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EMPIRICAL ARTICLE

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Tracking depressive and anxious symptoms during the first year of COVID-19: The search for moderators

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Abstract

A growing body of research suggests that the COVID-19 pandemic did not cause the severe and extensive mental health crisis predicted by some experts. However, this does not mean that everyone was resilient. The purpose of this study was to try to identify subgroups of people that may have experienced more severe and negative trajectories of symptoms during this time. To this end, we examined a host of individual difference factors (e.g., age, gender, race, country, parental status, medical conditions, lost wages, perceived support, initial symptom levels, and cognitive vulnerability) using a 1-year longitudinal design with 8 time points and participants (n = 233) from over 20 countries. We were unable to identify a single moderator associated with a robust and increasingly negative trajectory of depressive and anxious symptoms throughout the COVID interval. These results underscore the need for better theories of mental illness, stronger research designs that do not rely on simple cross-sectional between-group comparisons, and more caution when predicting mental health crises.

KEYWORDS
anxiety, cognitive vulnerability, COVID-19, depression, mental health, pandemic

1 | INTRODUCTION

Near the beginning of the pandemic a group of researchers (Gruber et al., 2021) wrote a call-to-action article about the unparalleled effects COVID was going to have on rates of mental illness. They called for the "identi-

fication of psychologically active interventions that can be delivered at scale (cf. fluoride in the water) to build resilience and reduce risks among large segments of the population" (p. 421). However, 3 years later, research shows that there has not been a population-wide mental health crisis (i.e., there have not been increasingly severe trajectories of mental health problems). The Lancet's COVID-19 Commission Mental Health Task Force (Aknin et al., 2021, 2022) reviewed data from nearly a thousand studies from approximately 100 countries and concluded: "A clear and consistent body of evidence suggests that psychological distress increased during the early months of the COVID-19 pandemic and that most (but not all) facets returned to pre-pandemic levels by mid-2020. While some components of subjective well-being showed signs of strain (e.g., increasing negative emotions), the data also reveal notable signs of resilience in life satisfaction, loneliness, social connection, and suicide" (p. 919).

So, what now? We suspect that researchers will look for moderators. Can we discover at least some groups of people for which there was a robust and increasingly negative trajectory of COVID-related mental health problems? To investigate this question, we used a year-long prospective research design (March 2020–March 2021) with adults from around the world. We tested a host of potential moderating demographic variables that were hypothesized to affect mental health outcomes in response to COVID such as age, race, parental status, medical conditions, wages, perceived support (Gruber et al., 2021); we also tested moderators identified in prior work such as gender, geography, and initial symptom levels (Hammen et al., 1986; Henríquez-Sánchez et al., 2014; Nolen-Hoeksema, 1987). Finally, we tested one theory-based variable: cognitive vulnerability to depression (Abramson et al., 1989, 1999). This is the only variable for which there is a formal theory (the hopelessness theory) that specifies the etiological sequence by which the factor confers risk for depression.

2 | METHOD

2.1 | Procedure

Participants were recruited via an online platform (Prolific; Palan & Schitter, 2018). They completed measures every month for the first 4 months of the pandemic (March 2020–June 2020) and then every other month for the rest of the year (August 2020–March 2021). All procedures were approved by the University's human subject review board.

2.2 | Participants

Participants were 233 adults (94 female, 137 male, 2 other-identified) from 22 countries (49% were from the United States and the United Kingdom) on four continents (48% North America, 40% Europe, 9% Australia, 3% South America). Participants were relatively homogenous (73% self-reporting as White, 3% Black, 12% Latin/Hispanic, and 6% Asian, 2% South East Asian, and 2% "Other") and young (*M* age = 28.80, median age = 26; SD = 11, range = 18–72). See Supporting Information S1 for information on power, recruitment, and attrition.

2.3 | Measures

2.3.1 | Baseline demographics

Participants reported their age, gender, race/ethnicity, and country of residence. Participants were also asked about their parental status, pre-existing medical conditions, lost wages, and perceived community support (see Supporting Information S1 for response coding).

2.3.2 | Cognitive vulnerability

The Cognitive Style Questionnaire (CSQ; Haeffel et al., 2008) was used to measure cognitive vulnerability to depression. Participants are presented with six hypothetical negative events and then make ratings (1–7) on dimensions of stability/globality; consequences; and the self-worth implications. An individual's CSQ score is his/her average rating for the three dimensions across the six scenarios (with these composite average scores ranging from 1 to 7). The CSQ has excellent psychometric properties (Haeffel et al., 2008). Coefficient alpha in the current study was 0.81.

2.3.3 | Depressive and anxious symptoms

Participants were administered the "depression specific" and "anxiety specific" subscales of the Mood and Anxiety Symptom Questionnaire (see Supporting Information S1), a widely used and psychometrically sound questionnaire (e.g., Clark & Watson, 1991; Watson et al., 1995). Coefficient alpha for both subscales was >0.80 across all time points.

2.4 | Data analytic approach

We tested hypotheses using mixed effect models. The dependent variables were depressive and anxious symptoms, respectively. The fixed factors were age, gender, race, country, parental status, pre-existing medical condition, lost wages, perceived community support, initial levels of depressive and anxious symptoms, and cognitive vulnerability, respectively. Participant was a random factor, continuous predictors were mean-centered, and time was tested for polynomial effects.

3 | RESULTS

3.1 | Time

3.1.1 | Depressive symptoms

There was a small, but significant, linear effect of time on depressive symptoms, $b = -1.70$, $SE = 0.79$, $t(1141) = -2.15$, $p = 0.03$, 95% CI: −3.26 to −0.15; omnibus test: $F(7,1125) = 2.37$, $p = 0.02$, R-squared marginal = 0.00. However, as shown in Figure 1, changes in depressive symptom levels only varied by about four points (range 67–71) on the 88-point scale (clinical cut-off = 76; Buckby et al., 2007). There were no other significant polynomial effects.

3.1.2 | Anxious symptoms

Anxious symptoms were stable over time (see Figure 1) with no significant increases or decreases throughout the year, $b = -0.82$, $SE = 0.48$, $t(1132) = -1.72$, $p = 0.09$, 95% CI: −1.75 to 0.12; omnibus test: $F(7,1119) = 1.00$, $p = 0.43$, R-squared marginal = 0.00; no significant polynomial effects. Overall levels of anxious symptoms never differed by more than two points on average (range 25–27) on the 68-point scale.

The next step was to identify subgroups that might have experienced particularly severe (or increasing) levels of symptoms. We report significant findings below; there were no effects for gender, race, pre-existing medical condition (depressive symptoms), parental status, country, perceived community support (anxious symptoms) or lost wages (see Supporting Information S1).

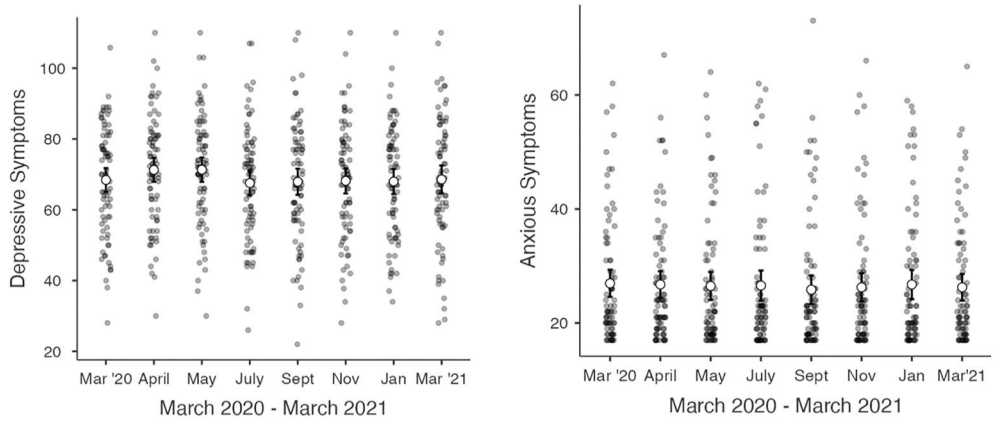

FIGURE 1 Observed scores for depressive symptoms (left) and anxious symptoms (right) as a function of time.

3.2 | Age

3.2.1 | Depressive symptoms

There was a medium sized main effect of age on depressive symptoms, $b = -0.19$, $SE = 0.08$, $t(222) = -2.41$, $p = 0.02$, 95% CI: −0.35 to −0.04; omnibus test: $F(1,222) = 5.81$, $p = 0.02$, R-squared marginal = 0.03. Younger participants reported significantly greater levels of depressive symptoms (M − 1 SD = 70.78, $SE = 1.25$) than older participants (M +1 SD = 66.23, $SE = 1.33$).

There was also a small time × age interaction effect, $F(7,1094) = 3.17$, $p = 0.003$. To probe the interaction, we followed an approach recommended by DeCoster et al. (2011) whereby we created artificial categorizations (after analyzing the data with continuous variables) to simplify presentation and interpretation of results. Specifically, we broke down age into statistically determined quartiles: 18–20, 21–25, 26–34, and >34 (the time × age interaction remained significant; $p = 0.002$). The rationale for using quartiles was to create equally sized groups that spanned the age range (see Supporting Information S1 for alternative age groupings). As shown in Figure 2, the younger age groups (18–25) showed greater levels of depressive symptoms at the start of the pandemic than the older age groups ($F[7,1085] = 3.66$, $p < 0.001$) with the 21–25 age group showing a slower recovery than the 18–21 group; however, by January the depressive scores of the 18–21 group were significantly lower than they were in April and May ($t = 3.74$, $p < 0.001$ and $t = 3.54$, $p < 0.001$, respectively). The interaction was characterized by a significant quadratic pattern with the youngest age group reporting an increase in symptoms the last 2 months while symptom levels remained level or declined for older participants, $b = 4.45$, $SE = 1.97$, $t(1096) = 2.89$, $p = 0.004$, 95% CI: 1.85–9.71. That said, the interaction effect should be interpreted with caution given the small effect and lack of clinically significant change in symptoms.

3.2.2 | Anxious symptoms

There was a small to medium sized effect of age on levels of anxious symptoms, $b = -0.13$, $SE = 0.06$, $t(222) = -2.35$, $p = 0.02$, 95% CI: −0.24 to −0.02; omnibus test: $F(1,221) = 5.39$, $p = 0.02$, R-squared marginal = 0.02. There was not a time × age interaction effect on levels of anxious symptoms, $F(7,1089) = 0.63$, $p = 0.73$. Younger participants reported greater levels of anxious symptoms (M − 1 SD = 27.64, $SE = 0.86$) than older participants (M +1 SD = 24.63, $SE = 0.92$; see Figure 3). To visual the main effect, we broke down age into quartiles: 18–20, 21–25, 26–34, and >34 (results were robust to grouping strategy). The 18–20 age group reported significantly greater levels of anxious symptoms

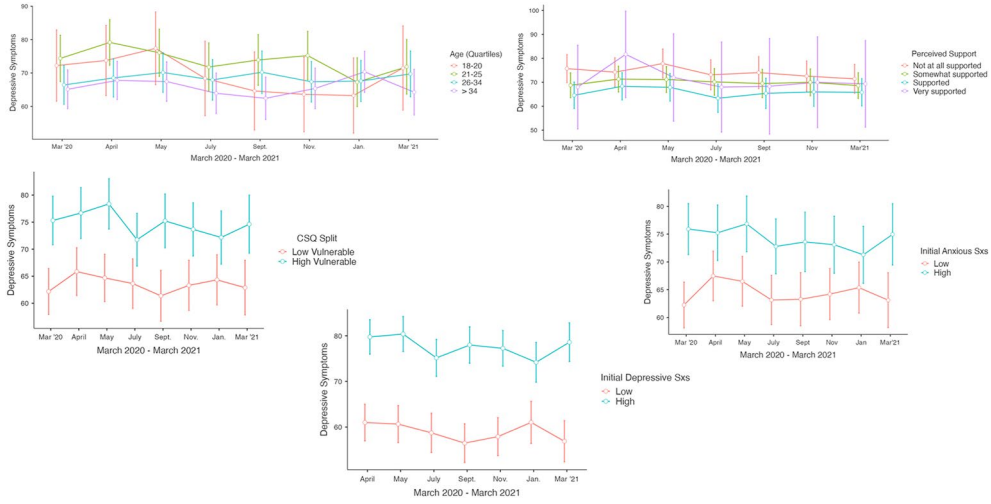

FIGURE 2 Depressive symptoms as a function of time and moderator.

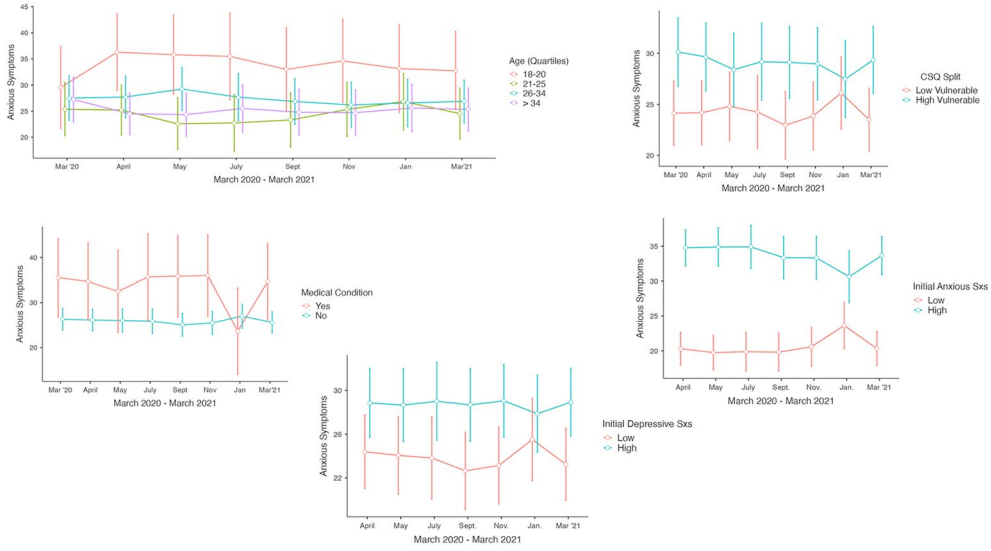

FIGURE 3 Anxious symptoms as a function of time and moderator.

than those aged 21–25 (t = 2.27, p = 0.03) and greater than 34 (t = 2.18, p = 0.03), but not those aged 26–34 (t = 1.65, p = 0.10). There were no significant polynomial effects.

3.3 | Pre-existing medical condition

3.3.1 | Anxious symptoms

There was a small, but statistically significant effect of pre-existing medical condition on levels of anxious symptoms, b = −3.75, SE = 1.79, $t(230)$ = −2.09, p = 0.04, 95% CI: −7.27 to −0.24; omnibus test: $F(1,230)$ = 4.38, p = 0.04, R-squared marginal = 0.02. Those with a pre-existing medical condition reported greater levels of anxious symptoms

(*M* = 29.45, *SE* = 1.66) than those without a pre-existing medical condition (*M* = 25.69, *SE* = 0.67). There was not a time × medical condition interaction on levels of anxious symptoms, *F*(7,1119) = 1.31, *p* = 0.24. There were no significant polynomial effects.

3.4 | Perceived community support

3.4.1 | Depressive symptoms

There was a small sized main effect of perceived support on levels of depressive symptoms, *b* = −7.55, *SE* = 2.69, *t*(235) = −2.81, *p* = 0.005, 95% CI: −12.82 to 2.28; omnibus test: *F*(3,233) = 3.55, *p* = 0.02, *R*-squared marginal = 0.04. There was not a time × community support interaction effect, *F*(21,1097) = 0.88, *p* = 0.62; see Figure 2. Participants who rated being "supported" (*M* = 66.07, *SE* = 1.60) or "very much supported" (*M* = 61.22, *SE* = 3.52) by their community reported significantly lower levels of depressive symptoms than those who rated their support as "somewhat" (*M* = 70.68, *SE* = 1.47) or "not at all supported" (*M* = 70.93, *SE* = 1.77). There were no significant polynomial effects.

3.5 | Cognitive vulnerability

3.5.1 | Depressive symptoms

There was a large significant effect of cognitive vulnerability on levels of depressive symptoms, *b* = 5.36, *SE* = 0.82, *t*(229) = 6.53, *p* < 0.001, 95% CI: 3.75–6.97; omnibus test: *F*(1,229) = 42.62, *p* < 0.001, *R*-squared marginal = 0.13. There was not a time × cognitive vulnerability interaction effect, *F*(7,1116) = 1.83, *p* = 0.08. As shown in Figure 2, those with high levels of cognitive vulnerability (visualized with median split) reported significantly greater levels of depressive symptoms (*M* + 1 SD = 74.2, *SE* = 1.17) than those with low levels of cognitive vulnerability (*M* − 1 SD = 62.90, *SE* = 1.19) throughout the year. There were no significant polynomial effects.

3.5.2 | Anxious symptoms

There was a medium sized significant effect of cognitive vulnerability on levels of anxious symptoms, *b* = 2.75, *SE* = 0.59, *t*(229) = 4.69, *p* < 0.001, 95% CI: 1.60–3.90; omnibus test: *F*(1,229) = 21.98, *p* < 0.001, *R*-squared marginal = 0.07. There was not a time × cognitive vulnerability interaction effect, *F*(7,1110) = 1.44, *p* = 0.19. As shown in Figure 3, those with high levels of cognitive vulnerability (visualized with median split) reported significantly greater levels of anxious symptoms (*M* + 1 SD = 28.97, *SE* = 0.84) than those with low levels of cognitive vulnerability (*M* − 1 SD = 23.31, *SE* = 0.85) throughout the year. There were no significant polynomial effects.

3.6 | Initial levels of depressive symptoms

3.6.1 | Depressive symptoms

There was a large effect of initial level of depressive symptoms on depressive symptoms, *b* = 17.12, *SE* = 1.37, *t*(239) = 12.50, *p* < 0.001, 95% CI: 14.43–19.80; omnibus test: *F*(1,239) = 156.27, *p* < 0.001, *R*-squared marginal = 0.32. As shown in Figure 2, those with high initial symptoms (visualized with median split) had significantly greater levels of depressive symptoms (*M* + 1 SD = 77.35, *SE* = 0.97) than those with low initial symptoms (*M* − 1 SD = 60.26, *SE* = 0.96) throughout the year.

There was also a small time × initial symptom interaction effect, $F(7,1130) = 7.83$, $p < 0.001$. The interaction pattern was best characterized by a significant quadratic effect ($b = 5.12$, $SE = 1.51$, $t[1132] = 3.38$, $p < 0.001$, 95% CI: 2.15–8.08), which can be seen with the high initial symptom group reporting an increase in depressive symptoms at the last time point (and the low initial symptom group reporting a decrease in symptoms).

3.6.2 | Anxious symptoms

There was a small to medium significant effect of initial depressive symptoms on levels of anxious symptoms, $b = 3.75$, $SE = 1.21$, $t(233) = 3.10$, $p = 0.002$, 95% CI: 1.37–6.12; omnibus test: $F(1,233) = 9.58$, $p = 0.002$, R-squared marginal = 0.04. There was not a time × initial symptom interaction effect, $F(7,1121) = 1.02$, $p = 0.42$. As shown in Figure 3, those with high initial symptoms (visualized with median split) had significantly greater levels of depressive symptoms (M + 1 SD = 28.12, $SE = 0.86$) than those with low initial symptoms (M − 1 SD = 24.38, $SE = 0.85$) throughout the year. There were no significant polynomial effects.

3.7 | Initial levels of anxious symptoms

3.7.1 | Depressive symptoms

There was a medium sized effect of initial anxious symptoms on levels of depressive symptoms, $b = 9.44$, $SE = 1.70$, $t(237) = 5.55$, $p < 0.001$, 95% CI: 6.11–12.77; omnibus test: $F(1,237) = 30.80$, $p < 0.001$, R-squared marginal = 0.10; there was not a time × initial symptom interaction, $F(7,1112) = 2.05$, $p = 0.05$. As shown in Figure 2, those with high initial anxious symptoms (visualized with median split) had significantly greater levels of depressive symptoms (M + 1 SD = 73.27, $SE = 0.1.19$) than those with low initial depressive symptoms (M − 1 SD = 63.89, $SE = 1.20$) throughout the year. There were no significant polynomial effects.

3.7.2 | Anxious symptoms

There was a large effect of initial level of anxious symptoms (on levels of anxious symptoms, $b = 11.80$, $SE = 0.93$), $t(237) = 12.66$, $p < 0.001$, 95% CI: 9.97–13.62; omnibus test: $F(1,236) = 160.16$, $p < 0.001$, R-squared marginal = 0.34; results also showed a small time × initial symptom interaction, $F(7,1123) = 8.50$, $p < 0.001$. As shown in Figure 3, those with high initial symptoms (visualized with median split) had significantly greater levels of anxious symptoms (M + 1 SD = 31.97, $SE = 0.65$) than those with low initial symptoms (M − 1 SD = 20.24, $SE = 0.66$) throughout year. The interaction was characterized by a significant sextic effect pattern, which was most pronounced at the January assessment ($b = 2.27$, $SE = 89$, $t[1115] = 2.55$, $p = 0.01$, 95% CI: 0.53–4.02) where those with high initial symptoms reported a sharp decrease in depressive symptoms and those with low initial symptoms reported an increase in depressive symptoms (after which both groups returned to near their baseline scores). There were no other significant polynomial effects.

4 | DISCUSSION

Our study corroborates a growing literature showing that people tend to be relatively resilient (Bonanno et al., 2011; Bryant et al., 2015; Galatzer-Levy et al., 2018; Johannesson et al., 2015; Wortman & Silver, 1989), even during a pandemic (Aknin et al., 2021, 2022). As noted by Aknin et al. (2021), "the astonishing resilience that most people

have exhibited in the face of the sudden changes brought on by the pandemic holds its own lessons. We learned that people could handle temporary changes to their lifestyle—such as working from home, giving up travel, or even going into isolation—better than some policy makers seemed to assume" (p. 5). That said, not everyone is equally resilient. The purpose of this study was to try to identify at-risk people by testing individual difference factors (i.e., potential moderators).

Results showed that there were some people who experienced significantly greater levels of depressive and anxious symptoms compared to the overall sample, namely, those who were younger (18–25), those with a pre-existing medical condition, those with initially high symptom levels, and those with high levels of cognitive vulnerability. However, we were unable to identify any moderators that predicted an increasing trajectory of depressive or anxious symptoms over the yearlong interval. In other words, the subgroups with greater symptoms did not seem to be unusually affected by the pandemic. They were already experiencing more mental health symptoms and difficulties than others, and that continued to be the case (i.e., the pandemic "raised all boats").

The study had both strengths and limitations. Strengths included the use of a year-long longitudinal design with eight time points, creating temporal precedence for the predictor variables. Further, the study was adequately powered to detect the differences of interest, and the participants were more diverse in terms of age and geography than most psychology studies (Clancy & Davis, 2019; Thalmayer et al., 2021). However, the study also had weaknesses. First, it was impossible to test all potential moderators and, thus, there may be factors (e.g., childcare availability, quarantine length) that did create crisis-like mental health problems. Second, there are several constraints on generalizability. Our sample was mainly white (73%) and Western, and we did not examine clinically significant forms of mental illness.

In conclusion, we were not able to identify individual difference factors associated with a robust and increasing trajectory of depressive and anxious symptoms during the first year of COVID. This underscores a problem—researchers have yet to identify many, if any, causal risk factors that are strong predictors of psychopathology to traumatic events (though is noteworthy that the only theory-based risk factor we tested, cognitive vulnerability, had the second largest effect size). It will be important for future research to develop stronger, more specific theories with greater predictive validity. Until then, researchers should be cautious about making predictions about widespread mental health crises.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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