, 2012; Zimmer-Gembeck, 2015, 2016). Thus, in an attempt to avoid the potential of rejection, and the negative emotions and attributions that may follow, high-RS young people may unintentionally and indirectly experience more symptoms through their overuse of these maladaptive emotional and behavioral responses to events.

4.2 | Bidirectional associations: ER-deficits and internalizing symptoms

Beyond a mediating role of ER-deficits, focusing on the last model, our findings point to ER-deficits and internalizing symptoms largely supported a deficit model, such that ER-deficits led to increases in symptoms, rather than vice versa. Although past research has been mixed regarding directions of effects (De France et al., 2019; McLaughlin et al., 2011), study findings accord with a growing body of research, indicating that dysregulated emotional expression (Gardner & Zimmer-Gembeck, 2018; McLaughlin et al., 2011; Peters et al., 2015; Roth et al., 2009), suppression of emotions (Compas et al., 2017; Gross & John, 2003; Nolen-Hoeksema & Aldao, 2011), and social avoidance (Watson & Nesdale, 2012; Zimmer-Gembeck, 2015; Zimmer-Gembeck & Nesdale, 2013) precipitate increasing symptoms over time. As such, ER-deficits seem to be a key source of risk for increased symptoms during adolescence and young adulthood (Gross & John, 2003; McLaughlin et al., 2011; Nolen-Hoeksema & Aldao, 2011), and these ER-deficits are more elevated among youth who report more RS.

4.3 | Anxiety as a precursor to social avoidance

Although not the primary direction of effect, we did find some support for a vulnerability model, such that anxiety symptoms predicted increases in social avoidance over the 1 year of this study. This pathway was supported in our bidirectional and alternative models (which also considered the effects of RS, age, and gender). This finding accords with previous research whereby individuals who report greater anxious symptoms (or greater anxious temperament) were more likely to also endorse greater avoidance in response to threatening experiences (O'Rourke et al., 2018; Wong & Rapee, 2016; Zimmer-Gembeck & Skinner, 2015). As such, one of the behavioral manifestations of individuals who experience increased anxiety seems to be an increasing avoidance of potentially anxiety-provoking situations. This may occur because avoidance is an effective strategy when one wants to flee from stimuli and avoid the future possibility of more threat and associated distress (Wong & Rapee, 2016). As a result, in the context of interpersonal relationships, avoidance seems to be reinforced over time, by providing, short-term relief from fears, worry, and distress.

Although anxiety was a precursor to avoidance, there was no evidence of a reciprocal effect. That is, social avoidance did not predict increased depression or anxiety in the present study. It may be the case that social avoidance precipitates internalizing symptoms over a longer period than was measured here. Indeed, relying on this response when distressed

may eventually place individuals at greater risk of further social and emotional problems because avoidance reduces the likelihood of learning appropriate coping strategies (e.g., seeking support from others). When young people are unable to manage their distress over the long-term, avoidance may further reduce opportunities for developing social skills and competencies, thereby reinforcing the RS bias as well (Levy et al., 2001; Wong & Rapee, 2016; Zimmer-Gembeck, 2016). Thus, social avoidance may still have longer term negative effects on internalizing symptoms as individuals progress further into adulthood.

4.4 | Gender differentiated patterns in ER-deficits and RS

Finally, as expected, women reported higher levels of emotion dysregulation and social avoidance, and young men reported higher levels expressive suppression across time. These findings are consistent with previous literature, indicating that gender does identify differences with how young people cope with negative emotions and interpersonal stress (Gardner & Zimmer-Gembeck, 2018; Masters et al., 2019; Nolen-Hoeksema & Aldao, 2011; Perry-Parrish & Zeman, 2011; Zimmer-Gembeck & Skinner, 2015). However, contrary to expectations, in multi-group path models, there were gender differences in the longitudinal RS model. Upon further examination, the only path that differed between men and women, albeit concurrently, from T1 RS to T1 emotion dysregulation. This link was stronger for young men than young women. This could perhaps be interpreted in that rejection may be perceived as a greater threat to men's overall sense of acceptance, belonging, and social status (Marston et al., 2010; Perry-Parrish & Zeman, 2011; Zimmer-Gembeck, 2016). Consequently, RS may concurrently be more strongly tied to greater dysregulated emotional expression in young men, because of the potential implications on their social status and acceptance when perceiving the possibility of rejection. Thus, although young women are known to be at greater risk for maladaptive coping responses in general, young men may be more sensitive to potential cues of rejection and the emotional fallout if rejection does occur.

4.5 | Limitations, future research directions, and implications

Though our study revealed several noteworthy findings, it is not without limitations. First, despite the strength of using a longitudinal design, all data were self-report, and were limited by shared method variance. Furthermore, because we utilized the standard period of 1 week on the CES-D measure for reporting of symptoms, relying on this self-report data for the outcome measures may have missed a portion of emotional experiences recalled over the duration of the follow-up period. However, because we used the STAI, a trait-based measure which is less sensitive to change over time, we believe this inclusion helps to further strengthen our findings of the predictive ability of RS in what can be considered a highly stable construct. Second, our use of two measurement waves, allowed us to offer unique insight into the reciprocal relations between ER-deficits and symptoms over time. However, a design including more measurement points will allow for the testing of a "true" mediation model, and will facilitate developmental tracking of experiences as young people develop into adulthood. Third, though our models found significant effects across all models tested that were similar in effect size to those found in previous literature (see Marston et al., 2010; Zimmer-Gembeck et al., 2016), future research should consider including attributions (such as self-and other blame) and perceived rejection in other salient relationships (e.g., romantic rejection), which may be correlates of the negative emotions and behaviors that increase risk for maladjustment over time. Finally, our sample of university students, though diverse, may not be particularly representative of young people who have not attended university or are from other cultural backgrounds. Further to this limitation, the average young adult in this study reported symptoms in the low/mild range, and while there was a good distribution of scores with some low and some high in symptoms, it is difficult to know how this might impact on the results. For example, we know that many young adults in community samples (even university students) report suffering from mental health disorders and many report they have had contact with mental health services for their symptoms (Orygen, 2017). However, it is possible that the effects (associations between variables) might be weaker or stronger if more participants had a very high level of symptoms than reported here.

Nonetheless, our findings build on understanding of RS as a risk for internalizing symptoms in young people, and highlight promising targets for prevention (Levy et al., 2001; Watson & Nesdale, 2012; Zimmer-Gembeck, 2015). Here, findings indicate that one plausible way to buffer against the toxic effects of RS is to directly target the mechanisms or components that increase risk of psychopathology over time. Thus, high-RS individuals may greatly benefit from learning adaptive coping strategies related to reappraising or being more flexible in interpreting rejection cues, experiencing and appropriately expressing sad and worry emotions, and decreasing avoidance-based coping strategies, while subsequently enhancing approach or behavioral distraction-based techniques (Downey & Feldman, 1996; Levy et al., 2001; Watson & Nesdale, 2012;

Zimmer-Gembeck, 2015, 2016). Additionally, as our findings indicate, that those individuals who may be higher in trait-anxiety may particularly benefit from learning more approach-based strategies that reduce emotional distress and increase well-being in the future (Wong & Rapee, 2016). Future research should build toward identifying which specific coping strategies buffer against specific components of the RS model (such as reappraising biased interpretations, acceptance or distraction during intense emotional experiences, and accessing social support from significant and accepting others). Such inquiry can be helpful in reducing the risk of RS for overall well-being.

5 | CONCLUSION

Difficulties in managing emotional reactions (i.e., dysregulation and suppression of emotion) and avoidance of social interactions that may potentially elicit rejection, appears to be a critical pathway linking RS and internalizing symptoms for adolescents and young adults over time. Moreover, anxiety symptoms, in particular, increases young people's risk for engaging in more avoidance over time. These findings build upon the original RS model (Levy et al., 2001), in which RS is associated with increased ER-deficits and internalizing symptoms over time. As such, reciprocal interactions exist in young people's perceptions of, and sensitivity toward, interpersonal rejection, in turn revealing important and novel implications for overall adjustment. Given that social relationships provide an essential context for acceptance and belonging, but also carry with them the potential for experiencing rejection, continued research in understanding how emotions and behaviors are implicated in this process is warranted.

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CONFLICT OF INTEREST

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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