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BRIEF REPORT

Census Tract Ambient Ozone Predicts Trajectories of Depressive Symptoms in Adolescents

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Exposure to ozone is a well-documented risk factor for negative physical health outcomes but has been considered less frequently in the context of socioemotional health. We examined whether levels of neighborhood ozone predicted trajectories of depressive symptoms over a four-year period in 213 adolescents (ages 9–13 years at baseline; 57% female; 53% of minority race/ethnicity). Participants self-reported depressive and other types of psychopathology symptoms up to 3 times, and their home addresses were used to compute ozone levels in their census tract. Possible confounding variables, including personal, family, and neighborhood characteristics, were also assessed. We found that higher ozone predicted steeper increases in depressive symptoms across adolescent development, a pattern that was not observed for other forms of psychopathology symptoms. These findings underscore the importance of considering ozone exposure in understanding trajectories of depressive symptoms across adolescence.

Keywords: ozone, air pollution, developmental trajectories, depressive symptoms, adolescence

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Depressive symptoms affect an alarmingly high number of children and adolescents, with rates increasing across development (Keyes et al., 2019). Not only are depressive symptoms in youth associated with distress, academic underachievement, and poorer social functioning (Jaycox et al., 2009), but they also forecast risk for enduring impairment across the life span. Efforts to identify and intervene around environmental contributors to developmental risk for depression have focused predominantly on individual and family-level psychosocial characteristics, such as the quality of interpersonal relationships or socioeconomic resources (Davis et al., 2015). While these foci have been fruitful, researchers have generally ignored children's *physical* environment, including exposure to pollution. Given that inhaling pollution activates biological pathways implicated in the development of depression, including immune, cardiovascular, and neurodevelopmental processes (Costa et al., 2020), exposure to ambient air pollution may influence the development and/or trajectory of depressive symptoms in youth.

Ozone, a primary component of air quality indices, is a gas that is the product of pollutants (Volatile Organic Compounds and Nitrogen Oxides) reacting to sunlight (Environmental Protection Agency, 2008). Exposure to higher levels of ozone has been linked consistently to physical health risk, ranging from increased susceptibility to respiratory viruses to asthma to premature mortality

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This study was not preregistered. The information presented in the current article utilizes families' home addresses and census tract information. Without IRB approval to share this information, we cannot place the data in a public repository. However, we will make the data and study materials available to any individual researcher who requests them. The scoring algorithm for the Traumatic Events Screening Inventory for Children is available at https://github.com/lucysking/els_stress_interview.

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(Bernstein et al., 2004). In fact, a study of European cities estimated that air pollution caused 6% of all deaths in France, Switzerland, and Austria (Pope et al., 2019). Furthermore, studies have demonstrated that associations between ozone and mortality are present even at levels substantially below national standards (Huang et al., 2019). Despite the ubiquity of this hazard and the seriousness of its effects, little research has been conducted examining the association between ozone exposure and risk for difficulties in *social and emotional* health.

Biologically, one of the ways in which ozone exposure confers risk to physical health is by activating immune processes. For example, inhaling ozone can contribute to both local and systemic inflammation, resulting in higher circulating levels of the inflammatory marker C-reactive protein (Arjomandi et al., 2015; Riediker et al., 2004). Notably, such inflammatory processes are also implicated in psychopathology symptoms—most robustly with symptoms of depression, including onset and trajectories of symptoms (de Baumont et al., 2019; Mills et al., 2013). Animal models further elucidate possible pathways by demonstrating that ozone can disrupt neurotransmitter activity, promote greater oxidative stress, and lead to differential expression of inflammatory proteins in the substantia nigra and striatum (Block & Calderón-Garcidueñas, 2009; Pereyra-Muñoz et al., 2006). Although more research is needed, these studies suggest that ozone can act on neuroimmune processes that could plausibly lead to symptoms of depression.

Recently, epidemiological studies of adults have linked exposure to high levels of ozone with depression-relevant psychological phenomena, such as higher use of antidepressants (Lu et al., 2018; Zhao et al., 2018). Researchers have also noted that suicides and emergency department visits for mental health covary with the seasonality of ozone exposure (Biermann et al., 2009; Szyszkowicz et al., 2016). While this initial work is important, these studies generally overlook within-person changes in specific psychological symptoms, which would strengthen directional interpretations and provide greater specificity to the findings. Moreover, the majority of research in this area has focused on adults (although see Perera et al., 2013 for an exception), despite calls from psychologists to consider the contribution of physical contaminants to *child developmental* outcomes (Trentacosta et al., 2016). Separately, research examining other components of air pollution, such as nitrogen dioxide (NO_2), has shown that ambient exposure is associated with greater risk for psychotic symptoms in youth (Newbury et al., 2019; Oudin et al., 2016). Importantly, however, similar studies have not yet been conducted examining ozone and trajectories of depressive symptoms.

Investigating associations between ozone and depressive symptoms in adolescents is likely to be particularly fruitful. Adolescence is a period of rapid physiological change during which individuals may be especially responsive to environmental input; indeed, adolescence has been proposed to be a "second sensitive period" (Fuhrmann et al., 2015), after infancy. In this context, late childhood and adolescence are associated with the onset of several forms of psychopathology. For example, anxiety and depressive symptoms rise precipitously during this period (Carter et al., 2011; Lewinsohn et al., 1995). Thus, adolescents may be particularly vulnerable to the effects of ozone on risk for depressive symptoms. Furthermore, adolescents are exposed to more air pollution than are individuals in other age groups due to greater time spent outdoors (Sacks et al., 2011).

In the current study we investigated in a community sample of adolescents whether levels of ozone in participants' census tracts predict the developmental trajectories of depressive symptoms across approximately four years of assessments in which we examined three depressive-symptom subscales from two well-validated measures of youth psychopathology. We then examined whether personal or community-level psychosocial factors (such as stress exposure, family income, parental education, or neighborhood disadvantage) accounted for the observed associations. Finally, in an exploratory analysis to test specificity, we examined whether ozone was related to other types of psychopathology symptoms by assessing the trajectories of other symptom subscales across the same period.

Method

Study Overview

English-speaking adolescents in the early stages of puberty at baseline (Tanner Stage III or below, rated by self-report; Morris & Udry, 1980) participated in a larger study examining exposure to early life stress and subsequent internalizing psychopathology. Participants in the San Francisco Bay Area were recruited through local advertisements beginning in Autumn 2013. During an initial assessment ("T1"), adolescents responded to self-report questionnaires assessing symptoms of psychopathology, completed a semi-structured interview regarding their exposure to stressful life events, and provided demographic information. Approximately two and four years later ("T2" and "T3"), participants returned to the lab and repeated the self-report measures of psychopathology symptoms. Using publicly available air quality data compiled by the California Environmental Protection Agency, we mapped participants' home addresses reported at their baseline visit to information about average maximum eight-hour ozone from the most recent Cal Enviro Screen (3.0) report cycle, which spanned the years 2012–2014. Because the current study utilizes participants home addresses, and without permission to share this information, data and study materials are not able to be placed in a public repository, but rather are available upon request to any individual researcher. This study was not preregistered.

Participants

Two hundred and thirteen participants (ages 9–13; 121 females) were enrolled at T1; 157 of those adolescents provided information on psychopathology symptoms again at T2 and 125 provided information on psychopathology symptoms at T3. Because growth curve modeling with robust standard errors is able to handle within-person missing data and time-varying assessments, all available data were included in the present analyses.

Average age of the sample at T1 was 11.37 years. Approximately 8.5% of participants self-identified as African American, 12.3% identified as Asian, 10.4% identified as nonwhite Latinx, 47.2% identified as White, and 21.7% identified as biracial or of another racial or ethnic group. On average, family income was between $75,001–100,000, with a range from less than $5,000 to more than $150,000 and parents had on average a two-year college degree. There were no differences between participants who provided data at all time points and those with missing data for main study

variables or demographic characteristics, with the sole exception that participants with complete data had slightly higher parental education (four-year college degree vs. two-year college degree).

Participation of human subjects in the current study was approved and overseen by the Stanford University Institutional Review Board (protocol title: "Stress and Puberty"; protocol number: 27671). At each time point, written informed consent was obtained from each adolescent's parent and written informed assent was obtained from the adolescents.

Measures

Ozone

Exposure to ozone was assessed using data compiled by the California Environmental Protection Agency (Cal Enviro Screen; OEHHA, 2017). Daily maximum eight-hour average concentrations of ozone for all air monitoring sites in California were extracted for the years 2012–2014 and inverse distance weighting was used to estimate ozone exposure for the center of each census tract from nearby sites. The maximum eight-hour average is the metric used by the EPA and clean air standard to assess ozone levels (Environmental Protection Agency, 2008). Participants' home mailing addresses at baseline were used to determine their census tract. Census tracts in California contain an average of 4,000 people (range: 1,200−8,000).

Psychopathology Symptoms

At each of the timepoints across the four years, participants completed two self-report questionnaires assessing their symptoms of psychopathology: the Children's Depression Inventory-Short Form (CDI-S; Kovacs, 1992) and the Youth Self Report (YSR; Achenbach & Rescorla, 2001). We administered the CDI-S to assess depressive symptomatology, which includes 10 items rated on a scale of 0= "once in a while," 1= "many days," and 2= "every day." The CDI-S yields comparable results to the full CDI (Allgaier et al., 2012) and has acceptable reliability and validity (Smucker et al., 1986). The YSR is a 112-item broadband assessment of mental health symptoms rated from 0 = 'Not True' to 2 = 'Very True' that captures a wide range of youth psychopathology symptoms and includes two subscales reflecting internalizing and externalizing profiles. The YSR has well-demonstrated reliability and validity (Song et al., 1994). Because we were primarily interested in depressive symptomatology, in addition to the CDI-S, two YSR subscales that capture depressive phenomenology—the Withdrawn/Depressed and Anxious/Depressed subscales—were used in main analyses; however, we examined all other YSR symptom subscales in secondary analyses to assess discriminant associations with ozone.

Neighborhood Disadvantage

Communities with fewer socioeconomic resources are often disproportionately affected by air pollution (O'Neill et al., 2003). Thus, it is possible that associations between ozone and youth psychopathology are due to higher levels of poverty or unemployment, and/or to lower levels of educational achievement in the community—factors previously shown to be related to mental health outcomes (Dallaire et al., 2008). To examine this possibility, we computed a composite factor of neighborhood disadvantage using the summed standardized values per census tract of (a) the percentage of the population within a tract who lived below 2x the federal poverty level using the 2011–2015 American Community Survey values (U.S. Census Bureau, 2015); (b) the percentage of the population over age 18 years with less than a high school diploma; and (c) the percentage of the population over age 16 years and unemployed but eligible for the labor force, as compiled by the Cal Enviro Screen.

Stressful Life Events

A related possibility is that youth who live in areas with higher ambient ozone experience higher levels of stressful life events, and that it is this stress exposure—not ozone—that confers risk for psychopathology. To test this alternative explanation, we also examined adolescents' lifetime exposure to acute stressful events at the T1 assessment using a modified version of the Traumatic Events Screening Inventory for Children (Ford et al., 2002), a semistructured interview that assesses over 30 types of experiences, such as parental divorce or major illnesses. A panel of three coders rated the objective severity of each stressor from a scale of 1 to 5 with half-point increments where 1 = *nonevent or no-impact* and 5 = *extremely severe impact*, with information about the adolescents' reactions or behaviors during the interview masked prior to rating (ICC = .99), using the UCLA Life Stress Interview coding system (Rudolph & Hammen, 1999). Events rated 2.5 or higher have been considered 'severe' stressors in other psychological research (e.g., Miller & Chen, 2010). We then summed the number of events rated 2.5 or higher to index adolescents' exposure to stress across their lifetime, an approach that overcomes problems with adding all events or the severity of all events, which may be more susceptible to reporting biases. The scoring algorithm is available at: https://github.com/lucysking/els_stress_interview (King, 2020).

Demographic Characteristics

Adolescents' age at baseline, sex, and minority status were assessed for inclusion as covariates. In parallel to community-level factors, participant-level family income and parental education attainment were also included.

Data Analytic Plan

For primary analyses, we conducted a series of growth curve models with Huber-White robust standard errors using HLM8 (Raudenbush et al., 2019) in which the slope and intercept of depressive symptom scores for the two YSR subscales and CDI-S over time (Level 1) were modeled separately as a function of community ozone (Level 2). Because participants were assessed at slightly different intervals across the study, we used the number of days since their T1 (baseline) assessment to model trajectories. We examined each symptom measure separately allows for an assessment of consistency in effects across theoretically related indicators, predicting that ozone would significantly predict increasing trajectories of depressive symptom across each measure. Although families were nested within census tracts, only 18 families shared a tract with another family in the study and tests of fully unconditional 3-level models indicated that only 4–9% of the variance in these outcomes were attributable to census tract. Consequently, 2-level models were conducted in the current study (although 3-level models yielded the same pattern of results). If

significant effects emerged, tests of simple slope were conducted to examine symptom trajectories for individuals whose census tracts experienced an ozone level of $-1SD$ below the mean, the mean, and $+1SD$ above the mean. In secondary analyses we then added individual- (sex, age at baseline, minority status, and number of severe life stressors), family- (household income category and parental educational attainment), and community-level (neighborhood disadvantage) covariates at Level 2 to test whether associations between ozone and changes in psychopathology symptoms are due to these demographic or psychosocial variables. Further, to begin to explore discriminant associations across forms of psychopathology, we repeated analyses to consider trajectories of other types of psychopathology symptoms by examining the YSR subscales indexing somatic-, delinquent-, aggressive-, social-, thought-, and attention-specific symptoms. Finally, given the positive skew of ozone in this sample (skewness = 1.29), we repeated analyses using a log-transformed ozone variable.

Results

Descriptive statistics are reported in Table 1. Notably, observed average ozone levels were all less than the National Ambient Air Quality Standard limit of .07 ppm (Environmental Protection Agency, 2008) and indices of neighborhood disadvantage ranged considerably across participants' communities. As presented in Table 2, ozone did not predict the intercept of symptoms in any of the primary models, meaning that ozone was not significantly related to T1 symptoms. However, there were significant interactions between days-since-T1-visit and ozone for both CDI-S and Withdrawn/Depressed YSR scores, such that the strength of the association between time and symptoms grew stronger as ozone increased ($f^2 = .02$ model of CDI; $f^2 = .01$ model of Withdrawn/Depressed). Although the pattern of results was similar for scores on the Anxious/Depressed YSR subscale, the interaction effect was not statistically significant ($\gamma_{11} = .16$, $SE = .12$, $t = 1.29$, $p = .198$). Tests of simple slopes examining trajectories of CDI-S symptoms for participants in census tracts with average exposure at $-1SD$, the mean, and $+1SD$ of ozone indicated that there was not a significant change in symptoms over time for individuals living in communities at $1SD$ below the mean (slope = .000, $SE < .001$, $t = .51$, $p = .605$); however, for individuals at the mean and $+1SD$ above the mean, there were significant increases in symptoms over time, with the slope becoming steeper as ozone increased ($\text{slope}_{\text{mean}} = .000$, $SE < .001$, $t = 3.85$, $p < .001$; $\text{slope}_{+1SD} = .000$, $SE < .001$, $t = 4.83$, $p < .001$; see Figure 1). Likewise, simple slopes tests for the Withdrawn/Depressed subscale of the YSR revealed that associations between symptoms and time were not significant for participants at $1SD$ below the mean (slope = .000, $SE < .001$, $t = -.17$, $p = .867$); however, there were significant associations between ozone and symptoms at the mean and $1SD$ above the mean ($\text{slope}_{\text{mean}} = .000$, $SE < .001$, $t = 2.75$, $p = .006$; $\text{slope}_{+1SD} = .001$, $SE < .001$, $t = 3.89$, $p < .001$).

Secondary analyses in which sex, age at baseline, minority status, household income category, parental education, number of severe life stressors, and neighborhood disadvantage were included as covariates indicated that these variables did not account for the associations between ozone and either CDI-S or Withdrawn/Depressed symptom trajectories (see online supplemental materials for full model results).

Although we hypothesized associations between ozone and trajectories of depressive symptoms due to associations between inflammatory pathways and depression, we also explored trajectories of symptoms from all other YSR subscales in order to examine specificity and consistency across different forms of psychopathology. We found that ozone did not significantly predict the slope of any other symptom subscales (see online supplemental materials).

Finally, we repeated analyses using a log transformation of ozone. The pattern of results remained the same, with the exception that the association between log ozone and trajectories of

Table 1
Descriptive Statistics for Primary Study Variables

Variable	*M*	*SD*	Observed range
CDI-S T1	2.20	2.38	0–11
CDI-S T2	2.36	2.66	0–13
CDI-S T3	3.34	3.29	0–13
Withdrawn/Depressed (YSR) T1	3.56	2.47	0–12
Withdrawn/Depressed (YSR) T2	3.24	2.63	0–11
Withdrawn/Depressed (YSR) T3	4.45	2.58	0–11
Anxious/Depressed (YSR) T1	5.85	5.18	0–26
Anxious/Depressed (YSR) T2	5.20	5.63	0–27
Anxious/Depressed (YSR) T3	7.71	6.10	0–24
Age T1	11.37	1.05	9.17–13.98
Age T2	13.24	1.08	11.00–15.85
Age T3	15.48	1.16	13.10–18.25
Income category	8.42	2.09	1–11
Parental education category	4.83	1.29	1–8
Number severe life events	2.78	2.64	0–16
Ozone (Census tract; parts per million)	0.0344	0.0039	0.0296–0.0531
Education (Census tract; % of population)	8.83	7.40	0.03–37.50
Poverty (Census tract; % of population)	19.39	12.40	2.00–62.50
Unemployment (Census tract; % of population)	7.11	3.28	1.10–20.30
Neighborhood disadvantage composite	0.00	2.57	−3.98–9.16

Note. CDI-S = Children's Depression Inventory-Short Form; YSR = Youth Self Report.

Table 2
Results of Growth Curve Models Predicting the Intercept and Trajectory of Symptom Scores Over Time as a Function of Ozone in the Participant's Census Tract

Fixed effect	Coefficient	*SE*	95% CI	*p* value
		Model of CDI-S Scores		
Intercept, β_0				
Intercept, γ_{00}	2.48	1.31	[−0.09, 5.05]	.061
Ozone, γ_{01}	−10.82	37.58	[−84.48, 62.84]	.774
Slope (Time), β_1				
Intercept, γ_{10}	−0.01	0.00	[−0.01, −0.01]	.002
Ozone, γ_{11}	0.18	0.05	[0.08, 0.28]	<.001
		Model of Withdrawn/Depressed (YSR) Scores		
Intercept, β_0				
Intercept, γ_{00}	4.09	1.62	[0.91, 7.27]	.012
Ozone, γ_{01}	−20.78	46.73	[−112.37, 70.81]	.657
Slope (Time), β_1				
Intercept, γ_{10}	0.00	0.00	[0.00, 0.00]	.010
Ozone, γ_{11}	0.13	0.04	[0.05, 0.20]	.004
		Model of Anxious/Depressed (YSR) Scores		
Intercept, β_0				
Intercept, γ_{00}	3.72	3.23	[−2.61, 10.05]	.251
Ozone, γ_{01}	51.16	93.66	[−132.41, 234.73]	.586
Slope (Time), β_1				
Intercept, γ_{10}	0.00	0.00	[0.00, 0.00]	.272
Ozone, γ_{11}	0.16	0.12	[−0.08, 0.40]	.198

Note. Time is calculated as days from T1 assessment. Interpretations of values: γ_{00} reflects the average symptom score for baseline visit; γ_{01} reflects the effect of ozone on baseline symptoms; γ_{10} reflects the effect of time on symptoms; and γ_{11} reflects the effect of ozone on the slope of symptoms over time.

Anxious/Depressed subscale symptoms became significant (γ_{11} = .02, *SE* = .01, *t* = 2.04, *p* = .042).

Discussion

The results of this study indicate that adolescents who live in census tracts with relatively higher average ozone are at greater risk for experiencing trajectories of increasing depressive symptoms over time relative to teens who live in areas with lower levels of ozone, even when levels are below the .07 ppm national standard for ozone. This is the first study of ozone exposure to use repeated measurement of depressive symptoms in adolescents and to test competing possibilities that findings are accounted for by stress, socioeconomic resources, or neighborhood factors. In doing so, this study adds to a growing body of research implicating air pollution not only in physical health outcomes, but also in youth mental health outcomes (Reuben et al., 2021). Given that symptoms of depression in childhood and adolescence are associated with enduring adverse difficulties in psychosocial functioning (Hofstra et al., 2002; Lewinsohn et al., 1995), these effects may have lifelong consequences.

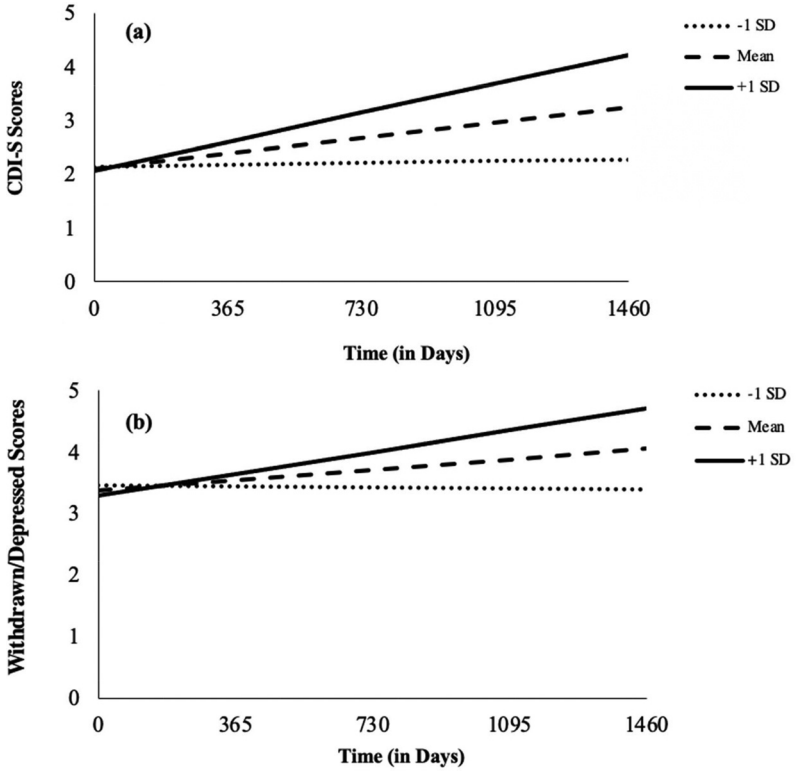
Interestingly, the association between ozone and symptom trajectories as measured by Anxious/Depressed subscale of the YSR was not as strong as it was for the CDI-S or Withdrawn/Depressed scales, suggesting that associations are more robust for behavioral withdrawal symptoms of depression than for other types of symptoms (although additional work is necessary before drawing strong conclusions). This interpretation was also supported by secondary findings that there were not significant associations when considering any other subscales of the YSR, including attention, thought, or aggressive symptoms, raising the possibility that associations between ozone and symptom trajectories are specific to depressive symptoms. It is important to note, however, that the parent study was designed to examine internalizing symptoms in adolescents and, consequently, provided only limited coverage of other types of symptoms. Such a pattern is consistent with a theorized neuroimmune mechanism, in which immune dysregulation has also been implicated more strongly in depression than in anxiety disorders or externalizing disorders (Anand et al., 2017; Kautz et al., 2019). It is plausible, however, that other types of air pollution are related to other forms of psychopathology. Indeed, recent work suggests that nitrogen oxide exposure in childhood predicts externalizing symptoms and cognitive difficulties at age 18, but only weakly predicts internalizing symptoms (Reuben et al., 2021).

Although most prior work examining air pollution has focused on adults, on young children, or on the prenatal period (e.g., Liao et al., 1999; Perera et al., 2013), the current study suggests that adolescents are also vulnerable to the effects of ozone exposure. Not only do adolescents spend more time outdoors than do individuals of other ages, given their developing neurobiological systems it is possible that adolescents are *especially* sensitive to these effects. Preliminary support for this possibility is reflected by the finding that baseline symptoms (assessed prior to puberty) were not associated with ozone, but that the association strengthened as a function of time. If replicated in other work, we may recognize that adolescence is not only a time of heighted risk, but also an opportune period during which to intervene early with the greatest impact.

The results of this study provide preliminary support for the possibility that ozone is an overlooked contributor to the development or course of youth depressive symptoms—expanding the range of risk factors that psychologists typically consider in

Figure 1

Trajectories of Depressive Symptoms as Assessed by CDI-S Scores (Panel a) and Withdrawn/Depressed YSR Subscale Scores (Panel b) as a Function of Ozone in Participants' Census Tracts



Note. CDI-S = Children's Depression Inventory-Short Form; YSR = Youth Self Report.

studying the etiology of depression. Moreover, these findings argue for a broader conceptualization of the health effects of ozone exposure that moves beyond those factors related to respiratory and cardiovascular health to include psychological health as well. Although it is well-documented that air pollution exposure affects physical health disproportionately in older individuals (Liao et al., 1999), this study highlights the possibilities that ozone also confers risk during youth and that negative consequences may unfold on a relatively short-term scale (i.e., over years vs. decades). Furthermore, given that exposure to air pollution disproportionally affects marginalized populations, these results also point to the possible role of ozone affecting depressive symptoms as a contributor to health disparities. Together, these implications underscore the urgency of reducing global air pollution in order to save lives and reduce suffering.

Exposure to ozone largely occurs without individuals' awareness. Nevertheless, there are several ways to reduce exposure. For example, accessible and low-cost interventions include efforts to remain indoors during ozone-warning days, such as relocating youth sporting events into indoor arenas when necessary—recommendations that can be reinforced by youth mental health practitioners and educational providers. Further, limiting driving and fueling during peak hours of ozone alerts can reduce both personal and community ozone exposure. Governmental interventions include greater promotion of air quality alerts by public health officials and larger investments in sustainable and low-emission sources of energy to reduce air pollution.

We should note several limitations of this study. First, while we analyzed longitudinal data, the study was essentially correlational, making it impossible to infer causality. Although secondary analyses tested several possible confounder variables, it is possible that associations are nevertheless due to a third, unstudied, variable. Second, the current study relied on single averaged assessments of ozone per census tract over a three-year period. While these composited data are frequently used in public-facing air pollution data communication (such as AirNow.gov), they lack the temporal and geospatial resolution that would allow for more precise characterizations of ozone per specific residence. Meteorologic data, land use regressions, or other geospatial approaches—as well as consideration of other air pollutants beyond ozone—would improve future work. As a related point, we were unable to examine *personal* ozone exposure; it is likely that families living in the same neighborhoods differ in how much time they spend outdoors or the quality of the air inside their house. Here, incorporating wearable technology would provide a more granular and personal assessment, as would assessing how long families lived in their home prior to their baseline assessment and their amount of outdoor activity. Third, we did not assess biological pathways through which risk may be conferred, which would have substantially bolstered causal and mechanistic interpretations. Fourth, we could not

explicitly assess the clinical significance of these associations, given that we did not generate formal psychiatric diagnoses in this study; we should note, however, that 59 participants at T3 had CDI-S scores that were above the cutoff of 3 recommended for clinical screening (Allgaier et al., 2012). Nonetheless, dimensional assessments of youth psychopathology symptoms provide important information about emotional development, can better capture the range of individual differences of symptom number and severity, and may better comport with the latent structure of mood disorders (Haslam et al., 2012; Hudziak et al., 2007). Relatedly, we administered self-report measures of psychopathology; future work should include informant reports, including clinician reports. Finally, all participants in the current study resided in a relatively small geographical area, specifically, within driving distance of the Bay Area in California, which may result in a more limited range of ozone levels. Importantly, however, this restricted range should have the effect of *underestimating* the true association between ozone and psychopathology symptoms.

This study provides evidence that ozone is associated with steeper trajectories of depressive symptoms in adolescents and offers several exciting directions for future research. Studies that trace changes in putative biological mechanisms, such as systemic inflammation, neurodevelopment, or stress reactivity, will deepen theoretical models. It will also be important for researchers to include participants with a wide age range within a single study to test more clearly whether there are sensitive developmental periods for exposure, perhaps by examining mental health in all members within a household. In addition, it will be important to test whether there are other psychosocial or biological factors, like social support or stress responsivity, that make some adolescents more vulnerable than others to the effects of ozone exposure. Finally, future research should seek to elucidate temporal associations between air pollution and mental health symptoms; for example, are daily changes in ozone related to differences in mood or symptoms? Nevertheless, despite its limitations and the questions that remain to be answered, the current study is important in providing preliminary empirical support for the association between ozone exposure and developmental trajectories of depressive symptoms in adolescents, identifying a novel potential contributor to mental health in youth.

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