Testing a Developmental Model of Parental Warmth, Amygdala–Subgenual Anterior Cingulate Cortex Connectivity, and Depressive Symptoms in Adolescents During the COVID-19 Pandemic

Jonas G. Miller¹, Tiffany C. Ho², Jaclyn S. Kirshenbaum¹, Rajpreet Chahal¹, Anthony J. Gifuni^{1,3}, & Ian H. Gotlib¹

1. Stanford University

2. University of California, San Francisco

3. McGill University

***Corresponding author:** Jonas G. Miller, Department of Psychology, Stanford University, 450 Jane Stanford Way, Stanford, CA 94305. Email: jgmiller@stanford.edu. Phone: 510-717-0412

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Abstract

Background: Neurobiological measures may serve as predictive markers of risk for and resilience to depressive symptoms during the COVID-19 pandemic. We tested a developmental model linking variation in amygdala–subgenual anterior cingulate cortex (sgACC) resting-state connectivity both to earlier experiences in the family environment and to subsequent vulnerability to depressive symptoms during the pandemic.

Methods: We used data from a longitudinal study that included three waves (N=214 adolescents; ages 9-15 years at Time 1 (T1), 11-17 years at Time 2 (T2), and 12-19 years during the pandemic at Time 3 [T3]). We assessed parental warmth (T1), depressive symptoms (T1 to T3), and functional connectivity between the sgACC and basolateral (BLA) and centromedial amygdala (CMA) (T1 and T2). We modeled associations among early parental warmth, amygdala–sgACC connectivity, and depressive symptoms before and during the pandemic.

Results: Less parental warmth was associated prospectively with stronger BLA–sgACC connectivity approximately two years later ($\beta = -.23, p = .021$) over and above the effect of BLA–sgACC connectivity at T1. Stronger BLA–sgACC connectivity, in turn, was associated with heightened depressive symptoms, both before ($r = .21, p = .031$) and during the pandemic ($\beta = .22, p = .031$; independent of the effect of pre-pandemic symptoms).

Conclusion: Adolescents who experience less parental warmth may develop a pattern of BLA–sgACC connectivity that increases their risk for mental health problems during the pandemic. BLA–sgACC connectivity in early to middle adolescence may be a predictive marker of risk for depressive symptoms in general and specifically during periods of heightened stress.

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2, or COVID-19) pandemic is a unique period of disruption and stress. Many individuals are facing multiple challenges related to the pandemic that pose serious threats to mental health, including economic hardship, threats to physical health, and enforced social isolation (1–3). Adolescents generally are at increased risk for stress-related mental health problems such as depression (4,5) and, therefore, may be particularly vulnerable during the COVID-19 pandemic. Indeed, depressive symptoms in adolescents appear to be increasing during this period (6,7). It is almost certain that some adolescents are more vulnerable than others to experiencing difficulties during the COVID-19 pandemic, differences that have been found to be indexed by neurobiological and physiological markers (8–10). We recently showed that aspects of neurobiology can moderate risk and resilience to depressive symptoms during the COVID-19 pandemic (8,10), and, therefore, might serve as targets for intervention and prevention. Moreover, identifying developmental factors that are associated with predictive markers of mental health during the COVID-19 pandemic could inform efforts to promote resilience to psychopathology during future periods of stress. In this context, therefore, the present study tests a developmental model linking variation in neural connectivity in adolescents to both earlier experiences in the family environment and subsequent vulnerability to depressive symptoms during the COVID-19 pandemic.

Depression has been found to be characterized, in part, by functional abnormalities in the amygdala and subgenual anterior cingulate cortex (sgACC) (11,12). In fact, resting-state functional connectivity (FC) between the amygdala and sgACC has been implicated in both stress and depression (13,14). Broadly, whereas the amygdala supports rapid detection and responding to salient events (15), the sgACC is involved in affective appraisal, sad mood, and

rumination (16). Several studies with adolescents have found that stronger positive amygdala–sgACC FC is associated with major depression (14,17,18), with perceived psychological stress (19), and with negative affectivity, or increased sensitivity to negative stimuli (20); this latter study also found that increases in amygdala–sgACC connectivity were associated with the onset of depression (20). Similar findings have been reported in adults (13); further, a stress reduction intervention has been found to weaken amygdala–sgACC connectivity (13). Taken together, it appears that increased intrinsic amygdala–sgACC connectivity plays a role in stress, negative affect, and vulnerability to depression. It is unclear, however, whether amygdala–sgACC connectivity is a promising candidate marker for risk and resilience to increasing depressive symptoms specifically during periods of heightened stress, such as the COVID-19 pandemic.

It is important that a predictive marker of risk and resilience during the COVID-19 pandemic be associated with well-being in an enduring manner. For example, it is possible that pre-pandemic amygdala–sgACC connectivity is associated concurrently with pre-pandemic depressive symptoms, and that this accounts for any prospective associations with functioning during the pandemic. In other words, pre-pandemic amygdala–sgACC connectivity may have a transient, rather than a unique and enduring, effect on the development of depressive symptoms during the pandemic. If amygdala–sgACC connectivity is a viable marker of risk versus resilience to depressive symptoms during periods of stress, it should prospectively predict depressive symptoms during the pandemic above and beyond pre-pandemic symptoms.

In addition to considering amygdala–sgACC connectivity as a predictive marker of functioning during the pandemic, it is important to examine whether variation in the development of this neural connection is related to a history of environmental risk for psychopathology. Indeed, Holz and colleagues (21) recently proposed that the amygdala and

ventral regions of the ACC are neural convergence sites for social risk and resilience factors. One such social environmental factor that has been implicated in adolescent depressive symptoms is parenting practices (22,23), which has also recently been linked to adolescent neurodevelopment (24–26). Specifically, parental warmth, including expressions of affection, acceptance, and sensitivity, has been found to be negatively associated with adolescents' development of depressed mood (27) and positively associated with adolescents' effective coping with stress (28). Thus, parental warmth may help to shape emotional development and influence how adolescents respond to challenging contexts (29).

A growing body of research is investigating how parenting practices are related to the development and functioning of neural regions implicated in adolescents' well-being. For example, in adolescents with a history of anxiety problems, parental warmth has been prospectively associated with reduced amygdala and sgACC activation during an emotion processing task, and has been indirectly linked with less severe internalizing symptoms via reductions in sgACC activation (30). In other research, adolescents with mothers who direct more negative affect towards them show blunted amygdala and sgACC response to a social reward (31) and alterations in amygdala resting-state FC (32). Thus, although prior studies have reported evidence that positive versus aversive parenting is related to amygdala and sgACC activation and amygdala FC, researchers have not yet examined parenting practices specifically in relation to amygdala-sgACC FC, despite their broader link with adolescent well-being. In addition, most studies have used cross-sectional samples or longitudinal designs with a single neuroimaging assessment (but see (33) for a longitudinal study of parenting and amygdala connectivity with two waves of neuroimaging data). Conducting multiple neuroimaging

assessments is critical for examining the effects of parenting on the development of amygdala–sgACC FC during adolescence.

The current longitudinal study examined whether amygdala–sgACC resting-state FC in adolescents is (a) related to parental warmth earlier in adolescence; and (b) is a predictive marker of risk for experiencing depressive symptoms both concurrently (i.e., pre-pandemic) and prospectively during the early phase of the COVID-19 pandemic. We expected that lower parental warmth earlier in adolescence would be associated concurrently and prospectively with stronger amygdala–sgACC connectivity. Further, given that amygdala–sgACC connectivity has been implicated in both stress and depression (13,14), we expected that stronger connectivity would, in turn, be associated with heightened depressive symptoms, both before and during the COVID-19 pandemic. Finally, we expected that the prospective association between parental warmth and amygdala–sgACC connectivity would be independent of the effect of earlier amygdala–sgACC connectivity on later connectivity, and that the prospective association between amygdala–sgACC connectivity and depressive symptoms during the COVID-19 pandemic would be independent of the effect of pre-pandemic depressive symptoms.

Methods and Materials

Participants and Procedures

Participants were adolescents from the San Francisco Bay Area who were part of an ongoing longitudinal study of the effects of early life stress on neurodevelopment during puberty (N=214, 121 females, mean age at baseline assessment=11.40, SD=1.0; 44.4% White, 20.1% biracial; 10.3% Asian; 8.4% Black; 8.4% Hispanic/Latinx; 6.5% other than what was listed; 1.9% did not report; median family income=\$100K-\$125K, range from less than \$5K to greater than \$150K). Families were recruited from the community using local flyers, media, and online

advertisements. Study exclusion criteria for the baseline assessment included inability to undergo MRI (e.g., had metal implants, braces), a history of neurological disorder or major medical illness, serious cognitive or physical challenges that might interfere with the ability to understand or complete procedures, non-fluent English speakers, and, for females, the onset of menses. Males and females were matched on self-reported pubertal stage at the baseline assessment. The current analysis included adolescents who completed up to 3 assessments, approximately 2 years apart between assessments ($N=214$; see Supplement for additional information about attrition and study design). At Time 1 (T1; $n=208$, 117 females, mean age=12.02 years, $SD=1.45$, range=9.17-15.82), adolescents reported on parenting behaviors in their primary caregiver, reported on their own depressive symptoms, and completed a resting-state functional magnetic resonance imaging (fMRI) scan from which we measured FC between the amygdala and sgACC. Approximately two years later at Time 2 (T2; $n=144$, 78 females, mean age=14.29, $SD=1.59$, range=11.08-17.91), adolescents completed a second resting-state fMRI scan and second assessment of their own depressive symptoms. At Time 3 (T3; $n=109$, 65 females, mean age=16.30, $SD=1.45$, range=12.82, 19.98), approximately three weeks after shelter-in-place was implemented in the Bay Area (April of 2020), adolescents reported on their depressive symptoms over the preceding one week using a measure administered online. The current analyses included participants who had useable data on any variable of interest at any study time point. All families signed assent and consent forms and were compensated for their participation. This study protocol was approved by the Stanford University Institutional Review Board.

Parental Warmth at Time 1

At T1, adolescents reported on parental warmth using the 11-item Warmth subscale of the Parenting Styles and Dimensions Questionnaire (e.g., “my parent gives comfort and

understanding when I am upset,” “my parent gives me praise when I am good”) (34).

Adolescents rated items on a 5-point scale ranging from 1 (“almost never”) to 5 (“very often”).

We averaged responses to the 11 items to yield a total score for parental warmth at T1 ($\alpha=.89$).

Resting-state fMRI at Time 1 and Time 2

At T1 and T2 (1.28 to 3.85 years after the T1 assessment; mean interval=2.03 years, SD=.34), neuroimaging data were acquired using a GE Discovery MR750 3T scanner (GE Medical Systems, Milwaukee, WI, USA) with a 32-channel head coil (Nova Medical, Wilmington, MA, USA). As described previously (35), for each participant, a high-resolution T1-weighted structural image was obtained using a spoiled gradient echo (SPGR) scan sequence (TR/TE/TI=6.24/2.34/450 ms; flip angle=12°; matrix size=256x256, 186 slices; FOV=23 cm; scan duration=5 min 15 s). A resting-state functional scan was acquired using an axial echo-planar imaging T2*-weighted sequence (TR/TE=2000/30 ms; flip angle=77°; matrix size=70x70; voxel size=3.2mm³; 37 slices; FOV: 224x224 mm; scan duration=6 min). For this scan, participants were instructed to stay awake and not fall asleep.

As described in (35), neuroimaging data were preprocessed using tools from Freesurfer, FSL, and AFNI. Structural and functional data were visually inspected to ensure data integrity. SPGR images were skull-stripped and white matter (WM) and cerebrospinal fluid (CSF) were parcellated and eroded using Freesurfer's *recon-all* routine before being registered to the MNI 2mm standardized atlas using linear transformations in FSL (*FLIRT*). The first six volumes of the functional time series were discarded to allow signal stabilization, leaving 174 volumes for subsequent analyses. Participants with greater than a mean framewise displacement of 0.2 mm or with more than 20 volumes with greater than 0.25 mm framewise displacement were excluded from further analyses. Functional data were de-spiked (*3dDespike*) and physiological correction

was applied from the cardiac signals collected during the functional scan (*RETROICOR*).

Functional data were slice-time corrected and spatially normalized via registration to the 2 mm MNI standardized atlas using a series of linear transforms and spatially smoothed using a 5 mm full width at half maximum Gaussian kernel (*3dBlurInMask*). Preprocessed data were submitted into a generalized least squares regression (*3dREMLfit*) in order to regress out spurious activation related to physiology and motion by including WM timeseries, CSF timeseries and 24 motion parameters (3 translational, 3 rotational at each time point and the prior time point, and each of these 12 motion parameters squared) as covariates of non-interest. Residuals were bandpass filtered ($.009 < f < .08$ Hz) and de-meaned and used in seed-to-seed FC analyses.

As described in (36), we focused on two major subdivisions of the amygdala: the basolateral amygdala (BLA) and centromedial amygdala (CMA). The BLA is involved in associative learning and evaluation of affective significance of environmental stimuli (15). The CMA is involved in allocation of attentional resources and increasing arousal via regulation of autonomic, endocrine, and behavioral responses to evocative stimuli (37). We defined BLA and CMA subdivisions as region of interest (ROIs; see Figure 1a) using the Juelich anatomic atlas of probabilistic cytoarchitectonic maps provided in FSL (38,39). Prior studies have used these BLA and CMA ROIs to study FC in different amygdala subdivisions (40,41). We defined the sgACC as BA 25 in the Mackey atlas of probabilistic cytoarchitectonic maps provided in AFNI (see Figure 1b) (42).

Adolescent Depressive Symptoms at Time 1, Time 2, and Time 3 (During the Pandemic)

At T1 and T2, adolescents reported on their depressive symptoms over the preceding two weeks using the 10-item version of the Child Depression Inventory (CDI) (43). Adolescents

rated items on a 3-point scale. We summed responses to the 10 items to yield a total score for pre-pandemic depressive symptoms at T1 ($\alpha=.78$) and T2 ($\alpha=.90$).

At T3, 1.08 to 4.35 years after the T2 assessment (mean interval=2.32, SD=.83), adolescents reported on their depressive symptoms over the preceding week using the Center for Epidemiological Studies Depression Scale for Children (CES-DC) (44). We used the CES-DC at T3 because some participants at this assessment were older than the validated upper age limit for the CDI. The CES-DC has 20 items, each of which adolescents rated on a 4-point scale ranging from 0 (“not at all”) to 3 (“a lot”). We summed responses to the 20 items to yield a total score for depressive symptoms during the pandemic ($\alpha=.76$).

Finally, to make depressive symptom scores comparable across the three time points, we converted CDI scores at T1 and T2 and CES-DC scores at T3 to proportion of maximum scale scores (45).

Statistical Analyses

We used path analysis to examine hypothesized relations among parenting at T1, amygdala–sgACC FC and depressive symptoms at T1 and T2 (both pre-pandemic), and depressive symptoms at T1, T2 (both pre-pandemic), and T3 (during the pandemic). We tested separate models using bilateral average BLA–sgACC and CMA–sgACC as the neural connection of interest. We conducted secondary analyses testing amygdala–sgACC FC as a mediator between parental warmth and depressive symptoms. Model fit was assessed using χ^2 tests, the comparative fit index (CFI), and the root mean square error of approximation (RMSEA). Good fit was indicated by nonsignificant χ^2 values, CFI values $\geq .95$, and RMSEA values $\leq .05$. Full information maximum likelihood estimation was used to produce model parameter estimates and to account for missing data. Little’s test suggested that data were

missing completely at random ($\chi^2=174.93$, $df=149$, $p=.072$). All models were tested using the *lavaan* package in R software (46). We used False Discovery Rate adjusted p -values to correct for multiple tests (i.e., all modeled paths) within each model.

Results

Descriptive statistics for the sample are presented in Table 1. Adolescents reported higher levels of depressive symptoms during the pandemic (T3) than before the pandemic (T2) (mean difference=.21, $SD=.18$, $p<.001$). Depressive symptoms were not significantly different across T1 and T2 (before the pandemic) (mean difference=.01, $SD=.13$, $p=.334$). Adolescents exhibited significant BLA–sgACC and CMA–sgACC resting-state connectivity at T1 and T2 (all $ps<.001$). BLA–sgACC connectivity was significantly stronger than CMA–sgACC connectivity at both time points (all $ps<.001$).

The model that included bilateral T2 BLA–sgACC as the connection of interest is presented in Figure 2. The paths from sex and age at T2 to BLA–sgACC at T2 were not statistically significant (both $ps>.66$), and removing them from the model did not diminish model fit ($\chi^2(2)=0.20$, $p=.907$). We excluded these paths from the model to increase model parsimony. Girls reported more severe depressive symptoms than did boys both prior to the pandemic at T2 ($\beta=.16$, $p=.028$) and during the pandemic at T3 ($\beta=.28$, $p=.002$). Older participants reported more severe depressive symptoms than did younger participants prior to the pandemic at T2 ($\beta=.25$, $p<.001$), but not during the pandemic at T3 ($\beta=.14$, $p=.121$). We retained these paths and variables in the final model but do not present them in Figure 2.

Our final model that included bilateral T2 BLA–sgACC as the connection of interest showed good fit with the data ($\chi^2(17)=23.75$, $p=.126$, $CFI=0.95$, $RMSEA=.04$ (90% CI: .00, .08)). Less parental warmth at T1 was associated concurrently with more severe depressive

symptoms ($r=-.25, p=.003$). This pattern of parenting at T1 was also associated prospectively with stronger BLA–sgACC connectivity at T2 ($\beta=-.23, p=.021$) over and above the longitudinal effect of BLA–sgACC connectivity at T1 ($\beta=.18, p=.063$). Stronger BLA–sgACC connectivity at T2, in turn, was associated concurrently with more severe depressive symptoms ($r=.21, p=.031$) and prospectively with more severe depressive symptoms during the pandemic ($\beta=.22, p=.031$). This longitudinal association was independent of the effect of pre-pandemic (T2) depressive symptoms on depressive symptoms during the pandemic (T3; $\beta=.31, p=.006$).

The model results raised the possibility that BLA–sgACC connectivity at T2 mediated the association between parenting at T1 and depressive symptoms during the COVID-19 pandemic at T3. In a separate model, we computed indirect effects as the products of path coefficients linking parental warmth at T1 to BLA–sgACC connectivity at T2, and BLA–sgACC connectivity at T2 to depressive symptoms at T3. We used a two-tailed Sobel test to test the statistical significance of the indirect effect. The indirect effect of parenting experiences at T1 on depressive symptoms at T3 through BLA–sgACC connectivity at T2 was not statistically significant ($\beta=-.06, p=.075$). Models that focused on either right or left BLA–sgACC connectivity are presented in the Supplement.

Finally, we fit a model that included bilateral CMA–sgACC instead of BLA–sgACC as the connection of interest. CMA–sgACC connectivity at T1 and T2 were not significantly associated with parenting at T1 or depressive symptoms at any assessment (all unadjusted $ps>.145$). The longitudinal stability path of CMA–sgACC connectivity at T1 to CMA–sgACC connectivity at T2 was not statistically significant after FDR adjustment ($\beta=.19, p=.066$).

Discussion

Depressive symptoms in adolescents have increased during the COVID-19 pandemic (6). Indeed, in the current study we found that adolescents reported significantly more depressive symptoms during the early phase of the pandemic than they did before the pandemic. In fact, during the pandemic, adolescents in our sample reported, on average, experiencing depressive symptoms in the clinically significant range (CES-DC proportion of maximum score $> .25$ (44)). Not surprisingly, however, the COVID-19 pandemic has been more consequential for some adolescents than for others (6,7,47). Elucidating relevant aspects of neurobiological functioning could increase our understanding of individual differences in risk versus resilience to mental health problems during the pandemic (8–10). In the current study, we examined whether amygdala–sgACC resting-state FC assessed 1 to 4 years prior to the pandemic indicated risk for depressive symptoms in adolescents during the pandemic. Further, we tested whether connections between the sgACC and specific sub-components of the amygdala were related to parental warmth and depressive symptoms. We found that increased BLA–sgACC resting-state connectivity in middle, but not earlier, adolescence is associated concurrently with higher levels of pre-pandemic depressive symptoms, and prospectively with higher levels of depressive symptoms during the pandemic. Importantly, the prospective association is independent of the longitudinal stability of individual differences in depressive symptoms. Thus, BLA–sgACC connectivity in middle adolescence prospectively indexed risk versus resilience to increasing depressive symptoms during the pandemic. Specifically, stronger BLA–sgACC resting-state connectivity during middle adolescence predicted increased vulnerability of adolescents to the adverse effects of the pandemic on mental health. Our findings build on prior studies linking stronger BLA–sgACC connectivity with stress and depression (13,14,17,19,20) by demonstrating that this neurophenotype is associated with depressive symptoms both before and

during the COVID-19 pandemic, and that these associations emerge by mid-adolescence. The present study is among the first to report on the utility of resting-state FC as a longitudinal predictor of mental health in adolescents during the COVID-19 pandemic (10).

We found that the development of BLA–sgACC resting-state FC was related to positive parenting. Specifically, higher parental warmth earlier in adolescence was associated prospectively with the development of weaker BLA-sgACC connectivity; further, this was independent of the longitudinal stability of BLA–sgACC connectivity. Our findings suggest that parental warmth is associated with the development of a neurophenotype that may confer resilience versus risk for depressive symptoms. Prior research suggests that the amygdala and the ventral regions of the ACC are sensitive to social-environmental protective and risk factors (21). Positive versus aversive parenting practices have been identified as social-environmental factors that are related to resilience and risk for depressive symptoms related to adversity and development in adolescents (22,23,27,48,49); these types of parenting practices have also been associated with neurophenotypes implicated in risk for depressive symptoms, including altered amygdala and sgACC functioning (30–32). Our study extends these literatures by explicitly examining longitudinally the link between parental warmth and intrinsic connectivity between the amygdala and sgACC. It is noteworthy, however, that the indirect effect of parental warmth on depressive symptoms during the pandemic through pre-pandemic BLA-sgACC connectivity did not reach statistical significance. It is likely that we needed more statistical power to adequately test this indirect path. It is also possible that other neural connections act as a more robust link between parental warmth and the development of risk versus resilience to depressive symptoms during the pandemic. Longitudinal studies with larger samples are needed to test these possibilities.

Notably, our findings linking amygdala–sgACC FC to parental warmth and depressive symptoms were specific to the basolateral subdivision of the amygdala (BLA). Prior research suggests that the BLA plays a critical role in associative learning and evaluation and regulation of emotionally evocative stimuli (15,37), that stronger BLA–sgACC connectivity is related to stronger encoding of negative-emotion related information in individuals with higher internalizing symptoms (50), and that disruptions of these psychological processes are related to depression (51). Considered together with our findings, it may be that adolescents who exhibit stronger BLA–sgACC resting-state connectivity engage in psychological processes that confer vulnerability for experiencing depressive symptoms and that are sensitive to parenting influences. Another possible explanation for our findings is that the BLA, compared to other amygdala subdivisions, is involved in coordinating activity with the sgACC. Nonhuman animal research shows that amygdala projections to the ACC, including the sgACC region, originate primarily from the BLA (52,53). Adolescents in our sample had significantly stronger BLA–sgACC connectivity than CMA–sgACC connectivity.

We should note several limitations of this study. First, we used adolescents' reports of parenting practices. Thus, it is possible that as a neurophenotype of depressive symptoms, BLA–sgACC connectivity is reflecting negative perceptions that adolescents have of their parents, not necessarily parenting behaviors per se. A few neuroimaging studies have now integrated observational measures of laboratory-based parent-adolescent interactions (24,32,33). Similarly, researchers have recently used ecological momentary assessment to gain a more comprehensive understanding of daily interactions, behaviors, and emotions outside of the laboratory in relation to adolescent neurobiology (54). In this context, it is important that investigators conduct multimethod assessments to further elucidate the role of parenting and parent-child relationships

in adolescent brain development in general and, specifically, during periods of stress. Second, our resting-state fMRI scan was 6 minutes in length; longer resting-state scans improve the reliability of connectivity estimates, which is important for biomarker research (55). Third, our model did not include potential mechanisms linking BLA–sgACC connectivity to earlier parenting practices and subsequent depressive symptoms. Thus, it is not clear precisely how parenting practices earlier in adolescence might lead to altered BLA–sgACC connectivity, or precisely how this connectivity could influence later depressive symptoms during the COVID-19 pandemic in an enduring manner. Finally, researchers have documented health and social inequalities in the effects of the COVID-19 pandemic (2). While our sample may not have spanned the full range of financial and family problems that have been implicated in adolescent mental health during the COVID-19 pandemic, it is important to note that almost one-third of our sample reported that a family member earned less money during the early phase of the pandemic than before the pandemic (8).

Despite these limitations, the results of the current study suggest that the development of BLA–sgACC resting-state connectivity in adolescents is related to the quality of parenting practices in early adolescence and, further, is a biomarker of concurrent depressive symptoms in middle adolescence and prospective risk for experiencing depressive symptoms during the COVID-19 pandemic. Adolescents who perceive experiencing less parental warmth may develop a pattern of BLA–sgACC connectivity that is implicated in risk for mental health difficulties broadly and specifically in response to a significant stressor, such as the COVID-19 pandemic.

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Table 1. Descriptive Statistics

	Mean or %	SD	Range
T1 Parental Warmth	4.03	.72	1.91-5.00
T1 BLA–sgACC	.38	.21	-.13-.82
T2 BLA–sgACC	.38	.19	-.15-.88
T1 CMA–sgACC	.22	.19	-.43-.76
T2 CMA–sgACC	.24	.18	-.20-.73
T1 Depressive Symptoms	.12	.13	0-.65
T2 Depressive Symptoms	.13	.15	0-.65
T3 Depressive Symptoms (During Pandemic)	.35	.19	0-.85

Note. T1=Time 1; T2=Time 2; T3=Time 3; BLA=basolateral amygdala; CMA=centromedial amygdala; sgACC=subgenual anterior cingulate cortex. Depressive symptoms at all time points presented as proportion of maximum scale scores.

Figure 1

Note. Regions of interest (ROIs) including (a) basolateral amygdala (BLA; yellow) and centromedial amygdala (CMA; red) and (b) subgenual anterior cingulate cortex (sgACC; red), used in the examination of amygdala–sgACC resting state functional connectivity.

Figure 2

Note. Path analysis model of the concurrent and longitudinal associations among Time 1 parenting, Time 1 and Time 2 BLA–sgACC connectivity, and Time 1, Time 2, and Time 3 depressive symptoms (during the pandemic). BLA=basolateral amygdala; sgACC=subgenual anterior cingulate cortex; CDI=Child Depression Inventory; CES-DC=Center for Epidemiological Studies Depression Scale for Children. False Discovery Rate Adjusted *** $p < .001$, ** $p < .01$, * $p < .05$, $^\dagger p = .063$.