

Emotion

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Childhood Emotion Dysregulation Mediates the Relationship Between Preschool Emotion Labeling and Adolescent Depressive Symptoms

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Deficits in emotion processing (e.g., emotion labeling and regulation) are widely implicated in depression risk. While prior literature documents these deficits in concurrence with depression, more research is needed to investigate emotion processing pathways of depression risk across development. The purpose of this study was to investigate if emotion processes (i.e., emotion labeling and emotion regulation/dysregulation) in early and middle childhood predict adolescent depressive symptom severity in a prospective sample. Data were analyzed from a longitudinal study of diverse preschoolers oversampled for depressive symptoms using measures of preschool emotion labeling of faces (i.e., Facial Affect Comprehension Evaluation), middle childhood emotion regulation and dysregulation (i.e., emotion regulation checklist), and adolescent depressive symptoms (i.e., PAPA, CAPA, and KSADS-PL diagnostic interviews). Multilevel models indicated that preschoolers with depression had similar development of emotion labeling in early childhood as peers. Mediation analyses revealed that deficits in preschool-aged anger and surprise labeling ability indirectly predicted higher adolescent depressive symptom severity through increased middle childhood emotion lability/negativity, not decreased emotion regulation. Adolescent depression may be predicted by an emotion processing pathway that spans from early childhood to adolescence, and findings may generalize to high risk for depression youth samples. Specifically, poor emotion labeling in early childhood may lead to increased childhood emotion lability/negativity, which increases the risk for adolescent depressive symptom severity. Findings may help identify specific emotion processing relations in childhood that increase the risk for depression and inform intervention aimed at improving preschoolers' anger and surprise labeling.


Keywords: depression, emotion regulation, emotion labeling, early childhood, adolescence

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Developmental psychology literature demonstrates that normative emotion development is associated with a myriad of adaptive psychosocial outcomes in youth (Calkins & Marcovitch, 2010; Cole et al., 1994). Conversely, atypical emotion development has been repeatedly linked to a range of child and adolescent psychiatric disorders (Bourke et al., 2010; Bradley, 2003; Casey, 1996; Guyer et al., 2007). Given impaired emotion processing is a broad, transdiagnostic mechanism of developmental psychopathology, it is important to investigate specific relationships between emotion

processes and psychiatric disorders. Indeed, prior developmental psychopathology research provides extensive evidence that emotional processes, including emotion recognition and emotion regulation, independently precipitate and characterize depression (Cole et al., 2008; Luby & Belden, 2006), a leading cause of disability in youth worldwide (WHO, 2021). However, further research is needed to investigate if relations between specific emotion processes produce a developmental pathway of depression risk.

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Within the depression literature, prior literature finds that emotion processes implicated in depression include both emotion recognition, the ability to identify others' emotion expressions (Keltner et al., 2019; rather than identifying one's own emotions, as indexed by emotion awareness), and emotion regulation, the ability to manage one's emotional experiences (McRae & Gross, 2020). Before conceptualizing an emotion processing pathway of depression risk, it is crucial to characterize the indices of emotion recognition and regulation implicated. One index of emotion recognition linked to depressive symptoms includes emotion labeling (Collin et al., 2013), the ability to match facial emotion expressions with emotion words. Emotion labeling is often measured according to response accuracy in matching emotion words to facial emotion expressions. These facial expressions may or may not vary in intensity. Like emotion recognition, indices of emotion regulation are implicated in depression. For example, studies suggest that depression is associated with the ability to effectively modulate emotion responses (Gonçalves et al., 2019; Schäfer et al., 2017). Prior literature also suggests that depression is linked to emotional reactivity and higher levels of negative affect intensity (Rydell et al., 2003; Siener & Kerns, 2012), indices of emotion dysregulation, a construct that is often conflated with emotion regulation in the literature.

Regardless of their independent associations with depression, emotion recognition and regulation may also relate to each other in ways that produce a pathway of depression risk. To conceptualize a pathway that implicates multiple emotion processes, it is important to investigate how such emotion processes are related. Accordingly, the constructionist theory of emotion suggests that emotion recognition is foundational to emotion regulation (Barrett et al., 2001). More specifically, this theory suggests that successful emotion recognition provides contextual information about one's current situation, which, in turn, helps a person identify and execute the appropriate emotion regulation strategies needed to enact context-specific goals (Barrett et al., 2001; Hoemann et al., 2019). Despite initial, empirical support for the constructionist theory of emotion suggesting that emotion recognition and related constructs (i.e., "emotion knowledge" and "emotion labeling") are positively correlated with emotion regulation in early childhood (Denham & Burton, 2003; Ornaghi et al., 2019) and adolescence (Belmonte-Darraz et al., 2021), little is known about the predictive nature of this relationship. Given longitudinal studies find that atypical emotional development is a robust risk factor for depression across the life course (Cole et al., 2008; Luby & Belden, 2006; Vogel et al., 2019), more research is warranted on specific emotion processing pathways of depression risk across development. Moreover, while initial research by Elsayed et al. (2021) found that preschoolers' emotion labeling skills did indeed predict emotion regulation in adolescence using the same study sample in this report, the link between emotion processing and psychopathology was not explored. To the authors' knowledge, no studies have assessed if and to what extent emotion recognition and emotion regulation relate to each other across development to predict depression. The current study, therefore, aims to investigate the prospective relationships between emotion labeling, emotion regulation, and depression across childhood and adolescence.

Emotion Processing in Depressed Youth

In addition to a dearth of studies investigating prospective associations between emotion labeling, emotion regulation, and

depression, limited empirical literature investigates the developmental specificity of these relationships. To further characterize these relationships in youth, it is important to discuss what constitutes normative emotional development. Some studies find that emotion recognition of basic emotion expressions of happiness and sadness begins as early as infancy (Barrera & Maurer, 1981; Mondloch et al., 2003) and that children become proficient in labeling these basic emotion expressions around age 5 or 6 (Bruce et al., 2000). While other studies report more protracted development of emotion labeling across childhood (Durand et al., 2007; Stifter & Fox, 1986), there is general consensus that emotion labeling abilities for basic emotions improve across childhood. Like emotion recognition, emotion regulation skills are similarly found to improve as children age. Research finds that infants have limited intrinsic emotion regulation skills and rely on adults to soothe them or distract them from emotionally challenging contexts (Zeman et al., 2006). Prior research suggests that children then progress to displaying emotions consistent with situational demands as early as the preschool period (Fuchs & Thelen, 1988; Zeman & Shipman, 1996). Regardless of these early advances, emotion regulation abilities typically do not mature until middle childhood or early adolescence (Cole et al., 2008). Overall, given these age-related changes in emotion labeling and emotion regulation abilities, understanding the emotion processing pathways of depression necessitates a developmental context.

Importantly, further research is needed to investigate if emotion processing deficits in depression vary across development. According to prior literature, a meta-analysis found evidence of emotion labeling deficits (indexed by low response accuracy scores) in depressed adults across basic emotions, including anger, disgust, fear, happiness, and surprise, but not sadness (Dalili et al., 2015), yet a recent meta-analysis of emotion labeling in adolescence found that depressed youth identified low-intensity expressions of sad emotions more accurately than controls (Auerbach et al., 2015; Nyquist & Luebbe, 2020; Seymour et al., 2016). However, it remains unknown if these mixed findings are linked to developmental differences or various study design characteristics, including response modalities, stimuli type, emotion labeling paradigms, and individual differences in participants beyond age. Moreover, an initial body of literature implicates emotion labeling deficits in depressive symptoms as early as the preschool period, such that a relatively worse ability to label sad facial emotion expressions was associated with increased preschool depressive symptoms in the context of elevated adverse childhood experiences (Sudit et al., 2021), and worse emotion labeling (indexed by an average of labeling accuracy scores across all emotions) was correlated with greater internalizing symptoms in 3-year-old children (Kujawa et al., 2014). Indeed, some studies even fail to find a relationship between facial emotion labeling of emotions and depressive symptoms in childhood and early adolescence (Ciarrochi et al., 2008; Smoller & Brosgole, 1993). In addition to mixed findings on this topic abounding across age groups, prior depression literature also suggests that differences in emotion processing deficits exists within age groups. For instance, although some studies reported that depressed adults were less accurate than controls in emotion labeling of sadness (Gur et al., 1992; Mikhailova et al., 1996; Rubinow & Post, 1992) and happiness (Gur et al., 1992), other studies reported that response accuracy in depressed adults was better for subtle expressions of sadness (Gollan et al., 2010; Liu et al., 2012), and still others reported no specific accuracy deficits in emotion labeling (Gollan et al., 2008;

Leppänen et al., 2004). In children and adolescents, while one study reported greater response accuracy for low-intensity expressions of sadness and anger (Schepman et al., 2012), another study found reduced response accuracy in identifying emotion expressions of anger and greater intensity of emotion expression needed to identify sad faces (Joormann et al., 2010). Compared to emotion labeling, prior developmental literature finds more robust evidence of associations between depression and emotion labeling across response modalities. In other words, a large body of research consistently finds relationships between depression and emotion regulation deficits, indexed by both poor emotion regulation and increased lability, in early childhood (Luby et al., 2018), middle childhood (Siener & Kerns, 2012), and adolescence (Durbin & Shafir, 2008). Prospectively, a large literature demonstrates that lower levels of childhood emotion regulation predict depressive symptoms in adolescence and across the life course (Casey, 1996; McLaughlin et al., 2011; Zeman et al., 2002).

Developmental Pathway of Depression Risk

Taken together, there is a growing literature across age groups demonstrating that poor emotion labeling and emotion regulation are both linked to depression and evolve throughout development. Indeed, emerging research finds that emotion knowledge, an index of emotion recognition (other than emotion labeling), predicts both appropriate emotion displays observed during in-lab tasks and parent reported emotion regulation during the preschool period (Di Maggio et al., 2016; Hudson & Jacques, 2014). While additional studies find emotion labeling and emotion regulation are related in youth (Denham & Burton, 2003; Ornaghi et al., 2019), it is unknown if associated deficits are implicated in a pathway of depression risk. Initial literature in adults demonstrates that emotion suppression, an emotion regulation strategy, moderates the association between emotion labeling deficits for angry emotion expressions and depression (Aldinger et al., 2013). However, to the authors' knowledge, no studies explore the longitudinal associations between emotion labeling, emotion regulation, and depression from early childhood to adolescence. Given emotion processes begin crystallizing as early as infancy (Mondloch et al., 2003), it is crucial to explore a developmental pathway of depression risk beginning in early childhood. Specifically, targeting deficits in early development may be particularly effective in mitigating future depression risk. It is also important to investigate if early emotion processing deficits are linked to adolescent depression, as teens experience numerous biopsychosocial stressors such as puberty, social role changes, and multiple, successive transitions throughout a short period of time (Graber & Brooks-Gunn, 1996) when they do not yet possess more sophisticated and effective emotion regulation strategies, making them particularly vulnerable to negative outcomes related to emotion processing deficits.

The current study aimed to investigate a developmental pathway linking distinct childhood emotion processes to adolescent depression. Due to evidence, albeit mixed, that emotion labeling deficits for basic emotions (i.e., anger, happiness, sadness, surprise, disgust, fear; Dalili et al., 2015; Gur et al., 1992) and emotion regulation/dysregulation indices (i.e., emotion lability/negativity and regulation; Gonçalves et al., 2019; Siener & Kerns, 2012) independently predict, and potentially contribute to, depression, the current study aimed to elucidate the nature of these relationships. As such, we

tested whether emotion regulation in middle childhood acts as a mechanism (i.e., mediator) by which early childhood emotion labeling ability confers risk for adolescent depressive symptoms. Stated otherwise, we tested whether poor early childhood emotion labeling of basic emotions predicts increased adolescent depressive symptom severity through middle childhood emotion regulation deficits (i.e., low emotion regulation ability) or emotion dysregulation (i.e., high emotional lability). Due to more robust evidence of emotion labeling deficits in depression for anger, sadness, and happiness (Gur et al., 1992) as compared to other basic emotions (i.e., surprise, disgust, fear), and we also hypothesized that only the former indices would be implicated in the suggested mediation.

Method

Participants

Participants were from the Preschool Depression Study (PDS), an ongoing longitudinal study at Washington University School of Medicine in St. Louis that examines emotion development and preschool-onset depression. The PDS originally recruited 306 children aged 3.0–5.11 years oversampling children at baseline for preschool depressive symptoms using the parental report preschool feelings checklist (PFC; Luby et al., 1999). Subjects with PFC scores of 2 or greater were eligible for the study, and additionally subjects with a PFC score of 0 were recruited as a comparison group. Participants have subsequently been assessed approximately yearly for 10 behavioral assessments. Detailed recruitment methods for PDS have been reported elsewhere (Luby et al., 2009). The current study analyzed data from 258 PDS participants. This included participants who had data from at least one of the first three preschool assessments (Time 1 [T1], T2, and T3), when emotion labeling was assessed, with school-age (T5) data, when emotion regulation was assessed, and at least one longitudinal assessment beyond the T5 assessment (T5–T10), when depression was assessed. Emotion labeling, emotion regulation, and depression measures are described below. Participant ages at the preschool assessments were $M (SD)$: T1 = 4.45(0.79), T2 = 5.47(0.79), T3 = 6.44(0.78), at school-age: T5 = 9.68(0.96), and at the last assessment in late adolescence: T10 = 18.69(1.10). Participants were 46.1% ($n = 119$) female, 55.0% ($n = 142$) White, 32.2% ($n = 83$) Black, and 12.8% ($n = 33$) other, as identified by parent report, and the mean socioeconomic status, as measured by income-to-needs was: $M (SD) = 2.07(1.18)$, with scores below one indicating living below the poverty line.

Preschool Emotion Labeling: The Facial Affect Comprehension Evaluation (Mrakotsky, 2001; FACE)—emotion labeling task was designed for use in preschoolers and assessed children's ability to identify and verbally label seven different emotions (i.e., *sad*, *scared [fear]*, *happy*, *mad [anger]*, *surprised*, and *yucky [disgust]*) in color photographs of individual facial expression stimuli (Mrakotsky, 2001). Stimuli included different numbers of emotional faces between emotions, as well as across sessions such that: sad was assessed with seven (T1/T2) or eight items (T3), scared [fear] and happy were assessed with three (T1/T2) or five items (T3), anger with four (T1/T2) or five (T3) items, and surprised, yucky [disgust] with five (T1/T2/T3) items. After each of 27 (T1, T2) or 38 (T3) stimuli were presented, participants were directed to verbally respond to the open-ended question: "How does he/she feel?" Response options (i.e., the seven outlined emotions) were listed

verbally to participants prior to administration and were repeated after every six items. Children were instructed to verbally provide their response to the above-mentioned question; synonym labels for emotions were also considered valid responses. Facial stimuli consisted of facial expressions displayed by male and female children and adults. Stimuli consisted of mostly White faces (with Black faces interspersed) and were not matched to participant demographic characteristics. The PDS administered this task at T1, T2, and T3 assessment waves when participants were aged 3.0–7.11 years old. For the current study, scores were calculated for the correct number of faces labeled—individual emotion category. Correct labeling of individual emotions was summed independently for T1, T2, and T3 for the emotions mentioned above. Of note, shame was also assessed, but only at T3 and so was not included in current analyses as we used these data to create trajectories across all three time points. The first and second assessments used prorated sum scores (i.e., Prorated N happy = $(N \text{ happy}/3) \times 5$) to ensure the trajectory models were not influenced by number of trials at the final assessment.

Middle Childhood Emotion Regulation: The emotion regulation checklist (ERC; Shields & Cicchetti, 1997) is a 24-item parent-report questionnaire validated for assessing behavioral proxies of emotion regulation in youth aged 6–12 years. Items were rated on a 4-point Likert scale according to parents' perceived frequency (i.e., *never, sometimes, often, almost always*) of their child's behaviors and contributed to calculation of two subscales: lability/negativity and emotion regulation. Higher scores on the lability/negativity subscale (15 items) indicate higher emotion regulation, or emotion intensity, and presence of negative emotions ($\alpha = .81$) (8) (e.g., "exhibits wide mood swings"), while higher scores on the emotion regulation subscale (eight items) indicate higher levels of context appropriate emotion and self-regulation (e.g., "displays appropriate negative emotions in response to hostile, aggressive or intrusive acts by peers"; $\alpha = .76$).

Depressive Psychiatric Diagnoses and Symptoms: Trained staff conducted in-person diagnostic interviews with children and caregivers at each wave to assess diagnosis of major depressive disorder (MDD). At T1–T3, the Preschool-Age Psychiatric Assessment (PAPA), validated for administration to primary caregivers of children aged 2–6 years, was administered to caregivers (Egger et al., 2006). At T5–T8, the Child and Adolescent Psychiatric Assessment (CAPA), validated for administration to children and adolescents aged 9 to 17 years, was administered to children and caregivers (Angold & Costello, 2000). Of note, the CAPA is not administered to children until they are 9 years old; prior to that, parents complete the CAPA on behalf of their children (i.e., ages 7–8 years). Raters for the PAPA and CAPA were trained to reliability and 20% of tapes were reviewed by a master coder for reliability. Consultation with a senior child psychiatrist was used to resolve discrepancies (Luby et al., 2009). At T9 and T10, the Kiddie Schedule for Affective Disorders and Schizophrenia—Present and Lifetime Version (KSADS-PL) was administered to children and caregivers and is purportedly validated for administration to children aged 6–18 years (Kaufman et al., 1997). Although a skip-out method is frequently used in the KSADS if screen items are not endorsed, interviewers assessed all MDD symptoms, allowing for parallel symptom counts to the PAPA/CAPA. Raters were trained to reliability, with excellent reliability for MDD diagnosis ($\alpha = .81$). Preschool depression severity was assessed at T1–T3 and was quantified as a dimensional variable of average depressive symptoms

from the PAPA. Preschool-onset MDD (PO-MDD) was indexed by an average of four or more of the nine MDD symptoms endorsed at any of the three preschool assessments prior to age 6. Dimensional depression symptom severity scores were assessed at T5–T10 and were quantified as the number of the nine core DSM-IV MDD symptoms endorsed by the child and/or parent (summary score of most severe endorsement from parent or child). In our analyses, mean depression severity scores across T5 to T10 assessments were used for our depression outcome to account for multiple follow-up assessments. Participants completed an average of 3.64 ($SD = 1.66$) of the possible six follow-up assessments. Rather than assessing depression at a single timepoint, we chose to average depressive symptoms across multiple time points to provide a better reflection of total depression experience and severity across adolescence.

Data Analytic Plan

To test the prospective relationship between preschool emotion labeling, middle childhood emotion regulation, and subsequent adolescent depressive symptom severity, we first used multilevel modeling (MLM) to examine developmental trajectories of emotion labeling across the three preschool assessments (T1, T2, T3), as was done in a subset of participants who had neural data in Elsayed et al. (2021). The sample size included 119 more subjects than those analyzed by Elsayed et al. and also extended analyses to examine relationships to depression. Separate MLMs were conducted using the number of emotions correctly identified for each of the six emotions in the Facial Affect Comprehension Evaluation (FACE)—Emotion Labeling task (Mrakotsky, 2001) as dependent variables. Time was defined as an assessment wave, with T1 = 0, T2 = 1, and T3 = 2, and the quadratic effect (time squared) was included in the models when significant at $p < .05$. Age at T1, centered at mean age 4.45 was included as an independent variable in the models, and the interaction between T1 age and time was included if significant at $p < .05$. The MLMs included both random intercept and slope components and assumed an unstructured covariance structure. Individual subject intercepts and slopes were extracted from the models for use as predictors in mediation analyses if they were significantly correlated with at least one of the emotion regulation measures (emotion lability/negativity and emotion regulation) and adolescent depressive symptom severity. We also examined whether children with and without PO-MDD differed in any of these slopes or intercepts or emotion regulation mediators using t -tests to determine if a measure of preschool depression needed to be included as a covariate in mediation models.

We then tested whether childhood emotion regulation mediated the association between preschool emotion labeling MLM slopes/intercepts and late adolescent depressive symptom severity. To do this, we estimated the indirect effects using bootstrap estimates and 95% confidence intervals. We conducted six mediation analyses, including baseline emotion labeling (MLM intercepts) or developmental growth in emotion labeling (MLM slopes from T1–T3) from FACE—Emotion Labeling scores as the independent variables, middle childhood ERC emotional lability and emotion regulation subscale scores (T5) as parallel mediators, and average depressive symptom severity scores from the CAPA and KSADS-PL (T5–T10) as the dependent variable. Covariates in all mediation analyses were biological sex (male/female) and preschool

depression severity. Methods were reviewed and approved in accordance with ethical guidelines by the Washington University School of Medicine Institutional Review Board and informed consent and child assent were obtained from all participants.

Transparency and Openness

The current study reports how we determined our sample size (i.e., using the largest possible sample from the PDS study), all data exclusions at each time point, all manipulations and measures relevant to this set of analyses (i.e., emotion labeling, emotion regulation, and depression measures), and follows Journal Article Reporting Standards (JARS; Kazak, 2018). MLMs were conducted using SAS V.9.4 (SAS Institute Inc.), and mediations were run using the PROCESS Macro, V2.16 (Hayes, 2012) in SAS V.9.4 (SAS Institute Inc.). Analytic code for analysis is available at osf.io/bf5da. Data for this study are not publicly available, yet materials and data will be provided in a timely manner to other researchers upon reasonable request. This study's design and analysis were not preregistered.

Results

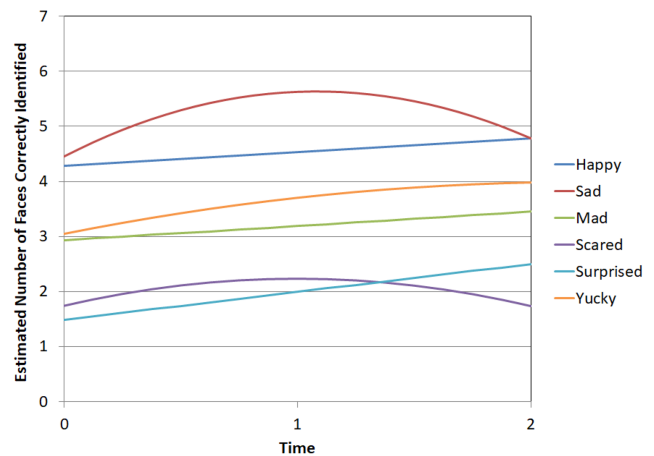
Demographic and Clinical Characteristics

Demographic and clinical differences included sex differences in intercepts from the MLM of preschool emotion labeling of surprised faces, $t(256) = -2.05, p = .04, d = 0.26$, childhood ERC emotion lability, $t(256) = 3.19, p = .002, d = 0.39$, and ERC emotion regulation, $t(256) = -2.01, p = .046, d = 0.26$, with females estimated to identify significantly more surprised faces at baseline and demonstrating lower emotion lability and higher emotion regulation than males. Moreover, given children were oversampled for depressive symptoms, we examined the impact of PO-MDD on analyses. Children with PO-MDD ($n = 89$) demonstrated no differences in any preschool emotion labeling metrics (intercepts or slopes) compared to children without PO-MDD ($n = 169$), but did demonstrate higher childhood ERC emotion lability, $t(256) = -6.13, p < .001, d = 0.41$, worse ERC emotion regulation, $t(256) = 4.10, p < .001, d = 0.41$, and higher adolescent depressive symptom severity $t(256) = -6.67, p < .001, d = 0.41$. As such, both sex and preschool depression severity were included as covariates in mediational models. We covaried for preschool depression severity, dimensionally assessed, instead of PO-MDD diagnosis to be consistent with our continuous depression severity outcome.

Developmental Trajectories of Emotion Labeling

Results of the multilevel models of the three preschool emotion labeling assessments are shown in Figure 1 and provided in Table S1 in the online supplemental material. Results demonstrated a linear increase in the labeling of happy, angry, and surprised faces across the three assessment periods during preschool. A quadratic trend in scared faces was found, such that number of scared faces identified increased from T1 to T2, but then showed a decrease from T2 to T3. For both sad and disgust face labeling, results demonstrated not only a significant quadratic trend such that number of sad faces identified was highest at T2 and disgust face labeling leveled off from T2 to T3, but also a baseline (T1) age by time interaction, such that between participants at T1, number of sad

Figure 1
Emotion Labeling Estimated Trajectories From Multilevel Models (n = 304)



Note. See the online article for the color version of this figure.

and disgusted faces correctly identified varied significantly more according to T1 age than at T3, when sad and disgust face labeling accuracy levels were more comparable between participants, regardless of T1 age (see Figures 1 and 2 in the online supplemental material).

Associations Between Emotion Labeling, Emotion Regulation, and Depressive Symptoms Across Development

Descriptive information and correlations between preschool emotion labeling, childhood emotion regulation, and adolescent depressive symptom severity are provided in Table 1. Specifically, lower baseline happy and anger labeling (i.e., MLM intercept) were significantly associated with higher childhood emotion lability and higher depressive symptom severity, while sharper increases in labeling of happy and anger faces across the preschool period (i.e., MLM slope) were associated with higher childhood emotion lability and higher depressive symptom severity. Unlike childhood emotion lability, childhood emotion regulation was not associated with preschool happiness or anger labeling. For surprise labeling, lower baseline surprise labeling was significantly associated with higher childhood emotion lability and higher depressive symptom severity, while more gradual increases in the labeling of surprised faces across the preschool period (i.e., MLM slope) were associated with lower childhood emotion regulation and higher depressive symptom severity. Lower baseline sadness labeling and sharper increases in the labeling of sad faces across the preschool period (i.e., MLM slope) were both significantly associated with higher childhood emotion lability and worse emotion regulation. Contrary to our hypotheses, there were no significant relationships between preschoolers' sadness labeling and adolescent depressive symptom severity. While higher baseline disgust labeling was associated with lower adolescent depressive symptom severity, no relationships between emotion lability/negativity nor emotion regulation and depressive symptoms emerged for labeling of scared faces.

Table 1

Descriptive Information and Correlations Between Emotion Labeling, Emotion Regulation and Preschool and Adolescent Depressive Symptom Severity (n = 258)

Construct	<i>M (SD)</i>	ERC emotion lability/ negativity	ERC emotion regulation	Preschool depressive symptom severity	Adolescent depressive symptom severity
Preschool emotion labeling					
Happy intercept	4.29 (.79)	-0.13*	0.10	-0.06	-0.13*
Happy slope	0.25 (.42)	0.13*	-0.10	0.06	0.13*
Sad intercept	4.47 (1.55)	-0.15*	0.14*	-0.08	-0.08
Sad slope	2.18 (.62)	0.13*	-0.14*	0.07	0.07
Anger intercept	2.94 (.60)	-0.13*	0.10	-0.11	-0.14*
Anger slope	0.26 (.12)	0.14*	-0.10	-0.01	0.15*
Scared intercept	1.76 (.79)	0.08	-0.01	0.02	0.02
Scared slope	0.99 (.19)	-0.06	-0.01	-0.001	-0.001
Surprised intercept	1.49 (.78)	-0.14*	0.12	-0.004	-0.13*
Surprised slope	0.51 (.30)	-0.11	0.14*	-0.10	-0.18**
Disgust intercept	3.07 (.92)	-0.10	0.12	-0.09	-0.13*
Disgust slope	0.83 (.28)	0.57	-0.08	0.01	0.08
Childhood emotion regulation					
ERC emotion lability/negativity	24.62 (6.9)		-0.63**	0.50**	0.53**
ERC emotion regulation	27.69 (3.5)			-0.35**	-0.40**
Depression symptom severity					
Preschool symptom severity	2.33 (1.55)				0.48**
Adolescent depressive symptom severity	2.78 (1.83)				

Note. Emotion labeling intercepts and slopes derived from baseline-centered (T0) multilevel models across three preschool assessments; ERC = emotion regulation checklist; preschool depressive symptom severity is the mean number of depressive symptoms across three assessments spanning the preschool period; depressive symptom severity is the mean number of depressive symptoms at up to six follow-up assessments across adolescence.

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

Predictive Relationships Between Emotion Labeling, Emotion Regulation, and Depressive Symptoms Across Development

In testing whether the prospective relationships between preschool emotion labeling and adolescent depressive symptoms act via middle childhood emotion lability and emotion regulation (i.e., assessed via ERC subscales), parallel mediation analyses were conducted only for emotion labeling variables that were significantly correlated with at least one of the emotion regulation measures (emotion lability/negativity and emotion regulation) and adolescent depressive symptom severity. This included emotion labeling variables for happiness, anger, and surprise. For anger, lower baseline anger labeling significantly predicted higher childhood emotion lability, which in turn was associated with significantly higher adolescent depressive symptom severity, and the indirect effect (mediation) was significant; Indirect effect: $B(SE) = -.15(.08)$, 95% CI [-0.33 to -0.02] (see Figure 2). For preschool anger labeling trajectories, a steeper slope, or sharper increases in the labeling of angry faces across the preschool period predicted significantly higher childhood emotion lability, which in turn was associated with significantly higher adolescent depressive symptoms, and the indirect effect was significant; Indirect effect: $B(SE) = .84(.40)$ [0.16-1.75] (see Figure 2). For emotion labeling of surprised faces, lower baseline surprise labeling predicted higher childhood emotion lability, which in turn was associated with elevated adolescent depressive symptoms, and the indirect effect was significant; Indirect effect: $B(SE) = -.11(.05)$ [-0.23 to -0.02] (see Figure 2). To aid in the interpretation of mediations, box plots of anger and surprise identification across the three preschool time points are included

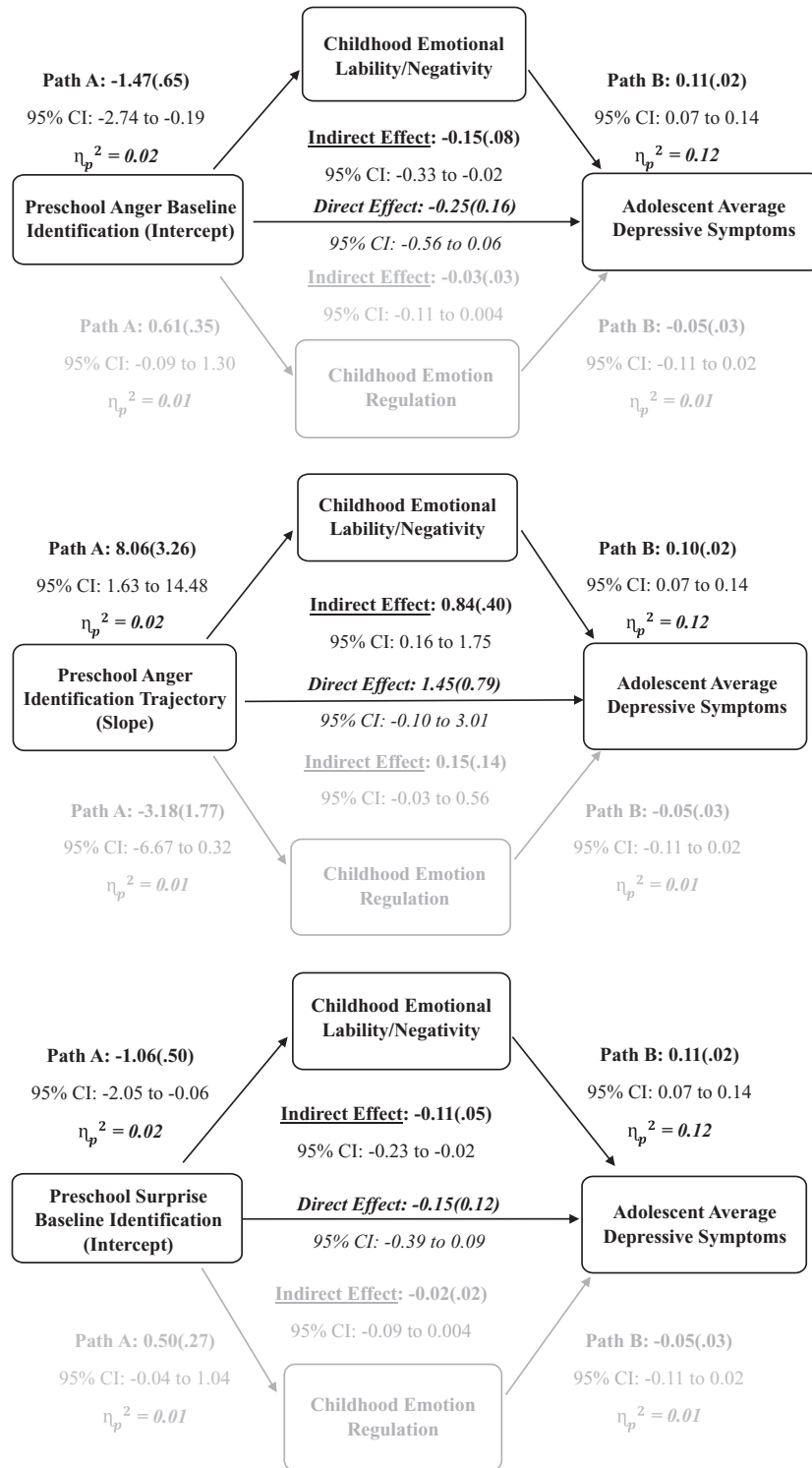
in Figure 3 in the online supplemental material. Neither emotion lability nor emotion regulation was significant mediators of the relationship between happy emotion labeling intercepts or slopes and later depression severity. Post hoc analyses for scared and disgust emotion labeling can be found in the supplement (see Tables 2 and 3 in the online supplemental material for all mediation results).

Discussion

The goal of the current study was to investigate if childhood emotion processes, including emotion labeling and regulation, were implicated in pathways of adolescent depression risk. At baseline, preschoolers demonstrated the highest labeling accuracy for happy and sad faces (indicating earlier development of labeling for these emotions), while demonstrating the lowest accuracy for surprised and scared faces (indicating a possible protracted development of labeling for these emotions). Furthermore, emotion labeling ability was found to generally increase for all emotions across the preschool period. Linking childhood emotion processes and depression, happy, anger, and surprise labeling in early childhood were associated with both childhood emotion regulation/dysregulation and adolescent depressive symptom severity. Above and beyond baseline preschool depressive symptoms, early childhood emotion labeling deficits for anger and surprise indirectly predicted higher adolescent depressive symptoms via childhood lability/negativity (but not worse emotion regulation ability). Overall, our findings support prior literature about the negative consequences of atypical emotional development related to depression (Cole et al., 2008). More specifically, our study provides initial evidence that early childhood emotion labeling

Figure 2

Emotion Lability/Negativity and Emotion Regulation as Mediators of the Relationship Between Preschool Emotion Labeling Trajectories and Adolescent Depressive Symptom Severity, Controlling for Sex and Preschool-Onset MDD (n = 258)



Note. Total effect of anger intercept: $B(SE) = -0.44(.17)$, 95% CI [-0.78 to -0.09]. Total effect of anger slope: $B(SE) = 2.45(.87)$ [0.73 to 4.17]. Total effect of surprise intercept: $-0.29(.14)$ [-0.55 to -0.02]. MDD = major depressive disorder; CI = confidence interval.

deficits, although not associated with concurrent depression, may be a risk factor for later adolescent depressive symptoms through a developmental pathway mediated by childhood emotion regulation deficits.

Given the current study indexed emotion labeling across early childhood, and emotion labeling ability is widely reported to improve with age (Durand et al., 2007; Stifter & Fox, 1986), relationships between emotion labeling and depressive symptoms may differ across development. Therefore, researchers believed it important to investigate age-related changes in emotion labeling. Results for developmental pathways of emotion labeling in the preschool period aligned with prior literature suggesting that emotion labeling ability across childhood generally increases with age, but fluctuates according to the emotion assessed. Specifically, preschoolers identified a higher number of “sad”, “happy,” and “angry” faces at baseline than “surprised,” “disgusted,” and “fearful” faces. In other words, findings were consistent with developmental models that suggest emotion labeling for certain emotions reaches adult-level accuracy earlier in development than for other emotions (Bruce et al., 2000; Elsayed et al., 2021; Mondloch et al., 2003). Moreover, since surprise labeling matures comparatively later in development (Durand et al., 2007; Mondloch et al., 2003), a higher level and greater increase in surprise labeling in the preschool period may indicate an overall greater competency for emotion labeling (in contrast to more modest improvements found in already developmentally matured labeling of sadness and happiness, see Figure 1). Another notable finding that contributes to the literature on emotion labeling development was that preschool-aged children oversampled for depression demonstrated similar trends in performance to that of nondepressed peers (Durand et al., 2007), indicating that depressive symptoms were not related to emotion labeling ability at this early age.

Crucially, the current study found that labeling deficits of specific emotions (i.e., anger and surprise) were related to depression via meditational effects of emotion dysregulation. As predicted, childhood emotion labeling deficits for anger (i.e., demonstrated as both decreased labeling of angry faces in early childhood (intercept) and increased anger labeling across early childhood (slope)) indirectly predicted adolescent depressive symptoms through middle childhood lability/negativity. These findings on emotion labeling are consistent with patterns of broader emotion recognition deficits found in prior depression literature, indicating that depression is associated with poor emotion labeling of negative emotions (Lenti et al., 2000), such as anger (Surguladze et al., 2004). Taking our findings into developmental context, our results may suggest that anger labeling deficits implicated in depression risk change across development. In other words, in early childhood, poor emotion labeling of anger may be a risk factor for depression; yet, as children age, at-risk children may instead demonstrate anger labeling that improves more rapidly, possibly becoming more attuned to emotions implicated in depressed mood, including anger (Lane & Terry, 2000). Of note, poor anger labeling at baseline (i.e., intercept) and a sharp increase in anger labeling (i.e., slope) are most likely not independent risk factors and most likely not unique mechanisms of depression risk. Specifically, those children with worse anger labeling at baseline may demonstrate relatively faster increases in anger labeling across development because they have more room for improvement. This suggests that poor early anger identification may be driving findings and may indeed, be the central emotional deficit to target.

Although not hypothesized, surprise labeling, an emotion that is not universally found to be altered in depressive symptoms, was implicated in the developmental pathway to adolescent depression. Specifically,

poor baseline surprise labeling during the preschool period indirectly predicted adolescent depression via higher childhood emotion lability/negativity. Prior literature finds that children do not possess adult-level labeling of surprised emotions until late childhood (Gosselin, 1995). Given its protracted development compared to basic emotions, surprise labeling may be a more sensitive indicator of depression risk in early childhood. In other words, uniquely low surprise labeling ability may index lower overall emotional competence compared to controls, a robust risk factor for depression (Egger & Angold, 2006; Luby & Belden, 2006). However, other explanations may exist regarding our finding on a developmental risk pathway of surprise recognition. It is possible that our results were subject to a floor effect in surprise recognition, such that the few preschoolers who had relatively *better* or more advanced surprise labeling ability earlier in development may possess a more adaptive emotional progression (see Figure 3 in the online supplemental material). In other words, advanced emotional competence by being able to identify surprise early in one’s development may compound into less emotional lability and hence putting them at lower risk for depressive symptoms. An alternative explanation for finding a relationship between surprise labeling and depression is that preschoolers at risk for depression are prone to misinterpret surprised faces. Prior literature suggests that young children confuse surprised and fearful faces (Gosselin, 1995) and mislabel each emotion as the other. In turn, although speculative, our finding that poor preschool surprise labeling predicted adolescent depressive symptom severity may indicate that at-risk children are particularly prone to perceiving surprised faces as fearful and over-identify surprised faces as negative, fearful expressions, consistent with prior literature regarding a negative response bias in depression (Bourke et al., 2010; Gur et al., 1992; Leppänen et al., 2004).

Contrary to our hypotheses, preschoolers’ sadness and happiness emotion labeling did not predict adolescent depressive symptom severity by way of emotion regulation. This finding is inconsistent with a large (albeit still mixed) literature on emotion labeling response bias that suggests depressed adults and adolescents over-identify sad emotion expressions and under-identify happy emotion expressions compared to nondepressed peers (Bourke et al., 2010; Dalili et al., 2015; Nyquist & Luebke, 2020). Despite these deficits, however, many studies find no group differences in sadness labeling (Dalili et al., 2015) and happiness labeling (Smoller & Brosgole, 1993; Tsypes et al., 2016) between depressed, at-risk, and nondepressed samples, suggesting that depression may be characterized by different indexes of emotion recognition than measured by the current study. In other words, sadness and happiness recognition deficits in depression may be due to a response bias for sad faces and away from happy faces (Nyquist & Luebke, 2020; rather than lower labeling ability assessed by the current study). Our results could also indicate that a developmentally nuanced pathway of sadness and happiness labeling exists in depression, such that associations between labeling deficits for these emotions and depressive symptoms may occur only later in childhood, rather than during the preschool period assessed. Another explanation for the study’s null findings linking happiness and sadness labeling deficits to depression points to alternative developmental pathways of risk, such that this relationship is instead mediated by negative interpersonal relationships.

Interestingly, contrary to our hypotheses, only one of the two types of emotion regulation/dysregulation assessed (i.e., higher childhood emotion lability/negativity, but not worse emotion regulation) mediated the relationship between preschool emotion labeling deficits

and adolescent depressive symptoms. Results indicated that aberrant anger and surprise labeling predicted increased emotion dysregulation (through lability/negativity) but did not predict increased emotion regulation through worse emotion regulation ability. One speculative hypothesis for these results indicates that if a child cannot recognize anger well in facial expressions, their own emotional experiences of anger may be similarly hard to identify, leading to exaggerated feelings of threat linked to increased emotion lability. In other words, conceptualizing one's physiological emotion experience in emotion labeling may decrease the perceived intensity or seemingly unpredictable nature of one's negative emotions. Thus, successful emotion labeling may only precipitate less emotional lability rather than increased emotion regulation. Indeed, research in adults demonstrates that simply labeling one's emotions is associated with decreased negative emotional experience (Torre & Lieberman, 2018) and decreased levels of negative affect intensity, which has also been directly linked with decreased depression (Rydell et al., 2003; Zeman & Shipman, 1996). Alternatively, our findings may merely demonstrate that parents are better raters of their child's labile and negative expressions of emotions than regulatory ability.

Despite its inherent strengths, the current study has several limitations. First, we were unable to examine preschool emotion labeling and regulation concurrently to assess how the two processes develop in parallel. Future research should characterize the interactive processes between emotion labeling and regulation as development is unfolding. Third, the current study does not explain how or why these two emotion processes relate. In other words, further research is warranted to investigate whether emotion labeling is a component of emotion regulation, or if these two variables can be isolated into separate emotion processes. Similarly, in expanding upon emotion developmental trajectories that the current study did not assess, future research should assess if specific emotion regulation strategies (i.e., cognitive reappraisal, emotion suppression, etc.) and emotion recognition deficits for complex emotions (e.g., shame) act as mechanisms that confer risk for depressive symptoms. Fourth, the current study only investigated relationships between one index of emotion recognition (i.e., emotion labeling ability) and depression. Deficits in basic emotion labeling (i.e., happiness, sadness, anger labeling), particularly for emotions implicated in depressed mood, may be found in emotion recognition deficits indexed by response bias.

Constraints on Generality

Participants in this study included a clinically heterogeneous sample with psychiatric illnesses that were oversampled for pediatric depressive symptoms at baseline. While analyses generated significant findings after controlling for baseline depression symptom severity and preschool depressive symptom severity, findings may not generalize to less severe or nonclinical high-risk samples. Additionally, the current study is possibly limited in the generalizability of study findings across racial and ethnic groups. The study's emotion labeling task did not include many racially diverse stimuli and these stimuli were not demographically matched to participant characteristics. Since evidence suggests that race-based differences may exist in emotion labeling (Segal et al., 2019), future research is warranted on the role of race, and associated indices of social disadvantage, in emotion processing pathways of depression risk.

Despite these limitations, the current study advances prior research on the etiology of one of the most burdensome illnesses

among today's youth. Our results suggest that an emotion processing pathway implicated in depression risk exists, beginning in preschool and continuing across childhood and adolescence. While the effect sizes of each component may be small, this study shows there is a significant effect within a longitudinal pathway spanning almost 10 years of development when youth experience complex intersections of depression risk (e.g., socioeconomic status, identity conflict, increased social demands). As such, although emotion labeling and regulation/dysregulation are only a tiny fraction of the developmental experience, our effect sizes may still index that these emotion processes were consequential in predicting future adolescent depression severity. Moreover, our current study includes novel temporal information about child/adolescent emotion development trajectories. Such delineations are crucial for targeted, effective early interventions for depression. In addition, our findings provide evidence of the potential long-term psychological health benefits of early emotion-based interventions (e.g., teaching emotion labeling of anger and surprise to mitigate later depressive symptom severity), adding to initial findings that early emotion-based interventions successfully improve preschoolers' emotion regulation ability and subsequent psychopathology (Finlon et al., 2015; Izard et al., 2008; Luby et al., 2012). Overall, we believe that disrupted emotion processing in early childhood, specifically for anger and surprise labeling, may be an important contributing factor for later depressive symptoms by means of increased lability/negativity. By investigating emotion processing mechanisms of depression in youth like those in the current study, we can better understand how emotional experiences go awry in a leading cause of disability and inform intervention during the malleable period of childhood.

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