

Understanding the Effects of the COVID-19 Pandemic on Youth Psychopathology: Genotype–Environment Interplay

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ABSTRACT

BACKGROUND: Adversity has consistently been found to predict poor mental health outcomes in youth. Perhaps the most omnipresent form of adversity in the past several decades has been the coronavirus pandemic of 2020, a global health crisis linked to elevated rates of numerous forms of youth psychopathology. The ongoing nature of the pandemic renders it critical to identify the mechanisms underlying its effects on mental health.

METHODS: The current study examines pandemic-related disruption across multiple domains (e.g., home life, finances) as an etiologic moderator of several common forms of youth psychopathology. Participants were 637 adolescent twin pairs from the Twin Study of Behavioral and Emotional Development in Children (TBED-C). Mothers reported on disruption experienced by the family, using the Epidemic-Pandemic Impacts Inventory.

RESULTS: A series of biometric genotype-by-environment interaction models revealed that disruption augmented the nonshared environmental contributions to emotional distress and conduct problems but had little effect on the etiology of attention-deficit hyperactivity problems.

CONCLUSIONS: Our results indicate that identical and fraternal twin similarity in both emotional symptoms and conduct problems decreased with greater disruption, such that children in the same family became less alike, and did so regardless of their degree of genetic resemblance. Put differently, each twin sibling appeared to have their own idiosyncratic experience of pandemic-related disruptions, with downstream consequences for their mental health.

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The ongoing COVID-19 pandemic has had devastating effects on many domains of life across the globe. Of particular concern is the pandemic's impact on youth mental health. Indeed, prior work indicates a sharp increase in psychiatric diagnoses among young people during the pandemic [e.g., (1)]. While the deleterious effect of the pandemic on mental health has become increasingly clear, the mechanisms underlying this effect have not. We sought to clarify both how and why the pandemic has contributed to such a rapid spike in youth psychiatric diagnoses.

Adversity and Youth Mental Health

Past work has indicated that adversity has a profound effect on youth mental health, particularly when it involves exposure to multiple, co-occurring adverse events [e.g., (2,3)]. Given that the pandemic represents an abrupt, widespread shift in environmental conditions that likely exacerbated pre-pandemic stressors, we would expect its implications for youth mental health to be far-reaching. Consistent with this expectation, youth have reported elevated depression, anxiety, and post-traumatic stress disorder symptoms since the pandemic began, as well as deterioration in overall well-being (1,4,5), heightened loneliness and anger, and concern about maintaining peer relationships (6,7). The virtual mode of education

has also affected mental health. Children and adolescents have reported increased worry about academics and difficulty concentrating on schoolwork, with particularly pronounced effects for youth with attention-deficit/hyperactivity disorder (ADHD) (6,8).

The effects of the pandemic on youth mental health have thus been profound. One question that remains, however, is how the pandemic has exerted these effects on mental health. Extant research suggests that the effects of adverse experiences go beyond phenotypic predictions to alter the underlying genetic and environmental contributions. The specific manifestation of this etiologic moderation can take several forms. For instance, Burt *et al.* (9) examined school-aged twin pairs and found that neighborhood disadvantage altered the etiology of conduct problems, with family environmental influences predominating in disadvantaged neighborhoods and genetic influences predominating in low-risk neighborhoods (9,10,11). Such results are typically interpreted as evidence of a bioecological genotype-by-environment ($G \times E$) interaction, which posits that genetic influences are best expressed in "average, expectable" environments (12), whereas environmental influences, whether shared by family members or unique to each individual, predominate under adverse conditions.

However, not all forms of adversity operate through environmental mechanisms. In other cases, adversity has been found to accentuate genetic risk for psychopathology. For example, Hicks *et al.* (13) examined exposure to several forms of environmental adversity (e.g., parent-child conflict, stressful life events) and found that they augmented genetic contributions to adolescent antisocial behavior and substance use. This pattern of results is consistent with the predictions of the diathesis-stress model, which proposes that genetic vulnerabilities to psychopathology will increase in high-risk contexts (14). Such predictions stand in direct contrast to the bioecological model discussed above.

Current Study

We sought to clarify the mechanism(s) underlying the dramatic increase in youth psychiatric symptoms during the pandemic. Using a sample of 637 families of adolescent twins who were reassessed in summer 2020, we examined pandemic-related disruption as an etiologic moderator of youth psychopathology (emotional distress, conduct problems, and ADHD, based on parent report). Because participants had completed a similar assessment of psychopathology before the pandemic, we were also able to investigate whether pandemic-related disruption altered genetic and/or environmental influences that were already active prior to onset of the pandemic, or alternatively, whether disruption altered novel genetic and/or environmental influences that were not present at the first assessment. We further wanted to evaluate the competing hypotheses presented by the bioecological (i.e., environmental influences should increase) and the diathesis-stress (i.e., genetic influences should increase) models of the etiology of psychopathology. While there is a growing body of research examining stressors as etiologic moderators of youth behavioral outcomes, we know of no studies specifically evaluating how exposure to natural disasters might shape etiology. We thus hypothesized that pandemic-related disruptions would alter the etiology of youth psychopathology but did not have specific hypotheses regarding the form of G×E interaction observed.

METHODS AND MATERIALS

Participants

Families participated in the Twin Study of Behavioral and Emotional Development in Children (TBED-C), a sample within the population-based Michigan State University Twin Registry (MSUTR) (15). The TBED-C includes both a population-based subsample and an independent at-risk subsample that required that participating families lived in modestly to severely disadvantaged United States Census tracts [for more details, see (15)]. Eligible families in the TBED-C ($N = 995$) were recruited to participate in the current COVID-focused study. Our response rate from recruited families was 89.5%, of which 751 families ultimately completed their assessment. For this study, we restricted our analyses to 637 families who participated between May and July 2020, both to standardize COVID-19 conditions across families and to assess child mental health immediately following the initial surge of cases in

Michigan during spring 2020 (<https://covid.cdc.gov/covid-data-tracker/>).

Mean household income for participating families was \$70,056 (SD = \$41,485), which is below the current median income for families in Michigan (\$79,594). Participating twins ranged in age from 11 to 22 years (mean = 16.88 years, SD = 2.48 years; 52% female). Families identified as White, 84%; Black, 7.2%; Hispanic, 1%; Native American, 1.3%; and Asian, 0.9%. There were 121 monozygotic (MZ) male twin pairs, 124 dizygotic (DZ) male pairs, 134 MZ female pairs, 135 DZ female pairs, and 123 DZ opposite-sex pairs.

Measures

COVID-19 Disruptions. Pandemic-related disruption was assessed via the Epidemic-Pandemic Impacts Inventory (EPII) (16), which was completed by the participating parent (almost exclusively the mother). The EPII contains eight subscales assessing detrimental effects of the pandemic across domains (Work and Employment, Education and Training, Home Life, Social Activities, Economic, Emotional Health and Wellbeing, Physical Health Problems, and Physical Distancing and Quarantine). All items were dichotomous (0 = no, 1 = yes) and referred to changes that had occurred since the COVID-19 pandemic began (e.g., “increase in work responsibilities”). Because factor analysis indicated that the eight negative subscales were captured by a single factor, we computed a single sum score for each family, representing pandemic-related disruption across domains ($\Omega = 0.99$). Raw sum scores ranged from 0 to 36 (mean = 14.87, SD = 5.82) in our sample and were available for 100% of participating families. As nearly all respondents were mothers, and the majority identified as White, tests of measurement invariance were not conducted.

Youth Psychopathology: COVID-19 Assessment. The participating parent from each family completed the Strengths and Difficulties Questionnaire (SDQ) (17), a 25-item measure in which parents rate the extent to which a series of statements describe their child's behavior during the past 6 months using a 3-point scale (0 = not true to 2 = certainly true). We examined the three psychopathology scales: Emotional Symptoms (e.g., many fears; $\Omega = 0.79$), Conduct Problems (e.g., lies, cheats; $\Omega = 0.67$), and Hyperactivity/Inattention (e.g., fidgety; $\Omega = 0.82$). Data were available for 99% of the sample ($n = 1271$ twins).

Youth Psychopathology: Prior Assessment. All participating families had completed at least one prior assessment. We used participants' most recent assessment prior to the COVID-19 assessment to examine continuity and change in symptom presentation. The timing of the most recent assessment varied across participants. Nearly half ($n = 582$ twins) were most recently assessed as part of the TBED-C (6–11 years of age). Other participants ($n = 562$ twins) in TBED-C were assessed more recently through the Michigan Twin Neurogenetics Study (MTwiNS), which comprised two follow-up assessments of the TBED-C twins during adolescence. Finally, a small handful of participants ($n = 130$) were assessed most recently as part of the

Michigan Twins Project (MTP), a population-based registry within the MSUTR, out of which TBED-C families were originally recruited and which has begun its own series of reassessments (see Figure S1 for a flow chart depicting our sample). Ages at the prior assessment ranged from 6 to 19 years (mean = 11.30 years, SD = 4.04 years; i.e., an average of 5 years before the current assessment, ranging from <1 year to 12 years).

The participating parent from each family completed the SDQ as part of the MTP and the Child Behavior Checklist (18) as part of the TBED-C and MTwiNS. To maximize comparability between the respective measures, we constructed three scales with five items each from the Child Behavior Checklist that were analogous to those on the SDQ (see Supplemental Methods for more details). Data were available for 100% of participants.

Data Analytic Plan

Classical twin studies leverage the differing degrees of genetic similarity between MZ twins, who share 100% of their segregating genes, and DZ twins, who share 50% of their segregating genes on average. This difference enables us to estimate the relative genetic and environmental contributions to the variance within observed behaviors, or phenotypes. Phenotypic variance is decomposed into three components: additive genetic (A), shared environmental (C) (experiences that contribute to sibling resemblance, e.g., similar parenting), and nonshared environmental (E) (experiences that

differentiate siblings). The latter include differential treatment by parents, different peer groups, and different perceptions of the same event (i.e., experiences that are objectively shared by siblings, such as parental divorce, may impact each child idiosyncratically and therefore be a nonshared environmental influence) (19). Unsystematic measurement error is also included in E [see (20) for additional information on twin studies].

We first used a standard univariate twin model to estimate genetic and environmental influences on each psychopathology scale at both assessments. Next, we fitted the univariate G×E model (21) to the COVID-19 psychopathology data, shown in Figure 1A, to estimate the extent to which the genetic and environmental etiology of each psychopathology scale shifted with increasing levels of pandemic-related disruption. Disruption was first entered in a means model of the outcome. Moderation was then modeled on the residual variance (i.e., variance that does not overlap with disruption). The least restrictive of these models allows for linear moderation of A, C, and E contributions. We then fitted the no-moderator model along with relevant submodels depending on the results of the full ACE moderation model, constraining the linear moderators to be 0 and evaluating the reduction in model fit. The estimates from these models allowed us to establish the presence or absence of etiologic moderation for a given form of psychopathology, and to determine whether the observed moderation was more consistent with the predictions of the diathesis-stress model (i.e., an increase in genetic influences with greater disruption, such that genetic vulnerabilities/

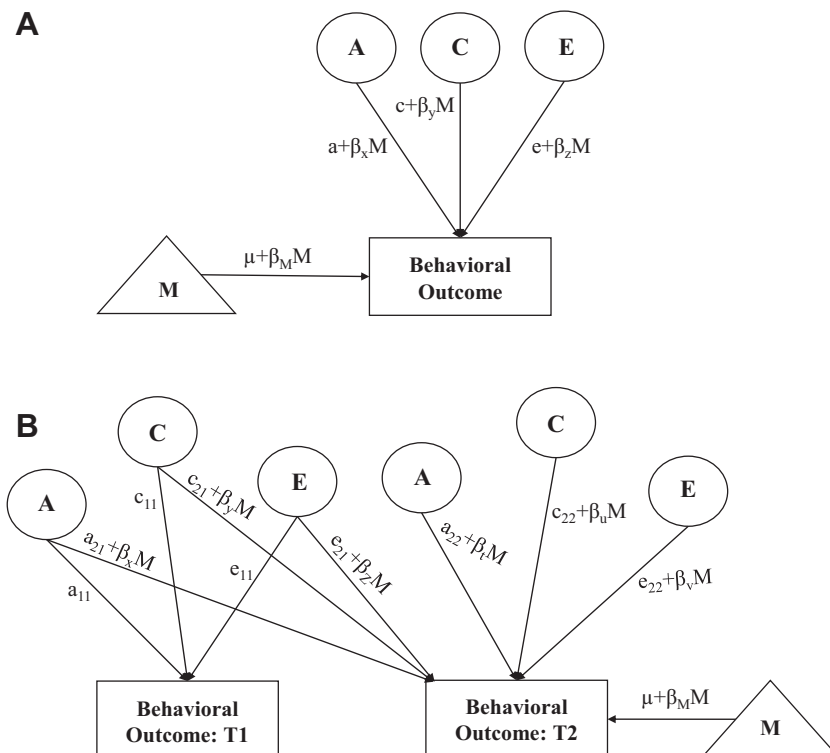


Figure 1. (A) Path diagram of the full linear moderation model (21). A, C, and E represent genetic, shared environmental, and nonshared environmental contributions, respectively, while M represents the moderator. For ease of presentation, the co-twin variables and paths are omitted here, although they are estimated in the model. (B) Path diagram of a bivariate moderation model. Parameters a_{11} , c_{11} , and e_{11} represent the genetic and environmental contributions to the outcome at time 1 (T1). Parameters a_{21} , c_{21} , and e_{21} represent the genetic and environmental contributions at time 1 that overlap with those at time 2, whereas parameters a_{22} , c_{22} , and e_{22} refer to the unique contributions at time 2 (T2). Moderation can influence either the common variance or unique time 2 variance, or both.

predispositions were activated and/or amplified in response to stress) or the bioecological model (i.e., an increase in shared and/or nonshared environmental influences with greater disruption, such that deleterious environments have a greater effect on development under stressful conditions). To adjust for the fact that these models were fit three times (once for each form of psychopathology), we used a more stringent p value ($\alpha = 0.05/3$; $p < .016$).

Lastly, we leveraged the pre-pandemic (time 1) psychopathology scores in a bivariate $G \times E$ model (21) (see Figure 1B) to evaluate whether the components of variance unique to the COVID-19 assessment (time 2) were moderated by disruption or whether this moderation was observed for the components of variance that overlapped with those already present at the prior assessment (time 1). If moderation was observed for the variance common to both time points, this would suggest that disruption amplified (or suppressed) etiologic influences already present prior to the pandemic. If, by contrast, moderation was observed only for those influences unique to time 2, then one would conclude that disruption altered the etiologic influences that were not present at the first assessment. This approach allowed us to reach more specific conclusions about the mechanisms underlying observed moderation. Of note, because the bivariate $G \times E$ model suffers from low power, among other things (22), we made use of the univariate $G \times E$ to illuminate the presence of $G \times E$ and then clarified results with the bivariate $G \times E$, as recommended previously (23).

$G \times E$ analyses were conducted in Mplus version 8.0 (24) using full-information maximum likelihood estimation. When models are fitted to raw data, means, variances, and covariances are first freely estimated to obtain a baseline fit index (minus twice the log likelihood; $-2\ln L$). Fit was evaluated using the Akaike information criterion (25), Bayesian information criterion (BIC) (26), and sample-size-adjusted BIC (SABIC) (27). The best-fitting model was indicated by the lowest Akaike information criterion, BIC, and SABIC values, as well as nonsignificant change in χ^2 , for at least three of the four fit indices. Disruption was examined as a continuous sum score, with higher scores indicating greater disruption. For ease of interpretation, raw scores on the EPII were transformed to range from 0 to 1 prior to analysis by dividing the raw score by 36 (the highest level of disruption we observed). Because disruptions were assessed at the family level, twins were

necessarily concordant on the moderator. To adjust for positive skew, SDQ scale scores were logarithmically transformed prior to analysis to better approximate normality (see Table 1). In addition, twin age, sex, and race were regressed out of the psychopathology data, consistent with prior recommendations (28). Questionnaire type (SDQ or Child Behavior Checklist) was also regressed out of the time 1 data for the bivariate analyses. Finally, as it is recommended that unstandardized parameter estimates be presented in moderation models (21), the logarithmically transformed and residualized psychopathology scores were standardized to have a mean of 0 and a standard deviation of 1 prior to analysis to facilitate interpretation of the unstandardized values.

RESULTS

Descriptive statistics are presented in Table 1 and described in Supplemental Results. Standardized variance estimates from the univariate ACE model are presented in Table S1. Pandemic-related disruptions predicted time 2 psychopathology even after controlling for time 1 psychopathology (see Table 2), consistent with prior work pointing to the negative mental health consequences of the pandemic.

Modeling Results

As shown in the Supplement, preliminary evaluation of the twin intraclass correlations suggested that nonshared environmental influences likely increased with disruption for emotional symptoms and conduct problems, but not for hyperactivity/inattention (see Supplemental Results; Table 3). We confirmed these impressions through formal tests of etiologic moderation (21), using participating families' disruption sum scores as a continuous moderator. For emotional symptoms, the AE moderation model (allowing for moderation of both A and E, though only the latter was statistically significant) and E-moderation-only model fit the data equally well (Tables 4 and 5). In both models, nonshared environmental influences increased with greater disruption. Put another way, twin dissimilarity in their emotional symptoms during the pandemic increased with greater disruptions (see Figure 2A). By contrast, results revealed moderate-to-large genetic contributions, regardless of the level of disruption. Nonshared environmental contributions to conduct problems also increased with greater pandemic disruptions. The AE moderation model best fit the

Table 1. Descriptive Statistics for Youth Psychopathology Scores at Time 2 (COVID-19 Assessment) and Time 1 (Prior Assessment)

Assessment	Phenotype	Mean (SD)	Range	Skew Before/After Transformation	Phenotypic r With Disruption Sum Score (95% CI) ^a
COVID-19 (Time 2)	Emotional symptoms	2.05 (2.26)	0–10	1.23/0.19	0.19 (0.14–0.24) ^b
	Conduct problems	1.03 (1.46)	0–9	2.07/0.75	0.13 (0.08–0.18) ^b
	Hyperactivity/inattention	2.68 (2.51)	0–10	0.95/–0.15	0.15 (0.10–0.20) ^b
Prior Assessment (Time 1)	Emotional symptoms	1.25 (1.60)	0–9	1.62/0.53	0.10 (0.04–0.15) ^b
	Conduct problems	0.99 (1.36)	0–10	1.84/0.66	0.09 (0.04–0.15) ^b
	Hyperactivity/inattention	1.71 (2.17)	0–10	1.44/0.46	0.09 (0.04–0.14) ^b

^aPhenotypic r values indicate the correlation between psychopathology scores and disruption sum scores on the Epidemic-Pandemic Impacts Inventory.

^bCorrelation was significantly different from 0 at $p < .05$.

Table 2. Pandemic-Related Disruption and Pre-pandemic Psychopathology as Predictors of Psychopathology at the COVID-19 Assessment

	<i>b</i> (SE)	β	<i>t</i>
Time 2 Emotional Symptoms			
Time 1 emotional symptoms	0.54 (0.04)	0.38	14.83 ^a
Disruption	2.09 (0.36)	0.15	5.84 ^a
Time 2 Conduct Problems			
Time 1 conduct problems	0.49 (0.03)	0.45	18.27 ^a
Disruption	0.80 (0.23)	0.09	3.54 ^a
Time 2 Hyperactivity/Inattention			
Time 1 hyperactivity/inattention	0.63 (0.03)	0.54	23.25 ^a
Disruption	1.55 (0.36)	0.10	4.28 ^a

^aEstimate was significantly different from 0 at $p < .05$.

data (Figure 2B), although as above, the genetic moderator was not significant. Instead, genetic effects were large regardless of the level of disruption. Such results are consistent with a bioecological pattern of etiologic moderation for conduct problems and emotional symptoms by disruption, with greater environmental influences in high-risk contexts. For hyperactivity/inattention, by contrast, all moderators could be constrained to 0 (Figure 2C). In other words, the etiology of hyperactivity/inattention was impervious to pandemic-related disruptions.

Given these results, we conducted a series of longitudinal (bivariate) analyses to clarify how disruption altered the etiologies of emotional symptoms and conduct problems (see Table 6). These analyses allowed us to separately estimate moderation on the components of variance that overlapped across times 1 and 2 as well as those unique to time 2. For emotional symptoms, the moderation of nonshared environmental variance common to time 1 and time 2 could be constrained to 0 without a decrement in model fit compared with the full model. By contrast, the nonshared environmental moderation unique to time 2 could not be constrained to 0, indicating that disruption altered child-specific environmental influences on emotional symptoms unique to time 2 (see Figure S2A). Likewise, for conduct problems, the additive genetic and the nonshared environmental moderation of variance common to time 1 and time 2, as well as the additive genetic moderation unique to time 2, could be constrained to 0, while the nonshared environmental moderation unique to time 2 could not. Collectively, these results suggest that disruption augmented nonshared environmental influences on conduct problems that were not yet present at time 1 (see Figure S2B).

DISCUSSION

We sought to illuminate how the pandemic influenced current symptoms of youth psychopathology, with a focus on possible G×E processes. To our knowledge, this is the first study of pandemic-related stress, or of any natural disaster, to incorporate a behavioral genetic twin design. Moreover, our analysis of psychopathology both subsequent and prior to the pandemic allowed us to better establish the direct link between pandemic-related stress and the recent spike in youth psychiatric diagnoses. Our results revealed that pandemic-related disruptions altered the etiologies of two of the three forms of psychopathology under study. Both emotional symptoms and conduct problems were subject to nonshared environmental moderation, such that these environmental influences were amplified with increasing disruption. These results are consistent with a bioecological pattern of moderation (i.e., stress amplifies environmental, rather than genetic, effects), with child-specific (but not family-wide) environmental influences becoming more salient in high-risk pandemic-related contexts. Pandemic-related disruption thus appears to contribute to sibling differentiation for psychopathology rather than resemblance.

Our finding of child-specific (but not family-wide) environmental moderation by pandemic-related stressors may appear counterintuitive given the increased time spent at home, an objectively shared environmental experience, during the pandemic. Moreover, the twins are necessarily concordant on parental reports of pandemic-related disruption, since these were assessed at the family level. Not all family-wide (or shared environmental) experiences within the home, however, serve to increase sibling similarity. Goldsmith distinguished between objective and effective environmental influences, noting that

Table 3. Twin Intraclass Correlations for Each Form of Psychopathology at Lower and Higher Disruption, Measured via the Epidemic-Pandemic Impacts Inventory

Phenotype	Lower Disruption				Higher Disruption			
	<i>r</i> MZ	<i>n</i>	<i>r</i> DZ	<i>n</i>	<i>r</i> MZ	<i>n</i>	<i>r</i> DZ	<i>n</i>
Emotional Symptoms	0.39 ^a	142	0.17 ^a	196	0.28 ^a	113	0.09	185
Conduct Problems	0.39 ^a	142	0.17 ^a	196	0.26 ^a	113	0.09	184
Hyperactivity/Inattention	0.20 ^a	142	0.10	196	0.36 ^a	113	0.10	186

DZ, dizygotic twin pair group; MZ, monozygotic twin pair group.

^aCorrelation was significantly different from 0 at $p < .05$.

Table 4. Biometric G×E Fit Indices for Univariate Models

Phenotype	-2lnL	χ^2 (df)	AIC	BIC	SABIC
Emotional Symptoms					
Linear ACE moderation	3519.06	-	3535.07	3570.72	3545.32
Linear AE moderation ^a	3519.06	0.00 (1)	3533.07	3564.26	3542.04
Linear A moderation	3530.56	11.50 ^b (2)	3542.56	3569.30	3550.25
Linear E moderation ^a	3522.60	3.54 (2)	3534.61	3561.35	3542.30
No moderation	3530.96	11.90 ^b (3)	3540.95	3563.24	3547.36
Conduct Problems					
Linear ACE moderation	3532.68	-	3548.68	3584.33	3558.93
Linear AE moderation ^a	3532.68	0.00 (1)	3546.68	3577.87	3555.65
Linear A moderation	3548.40	15.72 ^b (2)	3560.39	3587.13	3568.08
Linear E moderation	3540.14	7.46 (2)	3552.14	3578.88	3559.83
No moderation	3548.56	15.88 ^b (3)	3558.57	3580.85	3564.98
Hyperactivity/Inattention					
Linear ACE moderation	3564.24	-	3580.24	3615.89	3590.50
Linear AE moderation	3564.24	0.00 (1)	3578.24	3609.44	3587.21
Linear A moderation	3564.56	0.32 (2)	3576.57	3603.31	3584.26
Linear E moderation	3564.82	0.58 (2)	3576.81	3603.55	3584.50
No moderation ^a	3564.82	0.58 (3)	3574.81	3597.10	3581.22

A, additive genetic component; AIC, Akaike information criterion; BIC, Bayesian information criterion; C, shared environmental component; E, nonshared environmental component; G×E, genotype-by-environment interaction; L, likelihood; SABIC, sample-size-adjusted Bayesian information criterion.

^aIndicates the best-fitting model(s) for a given phenotype.

^bSignificant change in χ^2 at $p < .016$.

experiences that are objectively shared by siblings (e.g., moving to a new neighborhood) may impact each child in unique ways (29). Such experiences would be part of the effective nonshared environment and contribute to

differentiation among children raised in the same family regardless of their genetic resemblance. Our results suggest that pandemic-related disruption falls into this category of environmental influence for youth conduct problems and

Table 5. Unstandardized Path and Moderation Parameter Estimates for the Linear Moderation Models

Phenotype	Paths			Linear Moderators		
	a	c	e	A1	C1	E1
Emotional Symptoms						
Linear ACE moderation	0.77 ^a	0.00	0.54 ^a	-0.55	0.00	0.64 ^a
Linear AE moderation ^b	0.77 ^a	0.00	0.54 ^a	-0.55	-	0.64 ^a
Linear A moderation	0.50 ^a	0.00	0.81 ^a	0.13	-	-
Linear E moderation ^b	0.57 ^a	0.00	0.63 ^a	-	-	0.41 ^a
No moderation	0.55 ^a	0.00	0.81 ^a	-	-	-
Conduct Problems						
Linear ACE moderation	0.87 ^a	0.00	0.54 ^a	-0.80	0.00	0.66 ^a
Linear AE moderation ^b	0.87 ^a	0.00	0.54 ^a	-0.80	-	0.66 ^a
Linear A moderation	0.54	0.00	0.81 ^a	0.09	-	-
Linear E moderation	0.60 ^a	0.00	0.65 ^a	-	-	0.35
No moderation	0.58 ^a	0.00	0.81 ^a	-	-	-
Hyperactivity/Inattention						
Linear ACE moderation	0.61 ^a	0.00	0.80 ^a	-0.24	0.00	0.11
Linear AE moderation	0.61 ^a	0.00	0.80 ^a	-0.24	-	0.11
Linear A moderation	0.56 ^a	0.00	0.85 ^a	-0.11	-	-
Linear E moderation	0.51 ^a	0.00	0.85 ^a	-	-	0.01
No moderation ^b	0.51 ^a	0.00	0.85 ^a	-	-	-

Shared environmental contributions were not constrained to be 0, but were observed to not be significantly different from 0, in the biometric twin analyses.

A, additive genetic component; C, shared environmental component; E, nonshared environmental component.

^aParameter is significant at $p < .016$.

^bIndicates the best-fitting model(s) for a given phenotype.

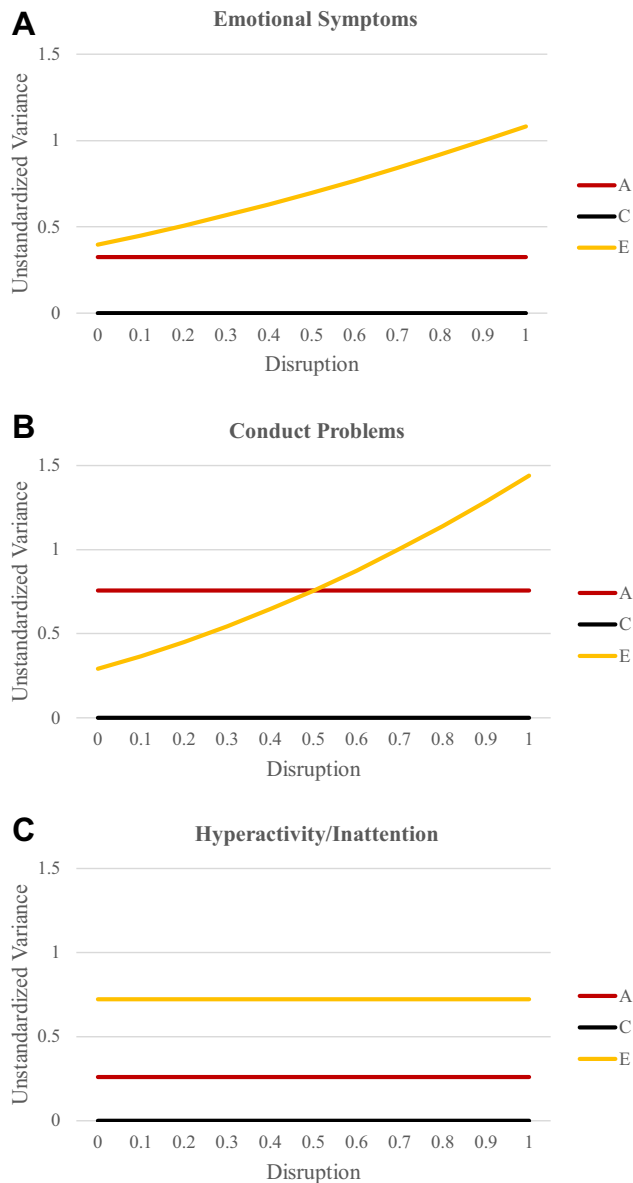


Figure 2. Etiologic moderation of (A) emotional symptoms, (B) conduct problems, and (C) hyperactivity/inattention by pandemic-related disruption. Disruption was measured as a continuous sum score based on parent reports on the Epidemic-Pandemic Impacts Inventory. Red, black, and yellow lines represent genetic (A), shared environmental (C), and nonshared environmental (E) influences, respectively.

symptoms of emotional distress. On both measures, MZ and DZ twin similarity decreased with greater disruption, meaning that children in the same family became less alike, and did so regardless of their degree of genetic resemblance. Put differently, each twin sibling appeared to be experiencing pandemic-related disruptions in their own unique way.

Not only does the pandemic appear to have affected the etiologies of emotional symptoms and conduct problems in similar ways, but the underlying mechanism appears to be

consistent across the two phenotypes. Results from the bivariate analyses indicate that disruption altered novel non-shared environmental influences on both emotional symptoms and conduct problems, that is, twin-specific environmental influences that emerged after the first assessment. At the first assessment, most participants were in either middle childhood or early adolescence (8–14 years of age). Previous literature indicates that the etiologies of emotional symptoms and conduct problems are in flux throughout the first 2 decades of life [e.g., (30)]. The current study contributes to this literature by showing that the pandemic altered environmental contributions that were not present early in development and thus contributed to the flux of emotional symptoms and conduct problems during this critical developmental period.

However, the pattern of child-specific environmental moderation for emotional distress and conduct problems was not observed for hyperactivity/inattention. Instead, its etiology appeared to remain constant regardless of the level of disruption, consistent with research implicating ADHD as a neuropsychiatric diagnosis primarily subject to genetic influences [e.g., (31)]. These null findings are unlikely to be a function of low statistical power, since prior G×E data simulations (32) have indicated that researchers have substantial power to detect E moderation in particular. Thus, despite the significant phenotypic association between disruption and scores on hyperactivity/inattention, disruption does not appear to moderate its nonshared environmental etiology.

This study has several key strengths, including its genetically informed analytic design and its longitudinal analysis of psychopathology before and during the pandemic. However, there are some limitations. First, scores on the EPII were significantly correlated with psychopathology assessed before the pandemic, suggesting that preexisting vulnerabilities present in participants' lives shaped their experiences of COVID-19. That said, EPII scores continued to predict psychopathology at the COVID assessment even when controlling for pre-pandemic psychopathology. Although between-person processes cannot be fully distinguished from within-person change with only two time points (33), such findings nevertheless suggest a more direct link between disruption and psychopathology.

Second, twins were assessed in adolescence and emerging adulthood. As such, our results may not generalize to other age groups. Given age-related increases in mobility and engagement with peers and decreases in parental supervision, adolescents may experience the pandemic differently from younger children. Although we did examine overlap and discontinuity in etiology across two time points, our study was not able to examine pandemic-related disruption during childhood, since this generally preceded the pandemic in our sample. Relatedly, our sample included emerging adults (18–22 years of age) who may have been living independently from their parents. As such, parental reports of psychopathology and pandemic-related disruption may be less accurate for these participants. To address this concern, we ran supplemental analyses restricted to participants under age 18 ($n = 417$ pairs). Results were consistent with those obtained in the full sample (see Tables S2 and S3).

Next, although our sample comprises both a population-based subsample and an at-risk subsample of youth from impoverished neighborhoods, we were unable to examine

Table 6. Biometric G×E Fit Indices for Bivariate Models

Phenotype	−2lnL	χ^2_1	AIC	BIC	SABIC
Emotional Symptoms					
Full bivariate ACE moderation	6361.34	–	6403.34	6496.93	6430.26
Common E moderator fixed to 0	6365.10	3.76	6405.10	6494.24	6430.74
Time 2 E moderator fixed to 0 ^a	6370.38	9.04 ^b	6410.38	6499.52	6436.02
Conduct Problems					
Full bivariate ACE moderation	6187.58	–	6229.57	6323.17	6256.49
Common A moderator fixed to 0	6187.62	0.04	6227.61	6316.75	6253.25
Common E moderator fixed to 0	6187.62	0.04	6227.61	6316.75	6253.25
Time 2 A moderator fixed to 0	6188.62	1.04	6228.62	6317.76	6254.26
Time 2 E moderator fixed to 0 ^a	6197.58	10.00 ^b	6237.57	6326.71	6263.21

A, additive genetic component; AIC, Akaike information criterion; BIC, Bayesian information criterion; C, shared environmental component; E, nonshared environmental component; L, likelihood; SABIC, sample-size-adjusted Bayesian information criterion.

^aSignificant decrement in fit when a given moderator was constrained to 0.

^bSignificant change in χ^2 at $p < .016$.

disruption and socioeconomic deprivation as joint etiologic moderators due to low power. Subsequent studies should examine whether the moderating effects of disruption are amplified (or suppressed) in disadvantaged contexts. Finally, MZ twin correlations were more than twice as large as their corresponding DZ correlations at higher disruption for all three forms of psychopathology and at lower disruption for emotional symptoms and conduct problems, raising concerns that the ACE model may only imperfectly fit these data (21). To address this concern, we ran supplemental analyses using a modified twin correlation model (34), which allows the moderator to directly modify the MZ and DZ twin correlations rather than the ACE components. Results from the modified twin correlation model were consistent with those observed above (see [Supplemental Results](#) and [Table S4](#)), indicating that our results are robust to G×E modeling strategy.

Despite these limitations, our study identified a consistent moderating effect of pandemic-related disruption on the etiologies of two forms of youth psychopathology. Moreover, this pattern of moderation was specific to emotional distress and conduct problems, as there was no evidence of moderation for ADHD. Such findings contribute to our understanding of why and how the ongoing pandemic has had such a detrimental effect on youth mental health. Specifically, for at least two common forms of psychopathology, disruption appears to amplify child-specific environmental influences that make youth in the same family less alike. What might this look like in practice? There is some evidence to suggest that differential parenting increases in the context of stress, for example, with both socioeconomic stress and marital dissatisfaction predicting greater differences in the level of positivity that parents show to each child (35). Future work should evaluate the extent to which differential parenting might account for our findings.

Evolutionary theories of person–environment mismatch can also help us understand these results. In particular, evolutionary theories posit that rapid, extensive environmental changes could render previously adaptive behaviors maladaptive [e.g., (36)]. In our predominantly adolescent sample, for example, healthy sociability may have in fact increased susceptibility to depression and anxiety, as well as to externalizing psychopathology, following the rapid decrease in

opportunities to socialize as the pandemic began. As another possibility, conscientiousness regarding one's physical health could contribute to high levels of illness anxiety, which may be both adaptive in preventing illness and maladaptive to one's mental health. We also note that the all-encompassing nature of pandemic-related changes may have provided children in the same family with ample opportunity to respond to these unusual circumstances in their own idiosyncratic ways.

Regardless of the interpretation, however, our results indicate that pandemic-related increases in emotional distress and conduct problems may be best addressed with interventions tailored to the individual, rather than to the entire family. In sum, even for children who have identical genes and reside in the same home, the pandemic appears to differentiate their mental health outcomes, highlighting the idiosyncratic nature of the ongoing pandemic as a potent risk factor for youth psychopathology.

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COVID-19 Pandemic and Youth Psychopathology

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