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Event-related PTSD symptoms as a high-risk factor for suicide: longitudinal observational study

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There is long-standing controversy as to whether suicide risk in those who have survived a traumatic event is highest when the severity of the survivors' psychiatric condition is worst or when they begin to recover. To tackle this problem, we extracted psychiatric conditions from an online cohort of Japanese participants during the COVID-19 pandemic, at five time points (T1–T5). For 12,578 responses from 3,815 participants (mean age 47.1 years; 46.8% women), 3,508 psychiatric conditions were extracted in T1, 2,680 in T2, 2,562 in T3, 2,022 in T4 and 1,806 in T5. We then investigated whether extracted conditions could predict suicide rates in the full Japanese population in a time-specific manner. We found that COVID-19-related PTSD symptoms are associated with increased suicide rates ($P = 3.0 \times 10^{-6}$, Bayesian information criterion (BIC) = −23.69), and are of greater concern than depression ($P = 7.6 \times 10^{-4}$, BIC = −13.19) and anxiety symptoms ($P = 5.9 \times 10^{-3}$, BIC = −9.35). Furthermore, associations of psychiatric states with increased suicide rates are time specific ($P = 0.011$), suggesting that a population shows higher suicide risk when symptom severity is high. Event-related PTSD symptoms may help to identify groups at high risk of suicide and improve prevention policies.

Over 70% of all people confront a hazardous event, such as an earthquake, hurricane, violence, childhood abuse, war, traffic accident or pandemic, at some point in their lives[1–3]. An imperative in the context of hazardous events is suicide prevention. In 1893, suicide rates in England and Wales increased to 8.5 per 100,000, a 25% increase from baseline, during the Russian influenza[4,5]. Similarly, the severe acute respiratory syndrome outbreak in 2003 led to suicide rates of 37.46 per 100,000 among older adults in Hong Kong, a 32% increase from baseline[6,7]. In addition, after the 2011 Tohoku earthquake and tsunami, standardized suicide rates in 2014 increased to 24.5 per 100,000 in Fukushima Prefecture, a 14.3% increase from baseline[8]. These statistics suggest that between 1.7 and 9.1 individuals per 100,000 resort to suicide as a result of such events, underscoring the need to identify population groups at risk for suicide. However, analyzing risk factors in variables of this magnitude requires a large sample size, and this challenge becomes even more pronounced when attempting to capture short-term changes in such variables.

In contrast to most hazardous events, the coronavirus disease 2019 (COVID-19) pandemic affected mental health worldwide[9]. If this pandemic affected suicidal tendencies in a manner similar to that of previous hazardous events, we would have witnessed a large number of suicides. In the early phase of the pandemic, many experts suggested that an increase in suicides was likely[10–12]. In the real world, however, empirical data on suicides were more nuanced than expected.

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Some countries experienced an increase in suicides during the COVID-19 pandemic[10,11,13–15]. Others reported no increases or even decreases in suicides[10,11,13–15]. Even within the same country or region, increases or decreases in suicides varied among demographic groups[16–19]. Furthermore, despite population heterogeneity, temporal heterogeneity is equally important. As has been observed with previous disasters[20,21], such as the 2011 Tohoku earthquake and tsunami[8], there may be a delayed increase in suicides[10,20]. A delayed trend may be more pronounced in pandemics like COVID-19 because although the majority of people return to their normal lives as the pandemic wanes, some may be left behind, continuing to adhere to lifestyles altered by the pandemic. Such divergence can cause tremendous suffering for those left behind and, in extreme cases, result in suicide. Even in countries or populations that so far have shown no obvious increase in suicides during the COVID-19 pandemic[10,15], suicide prevention remains a critical public health priority[12]. As heterogeneity in suicidal tendencies across populations cannot be explained by mere infection or mortality rates caused by the pandemic[14,15], it is essential to understand the underlying mechanisms linking hazardous events to suicides for effective suicide prevention.

Over 90% of those who die by suicide have a psychiatric disorder at their time of death[22–27]. Psychiatric states, such as depression, anxiety and post-traumatic stress disorder (PTSD)[12], are therefore likely to be key factors linking hazardous events to suicides[12,28–30]. Psychiatric conditions may be more informative for predicting suicide than suicidal ideation given that more than 60–70% of people who have died by suicide reported no suicidal ideation in assessments delivered within 30 days to 1 year of their deaths[31].

In short, we hypothesize that people or groups with severe psychiatric conditions will show higher suicidal tendencies. However, patients with psychiatric disorders have historically been thought to show a heightened risk of suicide as they begin to recover, when their energy and motivation return, rather than when their symptom severities are the greatest[32,33]. Emil Kraepelin, professor of psychiatry at the University of Heidelberg, wrote in 1896, 'Often, I saw precisely at that moment, suicide attempts that previously were not undertaken because of the lack of volition, despite great tedium of life'[32,34]. Our understanding of the temporal specificity of the association between psychiatric conditions and suicides is limited due to the difficulty in capturing short-term fluctuations in suicide rates, forcing previous studies to use suicidal ideation and incomplete suicide as proxy indicators, or to adopt cross-sectional designs[35,36]. Previous studies have not examined the temporal covariation between psychiatric states and suicidal tendencies during a single, large-scale, stressful event.

We hypothesized that event-related psychiatric states reliably predict increased suicide rates with high time specificity. Under this hypothesis, this study examined whether psychiatric states can reliably predict increased suicide rates and, if so, which psychiatric states can be used for such a prediction. Furthermore, we examined whether such predictability is temporally generalizable or time specific.

Results

Participants

After excluding participants with inconsistencies in their answers (Fig. 1), the current analyses included 3,815 responders at T0 (data before the pandemic; December 2019), 3,508 responders at T1 (first data during the pandemic; August 2020), 2,680 responders at T2 (second data during the pandemic; December 2020), 2,562 responders at T3 (third data during the pandemic; April 2021), 2,022 responders at T4 (fourth data during the pandemic; August 2021) and 1,806 responders at T5 (fifth data during the pandemic; December 2021) (Table 1).

Descriptive and outcome data

Table 1 shows the information on severity and prevalence of PTSD, depression and anxiety in each age–sex group from the online participants at each time point, along with information on the actual and estimated suicide rate increase. The actual suicide rate increase of each group was based on the full population from which this sample was drawn (full population of Japan). The estimated suicide rate increase of each group was calculated based on the prevalence of PTSD in the same group.

Main results

Effects of psychiatric states on the suicide rate. We first examined whether psychiatric states can predict increased suicide rates (and, if so, which psychiatric states can predict suicide rates) during a stressful event, the COVID-19 pandemic. These analyses were performed using severities of psychiatric scores (PTSD, depression and anxiety) for (1) T1 data, (2) T2–T5 data and (3) T1–T5 data, both at the individual and group level. In T1 data, our mixed-effect model analyses showed that the model based on PTSD scores had the smallest Bayesian information criterion (BIC) both in individual and group-level analyses compared with models based on depression or anxiety scores (Supplementary Fig. 1a,b; also see Supplementary Table 1 for the full statistics). We confirmed that adding depression scores and/or anxiety scores to PTSD scores (the psychiatric state that best predicted increased suicide rates in the T1 data) did not improve the model's goodness of fit. These findings held in T2–T5 data (Supplementary Fig. 1c,d) and in T1–T5 data (at the group level, for the main effect of psychiatric scores for PTSD alone, beta coefficient (β) = 0.04, t-statistics (t) = 5.29, degrees of freedom (df) = 48, $P = 3.0 \times 10^{-6}$, BIC = −23.69; for depression alone, $\beta = 0.04$, $t = 3.59$, df = 48, $P = 7.6 \times 10^{-4}$, BIC = −13.19; for anxiety alone, $\beta = 0.03$, $t = 2.88$, df = 48, $P = 5.9 \times 10^{-3}$, BIC = −9.35; Fig. 2, see Supplementary Fig. 1e for the individual-level analysis). The difference in BIC (ΔBIC) was larger than 10 (PTSD versus depression, ΔBIC = 10.5; PTSD versus anxiety, ΔBIC = 14.3), signifying a very strong difference. Although data were taken from the same group, group-level psychiatric severities were highly coherent across independent cohorts (see 'Other analyses'). Taken together, these findings attest to the robustness of the association of PTSD scores with suicide rate. In these models, neither confounder effects of depression nor those of anxiety can fully explain the association between PTSD and suicide increase.

The predictive power of estimated suicide risk. To further deepen our understanding of the association between PTSD and suicide risk, we estimated suicide risk (S_{risk}) based on known risk ratios for each sex ($PTSD_{risk}$ of 3.96 for men and 6.74 for women)[29] and PTSD prevalence based on our online survey ($PTSD_{prev}$) (model specification, $S_{risk} = (PTSD_{risk} - 1) \times PTSD_{prev}$). These analyses were performed using the prevalences of PTSD scores for (1) T1 data, (2) T2–T5 data and (3) T1–T5 data at the group level. We performed mixed-effects regression analyses to show the association between estimated suicide risk and the actual suicide rate across each age and sex group. This showed the strong predictive power of the estimated suicide risk in the T1 data ($\beta = 0.85$, $t = 6.3$, df = 8, $P = 2.3 \times 10^{-4}$; Supplementary Fig. 2a). This effect held in T2–T5 data ($\beta = 0.60$, $t = 5.5$, df = 38, $P = 2.8 \times 10^{-6}$; Supplementary Fig. 2b) and in T1–T5 data ($\beta = 0.63$, $t = 8.8$, df = 48, $P = 1.5 \times 10^{-11}$; Supplementary Fig. 2c) (Fig. 3). Furthermore, effects of the estimated suicide risk were not compromised by adding sex, age and/or time points as mixed effects (Supplementary Fig. 2). According to the coefficient of determination (R^2), 48% of the variability in the suicide rate during the pandemic across age groups and across time can be explained by variability of stress-related PTSD symptoms. These trends have also been observed in stratified analyses for each age group, suggesting that these effects were not only driven by stratified correlation (Supplementary Fig. 3). In other words, suicide risk is probably associated with psychiatric state. However, these analyses do not exclude the possibility that results in the base model are largely driven by stratified correlation, that is, the association between suicide risk and psychiatric trait in this context. In such a scenario, variability of suicide risk among groups

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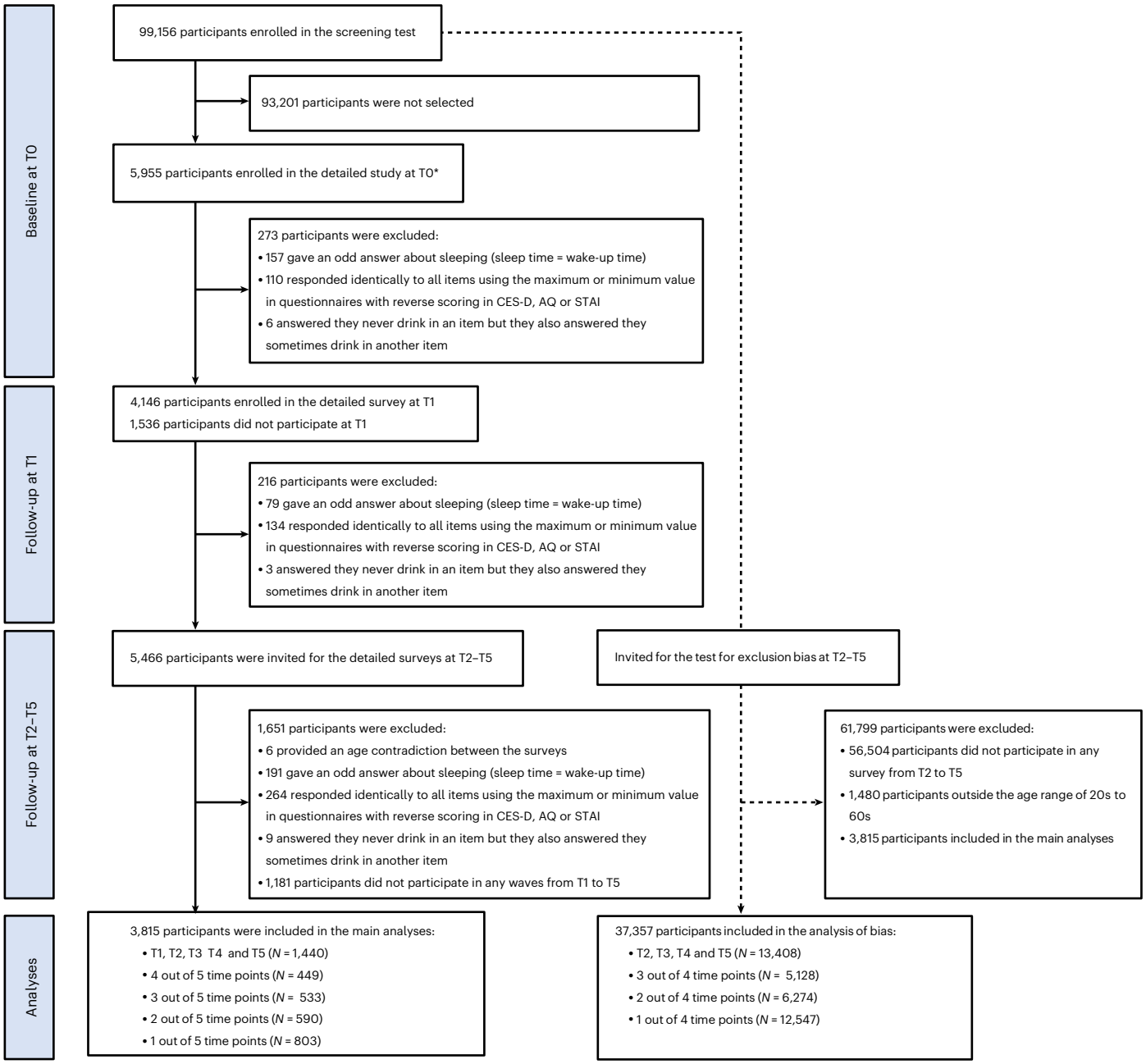

Fig. 1 | The study population. The procedures were decided based on the original survey (at T0 (*)). At that time, we aimed to collect data from enough individuals with high scores in PS use for a detailed survey. To do so, we performed a screening test (where participants reported demographics and PS scores). A total of 99,156 participants were enrolled in this screening test. These 99,156 participants were screened to include approximately equal numbers of individuals in each quintile relative to their PS score (assessed by the Japanese version of the Smartphone Addiction Scale Short Version). We also measured the Autism Spectrum Quotient (AQ) to capture participants' autistic characteristics. Because CES-D, STAI and AQ include reversed questions, individuals were excluded if they responded identically to all items using only the maximum or minimum values in the questionnaires. As a result, we extracted 5,955 participants from the screening population. The data at T0 were not analysed in this study.

should be explained by the variability of psychiatric conditions at any time point. There should be no time specificity in such an association.

Time specificity of estimated suicide risk. To examine the time specificity of the estimated suicide risk, we performed cross-lagged relationship analyses using the above base model. Specifically, we examined associations of the estimated suicide risk with the 'past' or 'future', in addition to the 'current' suicide rate. This analysis was performed using prevalences of PTSD scores for T1–T5 data at the group level. The mixed-effect model analysis showed that the model predicting current suicide increase had the smallest BIC compared with those predicting past or future suicide increases (Supplementary Fig. 4). The ΔBIC was larger than 2, which is a statistically meaningful difference. Therefore, the estimated suicide risk best predicted the suicide rate in the same month from which the scores were extracted, rather than in a past or future month. Interestingly, the association between estimated suicide risk and actual suicidal tendency appeared to decrease sharply moving back in time, but more gradually moving forward (Fig. 4). This qualitative view was statistically supported by the generalized linear model. Specifically, including the interaction between time distance and direction (toward the future or the past) in the model significantly improved the prediction of the Pearson correlation (distance, $\beta = -0.038$,

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Table 1 | Characteristics of study population

Variable	T0(December 2019)	T1(August 2020)	T2(December 2020)	T3(April 2021)	T4(August 2021)	T5(December 2021)
Total number of participants	3,815	3,508	2,680	2,562	2,022	1,806
Men						
Age 20–29 years (*n*)	144	133	86	81	53	45
PTSD	–	22.0 (19.7)/33.8% (47.5)	19.2 (19.5)/23.3% (42.5)	17.9 (18.9)/19.8% (40.1)	18.9 (18.7)/26.4% (44.5)	14.2 (16.3)/11.1% (31.8)
Depression	19.3 (11.6)/60.4% (49.1)	18.5 (11.4)/54.1% (50.0)	16.7 (10.7)/50.0% (50.3)	17.3 (12.0)/43.2% (49.8)	17.9 (11.0)/52.8% (50.4)	18.4 (13.1)/53.3% (50.5)
Anxiety	46.2 (10.9)/74.3% (43.8)	48.7 (10.1)/84.2% (36.6)	49.2 (11.0)/79.1% (40.9)	49.2 (10.4)/81.5% (39.1)	49.2 (10.5)/79.2% (40.9)	47.1 (13.0)/68.9% (46.8)
Actual/estimated suicide rate increase (%)	–	38.0/100.2	17.7/68.8	12.8/58.5	1.0/78.2	12.5/32.9
Age 30–39 years (*n*)	317	293	196	184	148	128
PTSD	–	19.5 (16.9)/21.8% (41.4)	18.0 (16.4)/19.9% (40.0)	17.3 (16.2)/21.7% (41.4)	17.9 (17.6)/21.6% (41.3)	14.5 (14.1)/15.6% (36.5)
Depression	18.4 (10.4)/55.5% (49.8)	17.8 (9.6)/50.5% (50.1)	15.9 (9.5)/41.3% (49.4)	15.7 (10.2)/40.8% (49.3)	17.2 (11.5)/46.6% (50.1)	16.7 (11.9)/46.1% (50.0)
Anxiety	46.9 (10.0)/76.3% (42.6)	50.4 (9.2)/88.4% (32.1)	50.3 (9.2)/87.2% (33.4)	49.4 (10.0)/85.3% (35.5)	50.6 (10.7)/86.5% (34.3)	48.5 (11.4)/78.1% (41.5)
Actual/estimated suicide rate increase (%)	–	22.0/64.7	−5.8/58.9	14.8/64.3	22.5/64.0	−16.3/46.3
Age 40–49 years (*n*)	614	566	449	425	353	334
PTSD	–	18.3 (17.9)/23.7% (42.5)	15.2 (15.9)/17.8% (38.3)	14.9 (15.5)/15.3% (36.0)	13.3 (14.5)/11.9% (32.4)	12.6 (15.4)/11.1% (31.4)
Depression	18.5 (11.0)/54.6% (49.8)	17.8 (11.3)/49.3% (50.0)	17.0 (11.0)/46.5% (49.9)	16.0 (10.9)/44.7% (49.8)	16.4 (11.3)/41.4% (49.3)	16.2 (11.6)/44.3% (49.7)
Anxiety	47.8 (10.9)/77.4% (41.9)	50.3 (10.5)/84.1% (36.6)	51.1 (10.9)/85.3% (35.4)	50.2 (11.3)/83.5% (37.1)	50.0 (11.3)/81.0% (39.3)	48.2 (11.5)/78.4% (41.2)
Actual/estimated suicide rate increase (%)	–	4.9/70.1	10.8/52.7	4.6/45.3	11.1/35.2	2.5/32.8
Age 50–59 years (*n*)	588	547	450	435	362	325
PTSD	–	18.9 (17.9)/24.9% (43.3)	13.5 (14.3)/13.8% (34.5)	12.8 (14.5)/11.3% (31.7)	11.7 (13.4)/9.7% (29.6)	11.6 (14.2)/9.5% (29.4)
Depression	17.0 (10.6)/47.4% (50.0)	16.8 (11.1)/45.7% (49.9)	15.0 (10.6)/40.0% (49.0)	15.1 (11.2)/40.5% (49.1)	15.3 (11.6)/39.2% (48.9)	14.3 (11.6)/35.4% (47.9)
Anxiety	46.6 (11.2)/71.1% (45.4)	50.8 (10.5)/84.3% (36.4)	50.4 (11.1)/83.6% (37.1)	50.2 (11.3)/82.1% (38.4)	49.1 (12.2)/79.8% (40.2)	47.4 (11.8)/75.4% (43.1)
Actual/estimated suicide rate increase (%)	–	7.1/73.6	11.9/40.8	−11.6/33.3	−2.6/28.6	2.4/28.2
Age 60–69 years (*n*)	295	273	230	228	192	170
PTSD	–	14.6 (14.2)/12.8% (33.5)	10.6 (11.4)/7.4% (26.2)	11.5 (12.7)/8.3% (27.7)	11.1 (11.9)/7.8% (26.9)	10.2 (12.0)/6.5% (24.7)
Depression	13.7 (9.6)/32.9% (47.1)	13.3 (9.4)/32.6% (47.0)	11.5 (8.7)/26.5% (44.2)	11.2 (9.1)/22.4% (41.8)	11.5 (9.1)/26.6% (44.3)	9.9 (8.5)/18.8% (39.2)
Anxiety	42.2 (10.1)/56.9% (49.6)	47.8 (9.5)/79.1% (40.7)	46.4 (10.2)/70.9% (45.5)	46.2 (11.0)/68.9% (46.4)	44.4 (11.0)/58.9% (49.3)	41.9 (11.3)/52.9% (50.1)
Actual/estimated suicide rate increase (%)	–	2.3/37.9	3.3/21.9	−3.3/24.7	−24.9/23.1	−24.4/19.2
Women						
Age 20–29 years (*n*)	204	172	113	115	70	62
PTSD	–	18.8 (18.0)/22.7% (42.0)	16.0 (18.1)/17.7% (38.3)	15.3 (16.4)/15.7% (36.5)	14.8 (15.7)/14.3% (35.2)	13.5 (14.0)/12.9% (33.8)
Depression	18.6 (10.9)/53.4% (50.0)	18.5 (9.8)/54.7% (49.9)	18.4 (11.9)/50.4% (50.2)	18.1 (10.2)/54.8% (50.0)	16.4 (9.5)/50.0% (50.4)	16.0 (10.7)/38.7% (49.1)
Anxiety	46.8 (11.5)/70.6% (45.7)	51.2 (10.3)/87.2% (33.5)	50.5 (11.9)/81.4% (39.1)	50.8 (11.4)/82.6% (38.1)	48.1 (12.1)/81.4% (39.2)	46.2 (10.4)/71.0% (45.8)
Actual/estimated suicide rate increase (%)	–	42.4/130.2	63.5/101.6	33.8/89.8	56.0/82.0	15.5/74.1
Age 30–39 years (*n*)	455	405	310	293	221	186
PTSD	–	18.9 (17.2)/21.7% (41.3)	15.8 (16.2)/16.5% (37.1)	15.5 (16.4)/13.7% (34.4)	14.9 (15.2)/12.7% (33.3)	12.9 (15.2)/11.3% (31.7)
Depression	17.9 (11.9)/48.8% (50.0)	18.0 (10.7)/50.9% (50.1)	17.0 (11.3)/49.0% (50.1)	17.7 (11.8)/49.8% (50.1)	17.2 (11.4)/45.2% (49.9)	17.0 (11.1)/48.9% (50.1)
Anxiety	46.5 (12.0)/65.1% (47.7)	52.4 (10.0)/86.7% (34.0)	51.3 (11.3)/81.9% (38.5)	52.1 (12.0)/82.6% (38.0)	51.3 (11.1)/83.7% (37.0)	48.5 (11.9)/77.4% (41.9)
Actual/estimated suicide rate increase (%)	–	77.3/124.7	20.5/94.4	41.3/78.4	51.0/72.7	18.9/64.8
Age 40–49 years (*n*)	487	451	341	330	247	224
PTSD	–	18.3 (17.5)/20.8% (40.7)	13.8 (14.6)/12.9% (33.6)	15.1 (15.8)/15.8% (36.5)	12.8 (13.7)/8.9% (28.5)	11.7 (15.4)/9.4% (29.2)
Depression	15.8 (10.2)/42.1% (49.4)	17.1 (10.5)/47.9% (50.0)	15.6 (10.3)/39.9% (49.0)	16.0 (10.4)/43.3% (49.6)	15.4 (11.2)/39.7% (49.0)	15.2 (10.9)/38.8% (48.8)
Anxiety	45.5 (11.0)/65.5% (47.6)	51.8 (10.0)/86.5% (34.2)	51.3 (10.8)/85.6% (35.1)	51.8 (11.3)/85.5% (35.3)	49.9 (12.3)/74.9% (43.4)	48.8 (12.1)/75.0% (43.4)
Actual/estimated suicide rate increase (%)	–	64.3/119.6	29.5/74.1	34.7/90.4	41.2/51.1	−8.0/53.8
Age 50–59 years (*n*)	486	457	348	321	260	225
PTSD		16.9 (15.7)/16.8% (37.5)	12.5 (13.9)/10.3% (30.5)	12.6 (13.3)/9.0% (28.7)	12.5 (13.8)/9.2% (29.0)	11.3 (14.0)/7.6% (26.5)
Depression	15.7 (10.6)/42.8% (49.5)	17.4 (11.4)/49.5% (50.1)	15.6 (10.6)/41.7% (49.4)	14.9 (10.8)/38.0% (48.6)	15.6 (11.1)/42.3% (49.5)	15.5 (11.3)/40.0% (49.1)

Table 1 (continued) | Characteristics of study population

Variable	T0(December 2019)	T1(August 2020)	T2(December 2020)	T3(April 2021)	T4(August 2021)	T5(December 2021)
Anxiety	44.7 (11.4)/60.5% (48.9)	52.4 (9.8)/86.4% (34.3)	51.0 (10.9)/81.6% (38.8)	51.6 (10.9)/83.2% (37.5)	50.2 (11.3)/78.8% (40.9)	48.9 (11.5)/74.7% (43.6)
Actual/estimated suicide rate increase (%)	–	36.5/96.7	3.0/59.4	17.2/51.9	8.0/53.0	13.1/43.4
Age 60–69 years (*n*)	225	211	157	150	116	107
PTSD	–	13.9 (13.4)/11.4% (31.8)	11.5 (13.7)/8.9% (28.6)	11.2 (13.2)/6.0% (23.8)	9.4 (12.5)/6.0% (23.9)	8.5 (12.3)/6.5% (24.8)
Depression	12.6 (9.2)/28.9% (45.4)	14.0 (10.0)/34.1% (47.5)	12.0 (8.9)/29.9% (45.9)	12.4 (10.4)/26.7% (44.4)	10.8 (8.9)/24.1% (43.0)	11.0 (9.7)/23.4% (42.5)
Anxiety	39.8 (10.5)/42.2% (49.5)	49.7 (11.0)/75.8% (42.9)	47.1 (11.1)/70.7% (45.7)	48.5 (11.0)/73.3% (44.4)	44.7 (10.7)/62.9% (48.5)	43.0 (11.8)/49.5% (50.2)
Actual/estimated suicide rate increase (%)	–	23.7/65.3	48.3/51.2	16.5/34.4	14.0/34.6	58.4/37.6

Data are presented as mean severity in raw value (s.d.)/prevalence in percentages (s.d.) for PTSD (as measured by IES-R), depression (as measured by CES-D) and anxiety (as measured by STAI-Y). Actual suicide rate increase denotes those actually increased compared to the pre-pandemic level of 2019. Estimated suicide rate increase denotes those estimated to increase compared to the pre-pandemic level of 2019 based on the prevalence of PTSD from our samples in a given population.

$t = -2.3$, df = 10, $P = 0.047$; interaction term, $\beta = -0.063$, $t = -4.2$, df = 10, $P = 0.002$, BIC = −12.1) compared with the prediction obtained without the interaction term ($\beta = -0.07$, $t = -3.0$, df = 11, $P = 0.011$, BIC = −3.7). The ΔBIC was larger than 2 (ΔBIC = 8.4), signifying a statistically meaningful difference. According to these analyses, the Pearson correlation shows a monthly decline of 0.10 when moving toward the past and declines by 0.038 when moving toward the future. This indicates that the Pearson correlation declines 2.65 times more rapidly toward the past than toward the future. This finding supports the idea that the increase in suicide rate at the population level does not precede exacerbations of psychiatric conditions; rather, the suicide rate increases in response to such exacerbations. Furthermore, it also suggests that effects of exacerbated psychiatric conditions on suicide are not instantaneous, but persist for several months.

Other analyses

In the test of selection bias based on our criteria, the estimated increase in the suicide rate showed a strong positive correlation between the screened and excluded populations (Pearson's $r = 0.89$, $P = 2.1 \times 10^{-14}$; Supplementary Fig. 5a), indicating selection bias was unlikely to account for our results. In the test of the effect of residence, the estimated increase in the suicide rate showed a strong positive correlation between a population from Osaka and those from locations other than Osaka (Pearson's $r = 0.95$, $P = 4.5 \times 10^{-21}$; Supplementary Fig. 5b), indicating that our findings are unrelated to residence.

Discussion

Key results

We showed that nearly half of the variability in suicide rate, regardless of age and sex, can be explained by a combination of COVID-19-related PTSD symptoms and previously reported sex differences in the suicide risk of PTSD. We further showed that the effects at T1 also held at T2–T5. Compared with other psychiatric symptoms, event-related PTSD symptoms appear to be reliable surrogate endpoints for increased suicide, outperforming depression or anxiety scores in predicting concurrent suicide risk. The most important finding was the strong temporal specificity of the association between symptoms and the suicide rate. This suggests, at least at a group level, that the population shows higher suicide risk when PTSD severity is higher. This finding provides a new avenue in research and prevention of suicide risk at the population level.

Interpretation

The high temporal specificity of the association found here indicates that reducing event-related PTSD symptoms could help prevent event-related suicides. Thus, event-related PTSD symptoms may work as a surrogate endpoint for suicide. Such a surrogate endpoint is beneficial for suicide prevention given the extremely low prevalence of suicides, usually less than 0.003% per month[37]. It is not realistic, or even possible, to observe such a large sample size in populations at risk, such as those who lost their jobs during the pandemic, those who got divorced during the pandemic and so on. Our data, however, suggest that measuring PTSD symptoms might be sufficient to identify populations at greater risk of suicide. If our findings apply to other traumatic events, we may be able to estimate the risk of suicide increase in other trauma-exposed populations (such as populations who witness genocide or who have experienced abuse) at given time points, that is, soon after the trauma, 1 year after trauma and so on. Policies and efforts to reduce PTSD severity within populations, or for individuals, are expected to reduce the overall suicide risk. Although further clarifications are needed to draw definitive conclusions, individuals at risk of suicide might be identifiable based on validated questionnaires in various settings, such as clinics, agencies and online consultations. Individuals identified through these channels should be guided to official clinical settings and given empirically validated treatments focused on PTSD, such as early intervention strategies[38].

Our approach is agnostic as to whether COVID-19-related PTSD symptoms are the primary reason for suicides of those who resorted to suicide due to the pandemic. Risk factors for suicide typically act additively or synergistically, that is, patient risk levels increase with the number of risk factors[33]. In such a scenario, even if suicides in a given population increased in proportion to the severity of COVID-19-related PTSD symptoms in that population, it is possible that COVID-19-related PTSD symptoms were not the primary reason for the suicides. These symptoms may have raised the likelihood of suicidal decision-making depending on other primary reasons, such as economic strain, social isolation and physical disorders. Regardless of the primary reason, it is possible that decreasing severe PTSD symptoms (a candidate warning sign of potential suicide susceptibility) may reduce suicide risk.

Limitations

Several limitations should be considered. First, PTSD scores based on online surveys are sometimes higher than those observed in other types of surveys[19,39], which may also explain the relatively high prevalence of COVID-19-related PTSD diagnoses in our online populations (5.3–34.6%) and in previous online research (7–35.6%)[40,41]. Consequently, estimated numbers of suicides based on PTSD scores are higher than actual numbers. It is important to note that although our data support the reliability of estimated numbers of suicides as ratio scales, raw values are likely to be overestimated. Second, we relied on self-report measures to evaluate psychiatric conditions from participants online, which have lower accuracy compared with clinician-administered diagnostic interviews. In particular, depression and anxiety were assessed with less-validated questionnaires compared with that used for PTSD. This

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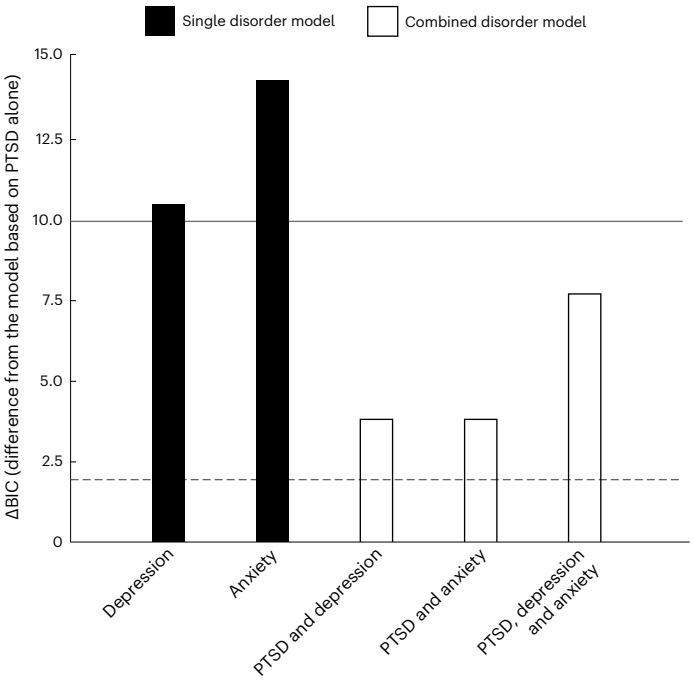

Fig. 2 | Comparison of models that test associations between psychiatric states and increases in suicide rate. Statistical tests were performed with a mixed-effects model analysis (two-tailed test). The BIC of each model is presented as a difference from the model based on PTSD symptoms alone (PTSD model). Positive values indicate that the PTSD model is superior to the model shown by each bar. ΔBIC values larger than 2 (dashed line) are considered significantly worse than the PTSD model, whereas differences larger than 10 (solid line) are considered 'very strong' differences. Filled bars represent models based on depression or anxiety and open bars represent models based on combinations of PTSD, depression and/or anxiety scores. This figure is based on data from T1–T5. The ΔBIC values for each bar from left to right are 10.50, 14.34, 3.85, 3.86 and 7.75.

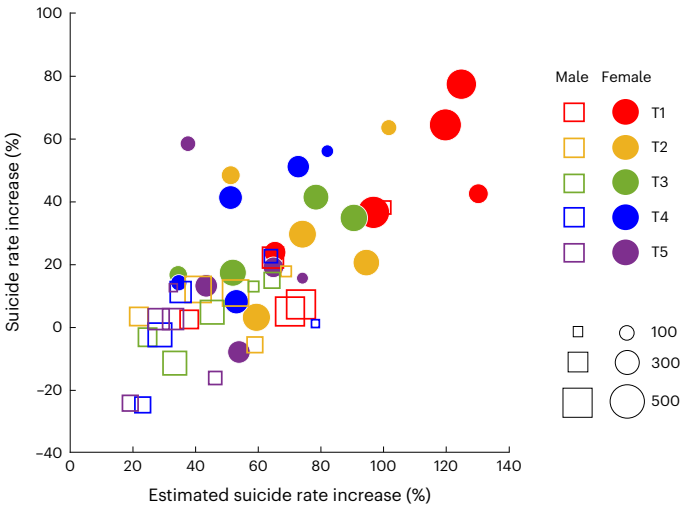

Fig. 3 | Association between the estimated suicide risk, based on COVID-19-related PTSD symptoms, and the actual suicide rate. Each circle and square represents the suicide rate increase estimated from PTSD probability (*x* axis) and the suicide rate increase (*y* axis) for each age group. Circles and squares represent female and male data, respectively. Colours represent data acquisition timing and the size of the shapes represents the number of online participants. Circles and squares of sizes representing 100, 300 and 500 individuals are shown for reference.

may explain why these questionnaires did not capture more variance in suicide rates than was obtained from the questionnaire about PTSD symptoms. Third, our correlational approach prevents us from drawing conclusions about causality. It is possible that an unmodeled external factor could explain the strong correlation between COVID-19-related PTSD symptoms. Further clarification must be obtained on replicability and causality of the reported associations and whether these group-level findings can be applied at the individual level.

Generalizability

Global surveys with similar approaches could help to better explain reasons behind differences in suicide rate changes across countries, leading to more effective prevention of suicide worldwide. It may be possible to generalize these findings to other large-scale, long-lasting, stressful events.

In summary, we found that COVID-19-related PTSD symptoms at given points in time can predict concurrent suicide increases. Importantly, the associations between PTSD symptoms and suicide decrease only gradually with time, making it possible to take action for identified groups at risk of suicide. Further research based on our findings may help governments and agencies to focus prevention resources on groups at high risk of suicide, especially groups showing higher PTSD symptoms.

Methods

Study design

This study examined the association between psychiatric states and changes in suicide rates during the COVID-19 pandemic in Japan, which experienced a tremendous increase in suicide that varied greatly across gender and age groups[14,42]. Cross-sectional psychiatric states at multiple points in time were estimated through online questionnaires. Specifically, we performed real-time, online monitoring of a large online cohort immediately before the pandemic and at five time points during the COVID-19 pandemic. The online surveys were conducted six times with the same population: once before the pandemic (December 2019; T0) and five times during the pandemic (T1 in August 2020, T2 in December 2020, T3 in April 2021, T4 in August 2021 and T5 in December 2021). Note that, for the purpose of this article, only time points during the pandemic (T1–T5) were analysed.

Setting

This work is part of a larger online survey on problematic smartphone (PS) use, which was approved by the Ethics Committee of the Advanced Telecommunications Research Institute International. Details of trajectories of psychiatric states and demographic data have been previously published[43,44]. The study was originally planned in 2019 before the COVID-19 pandemic, and was later expanded to examine the psychiatric impact of COVID-19. Given the real-time aspect of the pandemic, we compared three psychiatric conditions in their predictability of suicide increase using the T1 data as soon as they were collected (also see ref. 45) Then, for all subsequent analyses using T2–T5 data, we included in the model only the psychiatric condition with the highest predictability. This approach ensured the model was fixed with the initial T1 data during the pandemic and T2–T5 data points could be considered as real-time data (T2–T5). Therefore, all analyses were performed on T1 and T2–T5 separately and T1–T5 combined. The exception to this is the analyses of time specificity of the predictive power of estimated suicide risk because this analysis could not be done with T1 data alone.

Participants

From registrants of an online survey company (Macromill; https://monitor.macromill.com/) who were living in the Kansai region of Japan, 99,156 individuals were invited via email to participate in a screening for the original study. In that screening, participants reported their demographics and smartphone-related items, including PS use scores.

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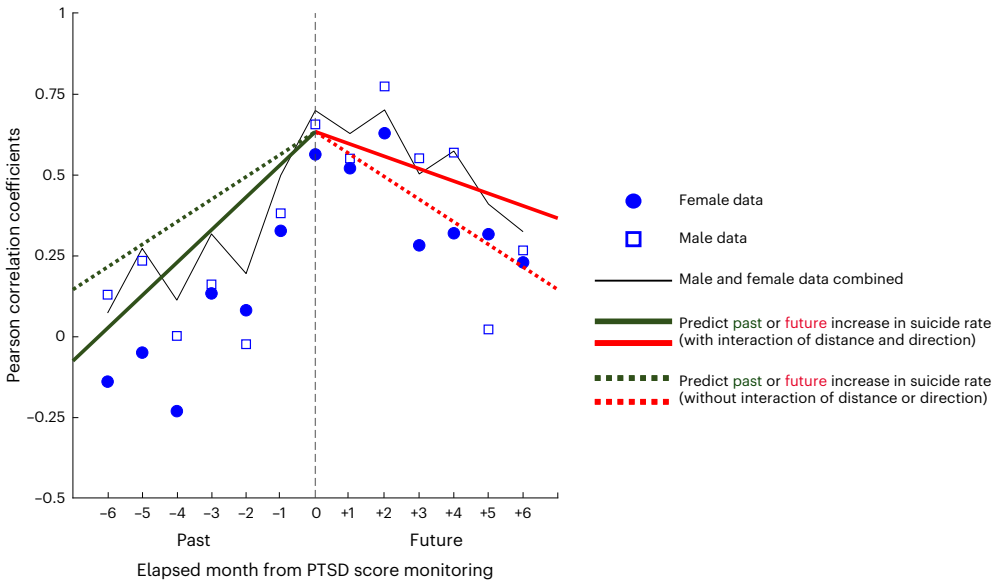

Fig. 4 | Cross-lagged association between the estimated increase in suicide rate and the actual increase. The *x* axis denotes the month from which the actual suicide rate increase was extracted. The estimated increase in the suicide rate in the *n*th month was compared with the actual increase in the suicide rate in the *n* + *x*th month. The *y* axis represents Pearson correlation coefficients between estimated and actual suicide rate increases. Circles and squares represent female and male data, respectively. The black line indicates male and female data combined. The green (red) line represents the fitted line from the generalized linear models (two-tailed test) used to predict past (future) increases in the suicide rate. The solid (coloured dashed) line represents the fitted line based on the generalized linear model (two-tailed test) with (without) the interaction between distance and direction toward future and past.

The email contained information about informed consent, and completion of the questionnaire was taken to indicate participant consent. Of these 99,156 individuals, 5,955 were screened and recruited in December 2019 (T0), such that the population evenly included individuals belonging to each quintile of PS use scores. In response to the COVID-19 pandemic, we invited the volunteers to participate in follow-up online surveys containing additional questions about COVID-19-related PTSD symptoms, at T1–T5. Participants were excluded if (1) they contradicted their answers across items (for example, in one question they answered that they never drink, but in another question they answered that they sometimes drink) or across surveys (for example, age differs more than 2 years within 1 year surveys); and (2) they answered using only the maximum or minimum rating in questionnaires that include reverse items (for example, Center for Epidemiologic Studies Depression Scale (CES-D) and Form Y of the State–Trait Anxiety Inventory (STAI-Y); also see refs. 43,44). Figure 1 shows the flow diagram of the number and proportion of participants retained at each stage. In response to the findings in T1 data that PTSD symptoms are the most predictive of suicide, we collected COVID-19-related PTSD symptoms from those excluded via the above screening processes in the T2–T5 data. These data were used to examine the possibility of selection bias (see 'Bias').

Variables

Depression, anxiety and COVID-19-related PTSD scores were taken from the online survey, as were age and sex. Suicide numbers were extracted from the provisional database provided by the Ministry of Health, Labour and Welfare (MHLW) in Japan. Potential confounding factors include selection bias based on our criteria, effect of residence of our online participants and self-selection bias that participants were selected from online registries. Variables used in the mixed-effect analyses are listed in Supplementary Table 2.

Data sources and measurement

Following standard procedures[46,47], we relied on self-administered questionnaires to measure COVID-19-related PTSD symptoms. We used the widely used 22-item Impact of Events Scale-Revised (IES-R)[48] to assess PTSD symptoms. Note that in our online survey, PTSD symptoms were assessed specifically with respect to COVID-19, for example, 'I thought about COVID-19 when I didn't mean to.' As is recommended[48], we regarded an IES-R score >32 as probable PTSD. To assess depression symptoms, we used the CES-D[49]. CES-D comprises 20-items, with scores higher than 15 signifying probable depression. The STAI-S[50] was used to assess state anxiety symptoms. STAI-S comprises 20 items, where scores higher than 40 or 41 denote probable anxiety disorder for men or women, respectively.

We extracted monthly suicide numbers from January 2019 to June 2022 from the provisional database provided by the MHLW in Japan (https://www.mhlw.go.jp/stf/seisakunitsuite/bunya/0000140901.html; accessed 8 June 2023).

Bias

To exclude potential confounding factors due to selection bias based on our criteria, we compared the estimated increase of the suicide rate for each subgroup in the screened population (a total of 9,070 participants) with those in the excluded population (a total of 94,111 participants) in the T2–T5 data (see Fig. 1).

To test the effect of residence, the estimated increase of the suicide rate was compared for each subgroup of participants from Osaka (a total of 38,034 participants) with those from locations other than Osaka (a total of 56,077 participants) in the T2–T5 data. Of note, in August 2020, Osaka had the second-largest number of people with COVID-19 in Japan.

Study size

Due to characteristics of the observational study during the COVID-19 pandemic, study size was restricted to that of the original larger study. We conducted a post hoc power analysis using G*Power v.3.1.9.7 (Franz Faul; Kiel University) to check the adequacy of our sample size. Using the coefficient of determination in the main result based on stress-related PTSD symptoms ($R^2 = 0.48$, $\alpha = 0.05$) for a sample size of 50 (the number of age–sex groups), the statistical power (1 − probability of a type-II error) was more than 0.99.

Quantitative variables

Psychiatric variables. In the individual-level analysis, each individual had their own value for psychiatric status at each time point. In the group-level analysis, averages of depression, anxiety and COVID-19-related PTSD scores were taken from the data during the COVID-19 pandemic, that is, data from T1–T5, for each sex and age group.

Suicide rates. Using the national database, we calculated suicide rates during the COVID-19 pandemic (that is, 2020, 2021 and 2022) for each sex and age group (10 year bins) compared with suicide numbers a year before the pandemic (that is, 2019). Specifically, the suicide rate for each group in a specific month was defined as follows: the number of suicides in that group in the specific month of 2019 subtracted from that in the same month in 2020, 2021 or 2022 divided by the number of suicides in 2019. Suicide numbers were adjusted based on the corresponding population at that time. Each value was calculated for each group and was therefore used as if it were in the group-level analysis. In the individual-level analysis, each online participant was assigned one value of suicide rate corresponding to their age and sex. This value was used to explain across-participant heterogeneity.

Estimated suicide risk. The suicide risk in a given population was estimated from the prevalence of probable PTSD, according to the following equation:

$$S_{\mathrm{risk}} = (\mathrm{PTSD}_{\mathrm{risk}} - 1)\mathrm{PTSD}_{\mathrm{prev}} \quad (1)$$

where $\mathrm{PTSD}_{\mathrm{risk}}$ is the risk ratio of suicide (PTSD:healthy)[29] (3.96 for men and 6.74 for women) and $\mathrm{PTSD}_{\mathrm{prev}}$ is the frequency of probable PTSD diagnosis from our samples in a given population, that is, the rate at which the threshold, IES-R >32, was exceeded. This model was designed before acquisition of T2–T5 data[45].

Statistical methods

Effects of psychiatric states on the suicide rate. To show associations between psychiatric states and suicide rate, we used psychiatric state data from an online survey (N = 3,508; T1 data). Specifically, we compared how well these different measures predict the impact of COVID-19 on suicide rate throughout the entire Japanese population (population of 125.9 million). We used mixed-effects models to test whether each of three psychiatric conditions could predict the suicide rate. In all models, psychiatric state, that is, PTSD, depression or anxiety, was considered a fixed effect, whereas sex was classified as a random effect (the model was specified as 'Suicide Increase ~ 1 + PsychiatricScore + (1 | sex)', where '~' indicates the relation between the response and predictor variables). We used the BIC to compare model goodness of fit, with smaller values indicating better models. Traditionally, a ΔBIC value larger than 2 is considered a significant difference between models, whereas a difference larger than 10 is considered a very strong difference[51]. Among the three psychiatric states examined, we extracted the best psychiatric state with the highest performance in predicting the suicide rate in the T1 data. We further examined whether adding other psychiatric state(s) to the best psychiatric state improved the model goodness of fit. Finally, applying each state to the equation above, six models were examined for T1 data. With these models, we performed individual-level analysis and group-level analysis. In the group-level analyses, the number of online participants in each group was included as weight term. These analyses were first applied to data from the T1 epoch and to-be-examined models were defined. We then examined whether effects for T1 data held for T2–T5 data.

Testing the predictive power of estimated suicide risk. We performed mixed-effects regression analyses to show the association between estimated suicide risk and the actual suicide rate across each age and sex group. Analyses were weighted by the online population size at a given time point, that is, for each group. This model was defined as the 'base model.' Again, these analyses were applied to (1) T1 data, (2) T2–T5 data and (3) T1–T5 data. We examined whether addition of age, sex and time point in analyses (2) and (3) to the base model as random effects compromised the results of the analysis on the base model.

Time specificity of estimated suicide risk. To examine the time specificity of the estimated suicide risk, we performed cross-lagged relationship analyses using the above base model for T1–T5 data. Specifically, we examined associations of the estimated suicide risk with the past or future, instead of the current suicide rate. The past was defined as the previous x months from current time point (−5, …, −1), and future was defined as x months following the current time point (+1, …, +5). For example, for the estimated suicide risk for August 2020, the actual suicide rates in July, August and September 2020 were respectively defined as past (−1), present and future (+1) suicide rates. We also calculated the Pearson correlation to examine estimation accuracy for demonstration purposes. Our subsequent investigation sought to determine any disparity in the correlation trend toward past or future time points. Using generalized linear models, we examined whether temporal distance from the present could effectively forecast the Pearson correlation. The primary objective of this analysis was to evaluate whether incorporating an interaction term across time distance and direction for future versus past enhanced the model's performance or remained inconsequential (model specification was Pearson correlation ~ 1 + distance + distance : direction). The main effect of direction was not included in the model because doing so differentiated the intercepts across the fitted lines toward the past and toward the future. Our focus was on examining the differential slopes across these directions while keeping the intercept, that is, the fitted value for the current time point, the same. Therefore, we only included direction as an interaction term in the model. We regarded the difference as statistically meaningful when the ΔBIC exceeded 2. Statistical analyses were performed using MATLAB v.R2019b.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

The main summary statistics that support the findings of this study are available in the Supplementary Information. Owing to company cohort data-sharing restrictions, individual data cannot be publicly posted. However, data are available from the corresponding authors upon request and with permission of KDDI Corporation. Data requests should be sent to the corresponding authors and will be responded to within 21 days.

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Author contributions
T.C., K.I. and A.C. contributed to study conception and design. T.C., N.K., T.O., Y.M., T.H. and M.H. contributed to data acquisition. T.C., M.M., T.O., F.N., R.Y., T. Kubo and A.C. conducted statistical analyses. T.C., K.I., T.O., H.T., T. Kanazawa, S.B., T. Kubo, A.H., M.K. and A.C. contributed to interpretation of data. T.C., K.I. and M.M. drafted the first version of the article. T.C., M.M., T.O., N.K. and A.C. had full access to the data. All authors revised and approved the final version of the article. T.C. and A.C. take responsibility for the integrity of the work.

Competing interests
N.K., Y.M., T.H. and M.H. are employees of KDDI Research. The other authors declare no competing interests.

Additional information
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nature portfolio

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Last updated by author(s): 22 Sep 2023

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Statistics

For all statistical analyses, confirm that the following items are present in the figure legend, table legend, main text, or Methods section.

n/a	Confirmed	
☐	☑	The exact sample size (*n*) for each experimental group/condition, given as a discrete number and unit of measurement
☐	☑	A statement on whether measurements were taken from distinct samples or whether the same sample was measured repeatedly
☐	☑	The statistical test(s) used AND whether they are one- or two-sided *Only common tests should be described solely by name; describe more complex techniques in the Methods section.*
☐	☑	A description of all covariates tested
☑	☐	A description of any assumptions or corrections, such as tests of normality and adjustment for multiple comparisons
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☐	☑	For null hypothesis testing, the test statistic (e.g. *F*, *t*, *r*) with confidence intervals, effect sizes, degrees of freedom and *P* value noted *Give P values as exact values whenever suitable.*
☑	☐	For Bayesian analysis, information on the choice of priors and Markov chain Monte Carlo settings
☑	☐	For hierarchical and complex designs, identification of the appropriate level for tests and full reporting of outcomes
☐	☑	Estimates of effect sizes (e.g. Cohen's *d*, Pearson's *r*), indicating how they were calculated

Our web collection on statistics for biologists contains articles on many of the points above.

Software and code

Policy information about availability of computer code

Data collection	No software used
Data analysis	We used matlab (version: R2019b)

For manuscripts utilizing custom algorithms or software that are central to the research but not yet described in published literature, software must be made available to editors and reviewers. We strongly encourage code deposition in a community repository (e.g. GitHub). See the Nature Portfolio guidelines for submitting code & software for further information.

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All manuscripts must include a data availability statement. This statement should provide the following information, where applicable:

- Accession codes, unique identifiers, or web links for publicly available datasets
- A description of any restrictions on data availability
- For clinical datasets or third party data, please ensure that the statement adheres to our policy

Main summary statistics that support the findings of this study are available in the Supplementary Data. Owing to company cohort data-sharing restrictions, individual data cannot be publicly posted. However, data are available from the authors upon request and with permission of KDDI Corporation. Data requests should be sent to the corresponding author, Toshinori Chiba, t.chiba0906@gmail.com, and will be responded to within 21 days.

Research involving human participants, their data, or biological material

Policy information about studies with human participants or human data. See also policy information about sex, gender (identity/presentation), and sexual orientation and race, ethnicity and racism.

Reporting on sex and gender	female: 46.8%
Reporting on race, ethnicity, or other socially relevant groupings	Japanese
Population characteristics	The main analysis used multidimensional psychiatric data taken immediately before and during the COVID-19 epidemic for 3,815 participants (mean age 47.1 years, 46.8% female).
Recruitment	From registrants of an online survey company (Macromill, INC; https://monitor.macromill.com/)
Ethics oversight	Ethics Committee of the Advanced Telecommunications Research Institute International

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Field-specific reporting

Please select the one below that is the best fit for your research. If you are not sure, read the appropriate sections before making your selection.

☐ Life sciences ☑ Behavioural & social sciences ☐ Ecological, evolutionary & environmental sciences

For a reference copy of the document with all sections, see nature.com/documents/nr-reporting-summary-flat.pdf

Life sciences study design

All studies must disclose on these points even when the disclosure is negative.

Sample size	
Data exclusions	
Replication	
Randomization	
Blinding	

Behavioural & social sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description	Quantitative data from a longitudinal survey of 3,815 people by an Internet research company (Macromill) during the COVID-19 pandemic and over a long period of time after the pandemic were used for the psychological effects of COVID-19.
Research sample	A repeat internet survey was conducted in cooperation with Macromill, Inc.. The initial survey was conducted in December 2019. Since the first COVID-19 case was identified in Japan in January 2020, the data in this survey are considered baseline data (T0); following the COVID-19 epidemic, the subjects in this survey were invited to participate in August 2020 (T1), December 2020 (T2), April 2021 (T3), August 2021 (T4), and December 2021 (T5). These follow-up surveys added several items related to COVID-19: multidimensional psychiatric data taken just before and during the COVID-19 epidemic were collected from a total of 3,815 participants (mean age 47.1 years, 46.8% female). This number included some who responded to all of T1 to T5 and others who responded only to multiple timings of T1 to T5. The data used in this study are not representative.
Sampling strategy	We relied on Macromill, INC. (https://monitor.macromill.com/), the largest online research company in Japan for participant recruitment. This company maintains a participant pool of about 1.2 million individuals in Japan. Among this participant pool, 99,156 randomly chosen individuals, aged 18 and above, living in the Kansai region of Japan, were invited via e-mail to participate in screening for the original study before the COVID-19 pandemic (at T0). The email invitation to the survey included information on informed consent, and participants were considered to have consented to answer all questions in the survey.

The procedures were decided based on the original survey (at T0). At that time, we aimed to collect enough individuals with high scores in problematic smartphone (PS) use scores for a detailed survey. To do so, we performed a screening test (where participants reported demographics and PS score). Participants were then screened to include approximately equal numbers of individuals in each quintile relative to their PS score. This study does not include qualitative data. According to the post-hoc power analysis, our sample size of 50 data points was larger than a sample size of 20 data points that would be sufficient to achieve 99% power to detect an effect size of r = 0.7.

Data collection

Macromill collected information directly from the study participants and provided it to the researcher. The researcher was not present at the time of data collection. This study is not an intervention study; therefore, the researcher was not blinded to the experimental conditions or other factors.

Timing

The online surveys were conducted 6 times from the same population: once before the pandemic (December 2019: T0) and 5 times during the pandemic (August 2020: T1, December 2020: T2, April 2021: T3, August 2021: T4, and December 2021: T5).

Data exclusions

Participants were excluded if 1) they provided contradictory answers across items, e.g., to one question they answered that they never drink, but to another they answered that they sometimes drink, or across surveys (e.g. age differed more than two years within one year surveys).
2) they answered using only the maximum or minimum rating in questionnaires which include reverse items (e.g. CES-D and STAI-Y).
When the study period was divided into three periods: baseline T0, immediate post-pandemic T1, and post-pandemic T2-T5, 273 were excluded in T0, 216 in T1, and 1,651 in T2-T5.

Non-participation

No participants withdrew consent. The participants were able to participate in the online survey any number of times at their discretion. The current analyses included 3,815 responders at T0, 3,508 responders at T1, 2,680 responders at T2, 2,562 responders at T3, 2,022 responders at T4, and 1,806 responders at T5.

Randomization

As this is a longitudinal observational study of the impact of the worldwide COVID-19 pandemic, randomization is not relevant in this study.

Ecological, evolutionary & environmental sciences study design

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Study description	
Research sample	
Sampling strategy	
Data collection	
Timing and spatial scale	
Data exclusions	
Reproducibility	
Randomization	
Blinding	

Did the study involve field work? ☐ Yes ☐ No

Field work, collection and transport

Field conditions	
Location	
Access & import/export	
Disturbance	

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Materials & experimental systems

n/a	Involved in the study
☑	☐ Antibodies
☑	☐ Eukaryotic cell lines
☑	☐ Palaeontology and archaeology
☑	☐ Animals and other organisms
☑	☐ Clinical data
☑	☐ Dual use research of concern
☑	☐ Plants

Methods

n/a	Involved in the study
☑	☐ ChIP-seq
☑	☐ Flow cytometry
☑	☐ MRI-based neuroimaging

Antibodies

Antibodies used	

Validation

Eukaryotic cell lines

Policy information about cell lines and Sex and Gender in Research

Cell line source(s)	
Authentication	
Mycoplasma contamination	
Commonly misidentified lines (See ICLAC register)	

Palaeontology and Archaeology

Specimen provenance	
Specimen deposition	
Dating methods	

☐ Tick this box to confirm that the raw and calibrated dates are available in the paper or in Supplementary Information.

Ethics oversight	

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Animals and other research organisms

Policy information about studies involving animals; ARRIVE guidelines recommended for reporting animal research, and Sex and Gender in Research

Laboratory animals	
Wild animals	
Reporting on sex	
Field-collected samples	
Ethics oversight	

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Clinical data

Policy information about clinical studies

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Clinical trial registration	
Study protocol	
Data collection	
Outcomes	

Dual use research of concern

Policy information about dual use research of concern

Hazards

Could the accidental, deliberate or reckless misuse of agents or technologies generated in the work, or the application of information presented in the manuscript, pose a threat to:

No	Yes	
☐	☐	Public health
☐	☐	National security
☐	☐	Crops and/or livestock
☐	☐	Ecosystems
☐	☐	Any other significant area

Experiments of concern

Does the work involve any of these experiments of concern:

No	Yes	
☐	☐	Demonstrate how to render a vaccine ineffective
☐	☐	Confer resistance to therapeutically useful antibiotics or antiviral agents
☐	☐	Enhance the virulence of a pathogen or render a nonpathogen virulent
☐	☐	Increase transmissibility of a pathogen
☐	☐	Alter the host range of a pathogen
☐	☐	Enable evasion of diagnostic/detection modalities
☐	☐	Enable the weaponization of a biological agent or toxin
☐	☐	Any other potentially harmful combination of experiments and agents

Plants

Seed stocks

Novel plant genotypes

Authentication

ChIP-seq

Data deposition

☐ Confirm that both raw and final processed data have been deposited in a public database such as GEO.

☐ Confirm that you have deposited or provided access to graph files (e.g. BED files) for the called peaks.

Data access links
May remain private before publication.

Files in database submission

Genome browser session
(e.g. UCSC)

Methodology

Replicates

Sequencing depth

Antibodies

Peak calling parameters

Data quality

Software

Flow Cytometry

Plots

Confirm that:

☐ The axis labels state the marker and fluorochrome used (e.g. CD4-FITC).

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☐ All plots are contour plots with outliers or pseudocolor plots.

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Methodology

Sample preparation

Instrument

Software

Cell population abundance

Gating strategy

☐ Tick this box to confirm that a figure exemplifying the gating strategy is provided in the Supplementary Information.

Magnetic resonance imaging

Experimental design

Design type

Design specifications

Behavioral performance measures

Imaging type(s)

Field strength

Sequence & imaging parameters

Area of acquisition

Diffusion MRI ☐ Used ☐ Not used

Preprocessing

Preprocessing software

Normalization

Normalization template

Noise and artifact removal

Volume censoring

Statistical modeling & inference

Model type and settings

Effect(s) tested

Specify type of analysis: ☐ Whole brain ☐ ROI-based ☐ Both

Statistic type for inference

(See Eklund et al. 2016)

Correction

Models & analysis

n/a	Involved in the study
☐	☐ Functional and/or effective connectivity
☐	☐ Graph analysis
☐	☐ Multivariate modeling or predictive analysis

Functional and/or effective connectivity

Graph analysis

Multivariate modeling and predictive analysis