ADOLESCENT EXPERIENCES OF EMPATHY, INTERNALIZING SYMPTOMS, AND STRESS: BEFORE AND DURING THE COVID-19 PANDEMIC

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ABSTRACT

Adrianna N. Richards: Adolescent Experiences of Empathy, Internalizing Symptoms, and Stress: Before and During the COVID-19 Pandemic
(Under the direction of Andrea M. Hussong)

In addition to links with prosocial outcomes, empathy may convey risk for adolescent internalizing symptoms, which may differ for affective and cognitive empathy. The empathy-internalizing symptom link may also be influenced by the stressful context and empathic decision making of the COVID-19 pandemic. No known studies have assessed the interaction between affective and cognitive empathy and their relation to internalizing symptoms, or allostatic load as a potential mechanism underlying this association. Data from before and during the COVID-19 pandemic were analyzed to assess links between adolescents' affective and cognitive empathy, internalizing symptoms, and pandemic-related stress. No concurrent or prospective links between internalizing symptoms and the two forms of empathy, or their interaction, emerged. Pandemic-related stress was not a significant moderator. Sampling bias and low-to-moderate internal reliability of adolescent self-reported affective and cognitive empathy may have contributed to null effects. Implications and future directions are discussed.

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Empathy contributes to prosocial outcomes (Eisenberg et al., 1991; Garaigordobil, 2009; Van der Graaff et al., 2018) but may also create risk for internalizing symptoms, such as those related to depression and anxiety (Tone & Tully, 2014). This risky association with internalizing symptoms, however, may differ for affective empathy (i.e., the emotional reflection of another's emotions involving more self-focus) and cognitive empathy (i.e., the ability to take another's perspective involving more other-focus), with prior literature showing stronger associations between affective, rather than cognitive, empathy and internalizing symptoms (Gambin & Sharp, 2018).

This association may be of particular salience in adolescence, as empathic capabilities (Eisenberg, 1986) and displays (Eisenberg et al., 2009) expand, and internalizing symptoms increase (CDC, 2020). Moreover, interventions based in mindfulness (Cheang et al., 2019) and social-emotional learning (Malti et al., 2016) seek to increase adolescent empathy but do not account for the potential accompanying risk for internalizing symptoms in some youth. As internalizing symptoms are among the most common mental health symptoms experienced in adolescence (CDC, 2020), understanding and accounting for the role of empathy in risk for adolescent internalizing symptoms is vitally important.

Stressful ecological contexts, such as that provided by the COVID-19 pandemic and related policies, may magnify the association between empathy and internalizing symptoms in multiple ways. First, increased stress exposure may enhance experiences of internalizing

symptoms in adolescents (Grant et al., 2004; Sirin et al., 2015; perhaps particularly in girls [Ge et al., 1994]). Second, the nature of the COVID-19 pandemic, as a public health crisis presenting an array of decisions about how to protect oneself and others from risk, may have specific implications for self- and other-oriented processes that involve empathic responding (Gardiner, 2020; Pfattheicher et al., 2020). The current study aims to explore the differential associations of cognitive and affective empathy with internalizing symptoms both before and during pandemic stress exposure. Broadening our understanding of the unique and possibly connected roles of cognitive and affective empathy in experiences of adolescent internalizing symptoms within this ecological context may inform interventions targeting adolescent empathy and internalizing symptoms while accounting for experiences of stress.

Empathy and Internalizing Symptoms in Adolescence

Heterogeneity in defining empathy has translated to variability in its measure and study in the literature. *Integrated* definitions of empathy describe it as a general way of responding to another's experiences (Davis, 1983), whereas *noncompound* definitions highlight empathy's cognitive (i.e., one's understanding of another's emotional state) and affective (i.e., one's emotional response to another's emotional state) components (Hoffman, 2000). The noncompound conceptualization, employed in the current work, provides greater distinction between the nature and impact of each form of empathy and allows exploration of whether affective and cognitive empathy relate to adolescent internalizing symptoms in different ways.

One important distinction between the two forms of empathy is their locus of focus—on the self or on the other. For example, affective empathy is often associated with a self-focus that may entail personal distress (Smith & Rose, 2011), wherein one's affective response to another's distress begins with a focus on the other person, but becomes either so strong or persists for so

long that focus shifts to the self—leading the individual to internalize the other person's distress. Cognitive empathy, on the other hand, involves differentiating the self from the other (Decety & Jackson, 2004) and taking the other's perspective so as to understand their feelings, reflecting an other-focus (Davis, 1983). As such, cognitive empathy involves similar neurological pathways to theory of mind, another other-focused cognitive process (Shamay-Tsoory et al., 2009).

In addition to the different foci of affective and cognitive empathy, scholars assert that there is dissociation between these two empathic processes (Shamay-Tsoory et al., 2004) and suggest that these two components of empathy operate via differential neural pathways (Shamay-Tsoory et al., 2009). Brain lesions in areas associated with affective empathy (e.g., the inferior frontal gyrus) are linked to decrements in affective, but not cognitive, empathy, while damage to areas of the brain linked to cognitive empathy (e.g., the ventromedial prefrontal region) are associated with decrements in cognitive, but not affective, empathy. Further supporting this distinction, a cognitive perspective taking intervention significantly increased cognitive empathy but had no effect on affective empathy (Van Loon et al., 2018). Cognitive and affective empathy also relate differently to altruistic sharing behaviors (Edele at al., 2013), prosocial bystander behavior in instances of cyberbullying (Barlinska et al., 2018), and adolescent conflict behaviors with their parents (Van Lissa et al., 2017).

Another difference between the two forms of empathy may be in how they relate to internalizing symptoms via their impact on physiological stress systems. Specifically, the increased self-focus of affective empathy may lead to internalizing symptoms by activating the stress-response system, producing allostatic load. Allostasis refers to the activation of one's stress-response system following exposure to a stressor (McEwen, 1998)—a normative process typically followed by homeostatic recovery, in which the stress-response system deactivates

following stress exposure and returns to a healthy baseline. When stress exposures are too intense or chronic, however, the stress-response system can remain activated, producing allostatic load and an imbalance of one's stress-response system due to its hyperactivation (McEwen, 1998). Persistent allostatic load is associated with numerous negative physical and mental health outcomes, including internalizing symptoms (Cicchetti et al., 2011; McEwen, 2003; Stroud et al., 2019).

Among the stressors that elevate allostatic load are psychologically-based stressors including burnout (Hintsa et al., 2016), social inhibition (Duijndam et al., 2020), and children's experiences of parental marital conflict (Hinnart et al., 2013). As these findings suggest, increased emotional distress, like that experienced at higher levels of affective empathy and in response to another's stressor, may trigger one's stress-response system in the same way that one's own intense or chronic stressor would. Over time, repeated activation of allostatic load diminishes the effectiveness of the stress-response system and may lead to dysregulation of the hypothalamic-pituitary-adrenal (HPA) and sympathetic-adrenal-medullary (SAM) axis as well as to disrupted neurobiological systems that underlie physical and mental health (Rogosch et al., 2011). Indeed, internalizing symptoms have repeatedly been linked to allostatic load in the literature (Cicchetti et al., 2011; Stroud et al., 2019).

In contrast to affective empathy, cognitive empathy involves maintaining focus on the perspective of another, is not linked with personal distress, and thus may not trigger allostatic load and related mental health consequences. Whereas the literature has yet to directly investigate cognitive empathy as it relates to allostatic load, research has shown that both elevated posttraumatic stress responses and psychological distress in disaster workers were positively linked to personal distress—the self-focused, affective component of empathy—but

were not related to perspective taking, the other-focused cognitive component of empathy (Nagamine et al., 2018). The ability to distinguish oneself and one's experiences from those of another, a self-other awareness inherent to cognitive empathy (Decety & Jackson, 2004), may help to explain why cognitive empathy in reaction to another's emotional display may not trigger allostatic load in the same way that affective empathy may. Without activating allostatic load, cognitive empathy may not produce physiological dysregulation and associated risk for internalizing symptoms.

Consistent with these posited distinctions, affective and cognitive empathy evidence differential associations with internalizing symptoms in adolescents. Macdonald and Price (2019) identified a positive relation between affective empathy and internalizing symptoms in a sample of older adolescents and emerging adult college students and Gambin and Sharp (2016) found that anxiety problems strongly predicted affective empathy in adolescent boys and girls. Additional work with adolescents by Green and colleagues (2018) revealed that affective empathy was not only associated with higher depression and lower self-esteem at two time points (grades 7-9, and 8-10), but also significantly predicted *increases* in depressive symptoms and *decreases* in self-esteem over that time. Cognitive empathy, however, was negatively related to depressive symptoms (when youth were in grades 8-10) and predicted *decreases* in depression and *increases* in self-esteem. In adolescent inpatients, Gambin and Sharp (2018) found that affective empathy was positively related to depression and all (seven) dimensions of anxiety assessed in the study, whereas cognitive empathy was negatively linked to depression and to three of the seven included dimensions of anxiety. This body of work suggests differential impacts of affective and cognitive empathy on internalizing symptoms in adolescents. Seeking to replicate this finding, the first hypothesis of the current study posits that affective empathy will

be positively associated with internalizing symptoms and that cognitive empathy will be negatively associated with internalizing symptoms in adolescents.

COVID-19 Stress as an Exacerbating Factor

Stressful ecological contexts increase internalizing symptoms (Grant et al., 2004; Sirin et al., 2015) and affect interpersonal dynamics (Krzysztof, 2020). Due to its public health consequences, the COVID-19 pandemic in particular may impact empathic responding and how individuals think about the self and the other (Gardiner, 2020; Pfattheicher et al., 2020). While COVID-19 is ubiquitous in that it is experienced globally, it does differ in its impact on various individuals. To account for this, scholars have assessed individual categories of COVID-19-related stressors which are experienced to varying degrees, including those related to general life disruption, interpersonal relations, finances, educational or professional goals, and health as it relates to the self and the other. Related studies have found that many of these forms of stress are related to symptoms of depression and/or anxiety during the pandemic (Kujawa et al., 2020). With potential implications for both empathy and internalizing symptoms, experiences of the COVID-19 pandemic will be tested in the current study as amplifiers of the association between affective empathy and internalizing symptoms.

Stress theories regarding allostatic load can explain why the pandemic context may act to moderate the relation between affective empathy and internalizing symptoms. Despite individual variation, the globality of the novel pandemic provides a ubiquitous and ongoing stress exposure, activating stress-response systems and perhaps maintaining this activation over time as the pandemic persists (producing allostatic load). This increased risk for allostatic load due to the stress of the pandemic context may combine with increased risk for allostatic load due to high levels of affective empathy to exacerbate the overall likelihood of adolescents' experiences of

allostatic load and related internalizing symptoms.

This synergistic effect of pandemic stress and affective empathy may be evident in at least two ways. First, we may see this risk cross-sectionally. If both the pandemic context and affective empathy are activating the stress-response system, then the association between affective empathy and internalizing symptoms may be stronger during COVID-19 than before COVID-19, as the combined effects of the pandemic context as a stressor and internalized distress by means of affective empathy may foster a greater likelihood of allostatic load and thus risk for internalizing symptoms. Second, we may see this risk prospectively. Higher levels of affective empathy before COVID-19 may result in entering the pandemic with an already activated stress-response system that results in problematic allostatic load once pandemic stress onsets, thus predicting greater risk for internalizing symptoms during COVID-19.

Additionally, given the wide range of individual stress exposures during the pandemic, the extent to which the pandemic context interacts with affective empathy to produce allostatic load and increase risk for internalizing symptoms may depend on the level of pandemic stress experienced by an adolescent. As such, I predict that adolescents' levels of perceived COVID-19 stress will moderate the association between affective empathy and internalizing symptoms during the COVID-19 pandemic such that the association will be stronger for those with higher levels of COVID-19-related stress.

Cognitive Empathy as a Potential Buffer

Research has considered both the affective and cognitive components of empathy in the study of internalizing symptoms in adolescents, but has not, to date, investigated the interaction between these two components. Although cognitive and affective empathy have shown moderate correlations in some prior work (Reniers et al., 2010; Davis, 1983), neurobiological and

intervention findings differentiate the two (Shamay-Tsoory et al., 2009; Van Loon et al., 2018). These findings indicate that affective and cognitive empathy are at least partially independent; these components may also then interact.

One way to conceptualize the ways that these forms of empathy may interact is through the strength model of self-control (Baumeister et al., 2007). This model elaborates on earlier theorizations of self-control or self-regulation as a muscle (Baumeister et al., 1998) and explains how self-control resources are finite. Effortful regulation depletes these resources and leaves fewer resources available to complete additional tasks. Thus, if two processes drawing upon effortful self-regulatory resources occur simultaneously, one's resources will need to be divided, with neither process drawing upon the full capacity of self-regulatory resources.

A central tenet of this model is that self-regulation resources act on effortful processes. An important question, then, is whether empathy is automatic or effortful. Scholars in areas of developmental, social, and neuropsychology have presented support for at least some automatic elements of empathy, particularly of affective empathy (Eisenberg & Mussen, 1989; Hatfield et al., 1994; & Ferrari et al., 2003, respectively). More recent work, however, suggests that in addition to these automatic elements, there are effortful processes inherent to empathic responding. For example, Schumann et al. (2014) highlight the ways that empathy can involve effort, and does so to varying degrees depending on one's context. Cameron et al., (2019) investigated preferences for engaging in empathic or non-empathic tasks and found that participants rated the empathic task as more effortful than the non-empathic task and that both affective and cognitive components of the empathic task were found to be effortful. In addition, they found that participants were less likely to choose the empathic task over time through repeated trials, consistent with Baumeister et al.'s assertion that resources to engage in effortful

tasks would deplete over time.

Given evidence, then, that both affective and cognitive empathy require some effortful control, the strength model of self-control may explain how affective and cognitive empathy may interact during empathic responding and relate to internalizing symptoms. Specifically, affective empathic processes (including emotion recognition, contagion, and/or mimicry; Shamay-Tsoory, 2009) occurring alongside cognitive empathic processes (including mentalization and a self-other awareness which distinguishes oneself from another; Decety & Jackson, 2004) may both draw from one's finite self-regulatory resources. More resource allocation to cognitive empathy via a self-other awareness, for example, may then limit resources available for affective empathic processes like emotional contagion. Reduced resources available for affective processes may reduce the potential for one's affective empathic response to trigger personal distress, allostatic load, and risk for internalizing symptoms. If this is the case, then higher levels of cognitive empathy may decrease the likelihood that one will experience internalizing symptoms, even at higher levels of affective empathy. Using this self-regulatory framework, a final research hypothesis is that cognitive empathy may serve to buffer links between affective empathy and internalizing symptoms.

The Current Study

In the current study, I aim to understand how affective and cognitive empathy relate to internalizing symptoms during adolescence and how this relation may be impacted by the context of the COVID-19 pandemic. Data from an ongoing longitudinal study, collected from adolescents before and during the initial months of the COVID-19 pandemic, will be analyzed to test five study hypotheses (see Figure 1). First, affective empathy is expected to positively relate to internalizing symptoms and cognitive empathy to negatively relate to such symptoms before

pandemic onset (hypothesis 1). Next, the relation between affective empathy and internalizing symptoms is expected to be stronger during the COVID-19 pandemic than before pandemic onset (hypothesis 2), and pre-COVID-19 affective empathy is expected to positively predict internalizing symptoms during COVID-19 (hypothesis 3). In addition, adolescents' perceived levels of COVID-19-related stress are expected to moderate the relation between affective empathy and internalizing symptoms during the COVID-19 pandemic, such that higher perceived stress will be linked to a stronger association between affective empathy and internalizing symptoms (hypothesis 4). Finally, cognitive empathy is predicted to buffer associations between affective empathy and internalizing symptoms in hypotheses 1 and 3, with increased cognitive empathy reducing the strength of the association between affective empathy and internalizing symptoms both cross sectionally (hypothesis 1) and longitudinally (hypothesis 3).

Method

Data for this study were drawn from the Raising Grateful Children (RGC) study, a longitudinal study of parent-child dyads recruited in the southeastern United States in 2013 and 2014 (Hussong et al., 2018). The RGC study has nine waves of collected data to date, two of which are included in the current analyses, with the goal of understanding conceptualizations and displays of gratitude in young children, as well as parents' socialization behaviors related to gratitude.

Participants

The current sample is comprised of 96 of the original 110 youth in the RGC study, who had data for at least one of the two waves of data collection included in the current study. Missing data were present such that there were 76 cases with data at waves four and nine, 10

cases with data at wave four only, six cases with data at wave nine only, two cases missing only child-reported data at wave nine, one case missing only parent-reported data at wave four, and one case missing only parent-reported data at wave nine, thus necessitating missing data analysis. Children in this study were identified by their parents as 52.1% female and were aged 8-13 at wave four (M=10.8) and 11-16 at wave nine (M=13.6). Parent participants, who could endorse multiple racial/ethnic identities, identified as 80.2% European American, 9.4% Asian or Asian American, 4.2% Black or African American, 4.2% Multiracial, 1% Alaska Native or American Indian, and 1% West Asian or Middle Eastern. Parents also reported on the highest level of education attained in the household, with 64.7% having completed a graduate or professional degree, 21.9% having completed college, 8.3% having completed some graduate or professional schooling, 3.1% having completed some college, 1% having completed technical or vocational schooling, and 1% having graduated high school or attained a GED.

Procedures

Recruitment procedures for the RGC study included emails sent to faculty, students, and staff at an affiliated university, distribution of study flyers in first- to third-grade classrooms at public and private schools, and community postings. To be included, parent-child dyads needed to include one parent and one child aged 6-9, who were both proficient in English. For wave four of the RGC study, which acts as T1 in the current study, parents and children completed lab-based surveys in separate rooms after providing consent and assent. Lab visits lasted approximately one hour and 45 minutes. This was followed either immediately or after one month (in a waitlist control design) by an online gratitude socialization parent-training program (completed by all but seven of the dyads in the current study's sample), with related assessments in the following months. Wave nine of the RGC study, which acts as T2 in the current study,

occurred in May-July 1, 2020, when parents and children completed 45-minute online surveys. All research activities were approved by the university's institutional review board.

Measures

Demographic and Control Variables. Parents reported their child's gender and their own race/ethnicity at wave one of the RGC study (or upon entry to the study, for dyads entering later than wave one). Because five of the parent-child dyads did not complete the parent training intervention between T1 and T2, program participation is tested as a potential covariate in the current analyses. Additionally, children's age and gender were assessed as potential control variables, based on research identifying developmental progression of cognitive empathic skills (Eisenberg, 1986) and gender differences in levels of empathic responding (Ge et al., 1994).

Affective and Cognitive Empathy. Two subscales from the Interpersonal Reactivity Index (Davis, 1980) assessed adolescent self-reported affective empathy (i.e., empathic concern) at T1 (M=3.85; SD=0.61) and T2 (M=3.93; SD=0.71) and cognitive empathy (i.e., perspective taking) at T1 (M=3.54; SD=0.77). Participants indicated the extent to which they felt each item described them using a scale ranging from 1 (does not describe me well) to 5 (describes me well), with five items reverse scored. Seven items assessed empathic concern and captured concepts such as feelings of concern for others, and seven assessed perspective taking, capturing concepts such as trying to see things from another's point of view. Averages of items for each subscale served as scores for affective and cognitive empathy, with higher scores representing higher levels of each construct. Parents also reported on their child's affective (M=4.1; SD=0.76) and cognitive (M=3.02; SD=0.75) empathy at T1 and affective (M=0.64; SD=0.43) empathy at T2, indicating the extent to which the aforementioned items described their child. In the current study, internal reliability for adolescent self-reports of empathic concern was poor at T1

(Cronbach's α=.49) and acceptable at T2 (Cronbach's α=.76), while internal reliability for adolescent self-reports of perspective taking was moderate at T1 (Cronbach's α=.64). Internal reliability was strong for parent-reported affective empathy at T1 (Cronbach's α=.85) and T2 (Cronbach's α=.83) and cognitive empathy at T1 (Cronbach's α=.80).

Internalizing Symptoms. Five items from the Youth Pediatric Symptom Self-Report Checklist (Jellinek et al., 1988) assessed youth's internalizing symptoms at T1 and T2. Participants indicated how often in the last month they had exhibited specific behaviors or emotions, with responses ranging from 0 (never) to 2 (often). The five items comprising the internalizing symptoms subscale assessed sadness, unhappiness, hopelessness, worry, feeling down, and feeling like one was having less fun. Scores for adolescents' self-reported internalizing symptoms were calculated by averaging scores on the five items at T1 (M=0.51; SD=0.42; Cronbach's α=.76) and T2 (M=0.80; SD=0.54; Cronbach's α=.83). Parents also reported on their child's internalizing symptoms, using the five-item internalizing subscale of the Pediatric Symptom Checklist (Jellinek et al., 1988). Parents indicated how often in the last month their child had exhibited the same emotions or behaviors, with responses ranging from 0 (never) to 2 (often). Items were averaged at T1 (M=0.41; SD=0.36; Cronbach's α=.75) and T2 (M=0.64; SD=0.43; Cronbach's α=.79) to create parent-report adolescent internalizing scores. The Pediatric Symptoms Checklist has shown strong internal reliability (Cronbach's α=.89) and test-retest reliability (.86; Jellinek et al., 1988).

Perceived COVID-19 Related Stress. Eleven items derived from the Responses to Stress Questionnaire-COVID-19, an adaptation of the original Responses to Stress Questionnaire for COVID-19 (Compas, 2020; Connor-Smith et al., 2020), in combination with 3 original items written for the RGC study assessed adolescents' experiences of negative stressors related to the

COVID-19 pandemic at T2. Participants were asked to indicate whether or not they experienced any of 14 negative COVID-related life events, with items assessing topics like school stress (difficulty completing online schoolwork and inability to complete schoolwork requirements), financial difficulties (money problems dur to COVID-19), illness concerns (uncertainty about self or others getting COVID-19), disruption of plans (having to change or reschedule important plans due to COVID-19), and isolation (inability to spend time with friends or participate in social activities). Participants then rated their perception of the event, with ratings ranging from -4 (extremely bad) to 0 (neither good nor bad) to 4 (extremely good). Scores were calculated by averaging ratings across the 14 items and then multiplying the averages by -1 so that higher scores reflected higher perceived stress (M=1.12; SD=0.63; Cronbach's α=.77).

Results

Preliminary Analyses

Internalizing symptoms by child- and parent-report at both T1 and T2 were checked for normality and, with skew values of less than two and kurtosis estimates of less than seven, were considered to be normally distributed (Byrne, 2010). Means, standard deviations, and estimates of internal reliability for study variables are presented in Table 1. Overall, elevated adolescent internalizing symptoms (using a cutoff score of 28; Jellinek et al., 1988) were indicated by 17.8% of youth at T1 and 46.9% of youth at T2, and by 7.9% of parents at T1 and 19.1% of parents at T2. Gender differences in study predictor and outcome variables were assessed using t-tests (Table 2), and were found such that girls scored higher than boys on child-reported internalizing symptoms at T1 ($t(88)=2.65$; $p<.01$) and T2 ($t(82)=2.69$; $p<.01$), child-reported COVID-19-related stress at T2 ($t(82)=2.52$; $p<.05$), and parent-reported affective empathy at T2 ($t(82)=2.28$; $p<.05$). Girls showed marginally higher scores on parent-reported cognitive

(t(87)=1.73; p=.09) and affective (t(87)=1.89; p=.06) empathy at T1 and child-reported affective empathy at T2 (t(82)=1.85; p=.07). No gender differences were found for child-reported affective or cognitive empathy at T1 or parent-reported internalizing symptoms at either T1 or T2. Correlations among study variables are presented in Table 3.

Additionally, t-tests were conducted to determine whether there were systematic differences in age and internalizing symptoms by child- and parent-report at T1 and T2 between participants who were missing data and those who were not (Table 4); no significant differences were found between these groups, and thus multiple imputation was performed to address missing data. Analyses were conducted using Proc MI and MI Analyze in SAS 9.4 to create and integrate across 100 imputed datasets (Little & Rubin, 2002; SAS, 2013).

Four models regressing adolescent- and parent-reports of adolescents' internalizing symptoms at both T1 and T2 on child's age, gender, and participation in the RGC intervention were estimated to assess for significant covariates. Gender was the only variable that significantly predicted internalizing symptoms by adolescent report, both at T1 (b=-0.23, t=-2.64, p<.01) and T2 (b=-0.26, t=-2.25, p<.05), such that girls had higher rates than boys. No variables significantly predicted parent-reports of adolescents' internalizing symptoms. Thus, gender was retained as a control variable in models with adolescent-reported internalizing symptoms included as an outcome, and no control variables were included in models with parent-reported internalizing symptoms included as an outcome. All hypotheses were tested using ordinary least squares multiple regression models. In order to control for shared reported variance, cross-reporter effects were examined with 6 additional models using parent-reports of adolescents' internalizing symptoms augmenting primary analyses that used adolescents' self-reported internalizing symptoms as outcomes.

G*power was used to conduct a power analysis on the most complex model tested in the study—namely, that used to test the second part of the fifth hypothesis, which posits that T1 affective empathy will predict lower T2 internalizing symptoms when paired with higher levels of T1 cognitive empathy. The power analysis revealed that with a sample size of 96 and a significant threshold of α=.05, the model was adequately powered (β=.80) to detect a small effect size (f^2=0.07), suggesting that this model was adequately powered to correctly reject the null hypothesis. As this was the most complex of the models in the study, adequate power was also indicated for all other models proposed.

Hypothesis 1: Pre-COVID-19 Empathy and Internalizing Symptoms

To test whether affective empathy was positively related to and cognitive empathy was negatively related to internalizing symptoms at T1, an ordinary least squares regression was conducted, regressing T1 internalizing symptoms on T1 levels of cognitive and affective empathy while controlling for adolescents' gender (Table 5). Results suggested that neither affective (b=-.04; t=-.55; p>.05) nor cognitive (b=.05; t=.84; p>.05) empathy was significantly related to adolescents' report of internalizing symptoms at T1. A sensitivity analysis regressing T1 parent reports of adolescents' internalizing symptoms on T1 affective and cognitive empathy maintained these null results as parent reports of adolescents' internalizing symptoms at T1 were not significantly predicted by adolescents' affective (b=-.02; t=-.24; p>.05) or cognitive (b=.09; t=1.62; p>.05) empathy.

Hypothesis 2: Comparing the Affective Empathy-Internalizing Symptoms Link Before and During COVID-19

To test whether the association between affective empathy and internalizing symptoms was stronger during COVID-19 than before its onset, two ordinary least squares regressions were conducted, regressing T2 internalizing symptoms on T2 levels of affective empathy and T1

internalizing symptoms on T1 levels of affective empathy, while controlling for adolescents' gender in both models (Table 6). Results indicated that affective empathy was not significantly related to internalizing symptoms at either T1 (b=-.02; t=-.22; p>.05) or T2 (b=.01; t=.11; p>.05). Following recommendations by Paternoster et al. (1998), a Z-score was then calculated to test for a significant difference between affective empathy-internalizing symptom associations at T1 and T2. No significant difference was detected (Z=.22), suggesting that adolescents' affective empathy was no more strongly related to their self-reported internalizing symptoms at T2, during COVID-19, than at T1, before COVID-19. Sensitivity analyses replicated these results, showing no significant links between affective empathy and parent-reported internalizing symptoms at T1 (b=.03; t=.45; p>.05) or T2 (b=.001; t=.01; p>.05), and no significant difference between regression coefficients for affective empathy at T1 and T2 (Z=-.30).

Hypothesis 3: Pre-COVID-19 Affective Empathy Predicting During COVID-19 Internalizing Symptoms

To test whether pre-COVID levels of affective empathy could positively predict internalizing symptoms during COVID-19, an ordinary least squares regression was estimated to predict residualized change in internalizing symptoms by regressing T2 internalizing symptoms on T1 affective empathy, while controlling for T1 internalizing symptoms and cognitive empathy as well as adolescents' gender (Table 7). This approach allows for interpretation of the result as a change score when those in the sample represent a single population (Castro-Schilo & Grimm, 2018). Adolescents' affective empathy at T1 did not significantly predict internalizing symptoms at T2 (b=.03; t=.31; p>.05), after controlling for T1 internalizing symptoms, T1 cognitive empathy, and gender. A sensitivity analysis showed that adolescents' affective empathy also did not significantly predict parent-reports of their internalizing symptoms at T2, after controlling for

parent-reports of internalizing symptoms at T1 and adolescents' self-reported cognitive empathy at T1 (b=.001; t=.01; p>.05).

Hypothesis 4: COVID-19-Related Stress as a Moderator

To test whether adolescents' perceived COVID-19-related stress moderated the relation between affective empathy and internalizing symptoms during the COVID-19 pandemic, an ordinary least squares regression was conducted, building upon the model in hypothesis 2 (which regressed T2 internalizing symptoms on T2 affective empathy) by adding an interaction term between T2 affective empathy and adolescents' perceived levels of COVID-19 stress experienced at T2 (Table 8). The model revealed that adolescents' COVID-19-related stress did not significantly moderate the relation between their levels of affective empathy and internalizing symptoms at T2 (b=-.05; t=-.41; p>.05). There was a main effect of COVID-19-related stress (b=.38; t=4.24; p<.001), such that increased COVID-19-related stress endorsement was linked to increased self-reported internalizing symptoms. These findings were replicated in a sensitivity analysis, where adolescents' COVID-19-related stress did not moderate the relation between their self-reported affective empathy and parent-reported internalizing symptoms at T2 (b=.04; t=.37; p>.05), and a positive main effect of adolescents' COVID-19-related stress on parent-reported internalizing symptoms at T2 was found (b=.16; t=2.01; p<.05).

Hypothesis 5: Cognitive Empathy as a Buffer

To test whether cognitive empathy acted to buffer the relation between affective empathy and internalizing symptoms in hypotheses 1 and 3, such that higher levels of cognitive empathy would weaken the association between affective empathy and internalizing symptoms in hypothesis 1 and decrease the levels of T2 internalizing symptoms predicted by T1 affective empathy in hypothesis 3, ordinary least squares regressions were conducted, building upon the

models outlined in hypotheses 1 and 3 by adding an interaction term multiplying T1 affective and cognitive empathy (Table 9). No hypothesized buffering effects of cognitive empathy on the relation between affective empathy and internalizing symptoms were found. Cognitive empathy did not moderate the relation between adolescents' self-reported affective empathy and internalizing symptoms at T1 ($b=-.07$; $t=-.85$; $p>.05$), nor did it moderate the association between adolescents' self-reported affective empathy at T1 and their self-reported internalizing symptoms at T2 ($b=.05$; $t=.43$; $p>.05$). Sensitivity analyses revealed that cognitive empathy also did not moderate the link between adolescents' self-reported affective empathy and parent-reported internalizing symptoms at T1 ($b=.07$; $t=.88$; $p>.05$) or the link between adolescents' self-reported affective empathy at T1 and parent-reported internalizing symptoms at T2 ($b=.07$; $t=.87$; $p>.05$).

Discussion

Contrary to study hypotheses, affective empathy was not linked to co-occurring internalizing symptoms either before or during the COVID-19 pandemic, did not predict future levels of internalizing symptoms experienced during the COVID-19 pandemic, and did not interact with either COVID-19-related stress or cognitive empathy to predict internalizing symptoms. Cognitive empathy also did not relate to internalizing symptoms. While the predicted relations were not confirmed, the current findings may inform approaches to studying affective and cognitive empathy in youth.

As noted in prior studies, cognitive empathy is a more advanced skill that requires additional cognitive development as compared to affective empathy (Hoffman, 1982; 2000). In the current study, cognitive empathy was assessed when youth were aged 8-13. Current null findings may have occurred because the current sample was too young to accurately report cognitive empathy and demonstrate a relation to internalizing symptoms for one of multiple

reasons. It could be that 8-13-year-olds are too young to have developed the capacities for cognitive empathy, or, it could be that cognitive empathy is present in a more rudimentary form in this age group, and would thus need to be measured in a different way from older adolescents and adults. This could also explain the limited internal reliability of the cognitive empathy measure in the current study. If a more developmentally appropriate form of measurement is needed, then the IRI, a measure developed with samples of undergraduate students (Davis, 1980), may not have enabled the current study to detect relations between cognitive empathy and other aspects of socioemotional functioning. Importantly, many empathy measures developed for children lack a distinctly cognitive component and assess a more general empathic disposition (Eisenberg et al., 1991; Eisenberg et al., 1996), underlining the difficulty in adequately assessing perspective taking in this age group.

Further, the current study did not replicate the well-documented relation between affective empathy and internalizing symptoms. Two explanations for this null effect, related to the age of the sample and contextual considerations, bear further investigation. First, the age of the current sample at T1 (8-13) is younger than most of the samples included in the literature that report positive associations between affective empathy and internalizing symptoms. This may suggest that the link between affective empathy and internalizing symptoms changes from early to later adolescence. Variations in the onset of adolescence (as marked by puberty and associated physiological changes) may mean that our sample, especially at T1 (aged 8-13) but also at T2 (aged 12-16), was more heavily saturated with youths who would better be described as pre-adolescent. Findings linking affective empathy to internalizing symptoms in adolescents may not extend to younger, more pre-adolescent samples, and thus not be replicated in the current study. More, the age of the current sample at T1, paired with the low internal reliability of the empathic

concern subscale at T1 *and not T2* could indicate that 8-12 is too young an age group for IRI administration. The same subscale showed satisfactory internal reliability when youths were aged 12-16, suggesting that a more developmentally appropriate measure of affective empathy may have particularly indicated at T1, and that use of the IRI could have hindered detection of possible links between participants' affective empathy and internalizing symptoms.

Second, there may be contextual factors impacting the current sample that would explain the lack of association between empathy and internalizing symptoms. For example, families in the current sample were recruited for participation in a research study on parenting and gratitude. It may be that parents and families who were introduced to and interested in such a study are qualitatively different from those who were not, resulting in a self-selection bias. These families may place more emphasis on positive emotion and/or parenting skills, and thus may experience additional protective factors that may affect the link between empathy and internalizing symptoms. Another potentially influential factor may be the relatively high socioeconomic status endorsed by the families in the current sample; those from higher socioeconomic households may be better insulated against the link between affective empathy and internalizing symptoms.

Socioeconomic status may also contribute to the current null findings related to COVID-19 stress as a moderator between affective empathy and internalizing symptoms, in that those from higher socioeconomic households may experiences fewer or qualitatively different COVID-19-related stressors, or may experience similar stressors but be equipped with differential coping abilities and resources which may mitigate their impact. In fact, researchers have noted the importance of considering heterogeneity of experiences related to COVID-19, citing that those with the most disadvantage prior to COVID-19 experienced the most negative effects during it (Branje & Morris, 2021).

It could also be that the allostatic load mechanism outlined in this paper works differently than hypothesized. The impact of stress may not be linear—stress may display a negative impact only when experienced to a certain degree. This can be viewed through the window of tolerance, a framework often used to describe humans' responses to stress or trauma (Siegel, 1999). The window of tolerance describes a range (or window) within which individuals' experiences allow for normative human functioning. It is experiences which fall outside of this window—stressors which may be complex, ongoing, or which otherwise supersede one's capacity to cope—that lead to adverse reactions and outcomes. In other words, negative consequences may not be observed until a certain amount or intensity of stressful or traumatic experiences are endorsed, which would shift one to falling outside of their window of tolerance.

It had been suggested that experiences of trauma may 'shrink' one's window of tolerance (Corrigan et al., 2011), thereby increasing the likelihood that one's future experiences of stress and/or trauma will fall outside of one's window. Applying this framework, it could be that our population of adolescents from a relatively high socioeconomic class came equipped with larger windows of tolerance, and thus showed fewer negative outcomes that may be associated with these experiences. If this is true, then investigating empathy, stress, and internalizing symptoms in populations with higher endorsement of COVID-19-related stressors may uncover effects of stress which are only observable past some thresholds. Of course, it may also be that allostatic load is not indeed triggered by distress associated with higher levels of affective empathy, and that some other mechanism is acting on these variables.

Conclusions

The current study did not identify links between adolescent affective and cognitive empathy and internalizing symptoms or moderating effects of cognitive empathy or COVID-19-

related stress on the relation between affective empathy and internalizing symptoms. It did, however, offer an opportunity to differentially consider two distinct forms of empathy in a context rife with change—both the change accompanying adolescent development and the changes brought on by the COVID-19 pandemic. Strengths of the current study include the investigation of multiple time points, allowing for observations of change over time. Moreover, the differentiation between cognitive and affective empathy contributes to a more nuanced understanding of adolescents' experiences of empathy. The inclusion of both adolescent- and parent- report of study variables controlled for the possibility of biased findings due to shared reporter effects.

In addition to these strengths, the current study also has limitations. First, the sample size of 102 youth limits the study's statistical power, although a power analysis did reveal that the study was sufficiently powered at (beta=.80) to detect an effect size of 0.07 in a regression with 6 predictors. Further, the sample maintains a certain degree of societal privilege, and thus offers a limited view into the link between adolescent empathy, internalizing symptoms, and COVID-19-related stress. The sample also consists of parents and families who opted to participate in a study on gratitude and parenting, further limiting the generalizability of the current findings. Finally, internal reliability estimates for child-reports of empathy using the Interpersonal Reactivity Index (IRI) at T1 were low for the empathic concern subscale (α=.50) and only moderate for the perspective taking subscale (α=.64), further supporting the possibility that the measure may not have been developmentally appropriate for the participants when they were aged 8-13.

Future work would benefit from including a more representative sample, particularly with respect to socioeconomic status, to better capture a range of stressful experiences related to

COVID-19. It may also prove fruitful to investigate affective and cognitive empathy in two different ways. First, studies may use the IRI but in a sample of older adolescents, as this is the age range with which the scale was validated (Davis, 1980) and for whom the scales may provide the clearest insight. Second, studies may focus on a sample of young adolescents, but with empathy measures more developmentally tailored to children and early adolescents. Other avenues for research include the presentation of cognitive empathy in younger and pre-adolescents and links between empathy and internalizing symptoms in childhood and early adolescence.

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Table 1

Means, Standard Deviations, and Internal Reliability Estimates for Continuous Variables

	Variable	T1			T2		
		M	SD	α	M	SD	α
	Age	10.65	1.17		--	--	--
Child Report	Affective Empathy	3.85	0.62	0.49	3.93	0.71	0.76
	Cognitive Empathy	3.54	0.77	0.64	--	--	--
	Internalizing Symptoms	0.51	0.42	0.76	0.80	0.54	0.83
	COVID-19-Related Stress	--	--	--	1.12	0.63	0.77
Parent Report	Affective Empathy	4.10	0.76	0.85	4.02	0.77	0.83
	Cognitive Empathy	3.02	0.75	0.80	--	--	--
	Internalizing Symptoms	0.41	0.36	0.75	0.64	0.43	0.79

Table 2

T-Tests: Comparing Study Predictor and Outcome Variables by Biological Sex

		T1			T2		
	Variable	M_{female}	M_{male}	t-value	M_{female}	M_{male}	t-value
Child Report	Affective Empathy	3.92	3.78	1.02	4.06	3.78	1.85
	Cognitive Empathy	3.50	3.58	-0.46	--	--	--
	Internalizing Symptoms	0.62	0.39	2.65**	0.94	0.64	2.69**
	COVID-19-Related Stress	--	--	--	1.28	0.94	2.52*
Parent Report	Affective Empathy	4.24	3.94	1.89	4.19	3.81	2.28*
	Cognitive Empathy	3.15	2.88	1.73	--	--	--
	Internalizing Symptoms	0.41	0.40	0.10	0.68	0.58	1.10

Note. *p<.05; **p<.01; ***p<.001

Table 3

Correlations Between Predictor and Outcome Variables, by Child- and Parent-Report, at T1 and T2

			T1						T2				
			Child Report			Parent Report			Child Report			Parent Report	
			Affective	Cognitive	Internalizing	Affective	Cognitive	Internalizing	Affective	Internalizing	COVID-19 Stress	Affective	Internalizing
T1	Child Report	Affective	1										
		Cognitive	0.419***	1									
		Internalizing	0.007	0.055	1								
	Parent Report	Affective	0.242*	0.236*	0.247*	1							
		Cognitive	0.152	-0.021	0.022	0.568***	1						
		Internalizing	0.055	0.186	0.239*	0.102	-0.051	1					
T2	Child Report	Affective	0.382***	0.358**	0.134	0.170	0.074	-0.017	1				
		Internalizing	0.005	-0.107	0.425***	0.140	0.175	0.188	0.095	1			
		COVID-19 Stress	0.106	-0.107	0.254*	0.173	0.126	0.073	0.192	0.485***	1		
	Parent Report	Affective	0.309**	0.201	0.142	0.666***	0.476***	-0.046	0.276*	-0.066	0.096	1	
		Internalizing	0.058	0.148	0.226*	0.086	0.060	0.552***	0.015	0.441***	0.228*	-0.193	1

Note. *p<.05; **p<.01; ***p<.001

Table 4

T-Tests: Comparing Age and Outcome Variables by Missingness of Data

		T1			T2		
	Variable	$M_{complete}$	$M_{missing}$	t-value	$M_{complete}$	$M_{missing}$	t-value
	Age	10.61	10.87	-0.79	--	--	--
Child Report	Internalizing Symptoms	0.50	0.59	-0.71	0.81	0.73	0.42
Parent Report	Internalizing Symptoms	0.40	0.46	-0.56	0.62	0.76	-0.95

Table 5

Regression Results: Hypothesis 1

		b	t	p
Regressed on T1 CR Internalizing Symptoms	Intercept	0.11	1.85	0.06
	Gender	-0.23	-2.61	0.009**
	T1 CR Affective	-0.04	-0.55	0.58
	T1 CR Cognitive	-0.05	0.84	0.40
Sensitivity Analysis Regressed on T1 PR Internalizing Symptoms	Intercept	0.00	-0.01	0.99
	T1 CR Affective	-0.02	-0.24	0.81
	T1 CR Cognitive	0.09	1.62	0.11

Note. CR= child-report; PR=parent report; *p<.05; **p<.01; ***p<.001

Table 6

Regression Results: Hypothesis 2

		b	t	p
Regressed on T1 CR Internalizing Symptoms	Intercept	0.11	1.79	0.07
	Gender	-0.22	-2.54	0.01*
	T1 CR Affective	-0.02	-0.22	0.83
Regressed on T2 CR Internalizing Symptoms	Intercept	0.92	11.55	<0.001***
	Gender	-0.26	-2.19	0.03*
	T2 CR Affective	0.01	0.11	0.91
Sensitivity Analysis Regressed on T1 PR Internalizing Symptoms	Intercept	0.00	0.00	1.00
	T1 PR Affective	0.03	0.45	0.65
Sensitivity Analysis Regressed on T2 PR Internalizing Symptoms	Intercept	0.64	13.55	<0.001***
	T2 PR Affective	0.00	0.01	0.99

Note. CR= child-report; PR=parent report; *p<.05; **p<.01; ***p<.001

Table 7

Regression Results: Hypothesis 3

		b	t	p
Regressed on T2 CR Internalizing Symptoms	Intercept	0.86	11.36	<0.001***
	Gender	-0.15	-1.23	0.22
	T1 CR Cognitive	-0.11	-1.45	0.15
	T1 CR Affective	0.03	0.31	0.76
	T1 CR Internalizing	0.48	3.45	<0.001***
Sensitivity Analysis Regressed on T2 PR Internalizing Symptoms	Intercept	0.64	15.17	<0.001***
	T1 CR Cognitive	0.00	0.04	0.97
	T1 CR Affective	0.00	0.01	0.99
	T1 PR Internalizing	0.67	5.77	<0.001***

Note. CR= child-report; PR=parent report; *p<.05; **p<.01; ***p<.001

Table 8

Regression Results: Hypothesis 4

		b	t	p
Regressed on T2 CR Internalizing Symptoms	Intercept	0.87	11.82	<0.001***
	Gender	-0.16	-1.41	0.16
	T2 CR Affective	-0.03	-0.39	0.70
	T2 CR COVID-19 Stress	0.38	4.24	<0.001***
	T2 CR Affective*COVID-19 Stress	-0.53	-0.41	0.68
Sensitivity Analysis Regressed on T2 PR Internalizing Symptoms	Intercept	0.64	13.39	<0.001***
	T2 CR Affective	-0.03	-0.41	0.68
	T2 CR COVID-19 Stress	0.16	2.01	0.04*
	T2 CR Affective*COVID-19 Stress	0.04	0.37	0.71

Note. CR= child-report; PR=parent report; *p<.05; **p<.01; ***p<.001

Table 9

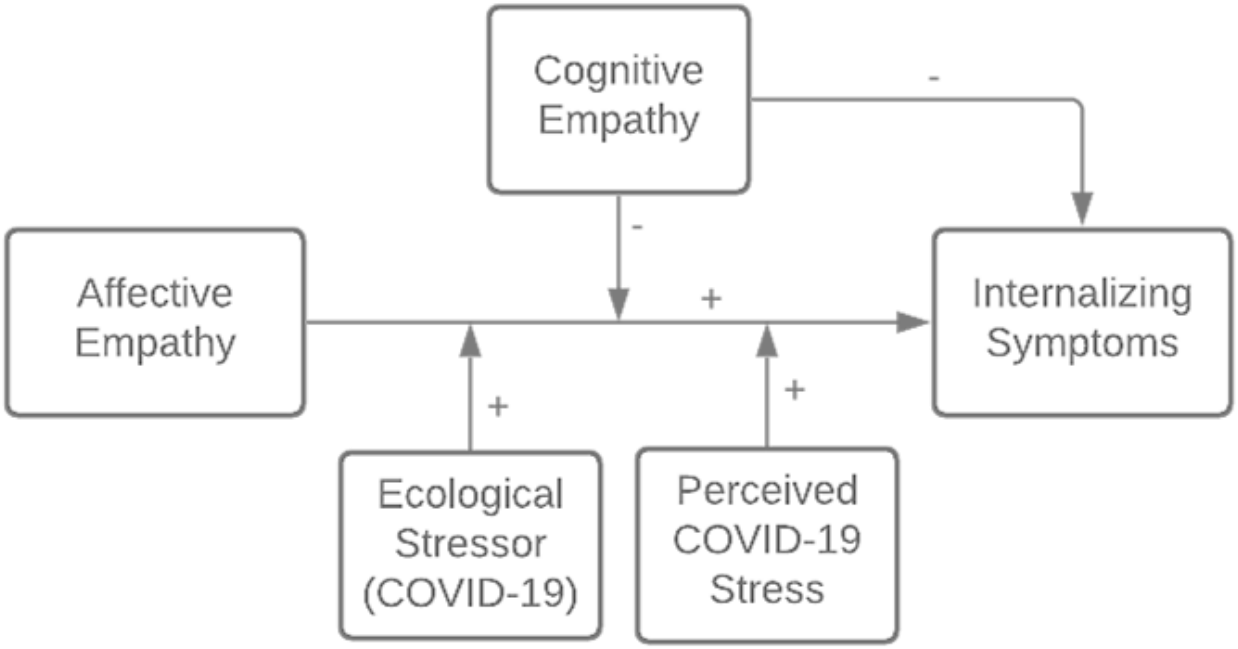
Regression Results: Hypothesis 5

		b	t	p
Regressed on T1 CR Internalizing Symptoms	Intercept	0.13	2.02	0.04
	Gender	-0.23	-2.67	0.008**
	T1 CR Affective	-0.05	-0.58	0.56
	T1 CR Cognitive	0.06	0.92	0.36
	T1 CR Affective*Cognitive	-0.07	-0.85	0.39
Regressed on T2 CR Internalizing Symptoms	Intercept	0.85	10.72	<0.001***
	Gender	-0.14	-1.18	0.24
	T1 CR Affective	0.03	0.32	0.75
	T1 CR Cognitive	-0.12	-1.48	0.14
	T1 CR Internalizing	0.49	3.46	<0.001***
	T1 CR Affective*Cognitive	0.47	0.43	0.66
Sensitivity Analysis Regressed on T1 PR Internalizing Symptoms	Intercept	-0.01	-0.32	0.75
	T1 CR Affective	-0.02	-0.22	0.83
	T1 CR Cognitive	0.09	1.53	0.13
	T1 CR Affective*Cognitive	0.07	0.88	0.38
Sensitivity Analysis Regressed on T1 PR Internalizing Symptoms	Intercept	0.63	14.15	<0.001***
	T1 CR Affective	0.00	0.03	0.97
	T1 CR Cognitive	0.00	-0.03	0.97
	T1 PR Internalizing	0.66	5.64	<0.001***
	T1 CR Affective*Cognitive	0.07	0.87	0.38

Note. CR= child-report; PR=parent report; *p<.05; **p<.01; ***p<.001

Figure 1

A Conceptual Model of Links Between Empathy, Internalizing Symptoms, and Stress



Note. Hypothesized positive (+) and negative (-) links are outlined above.