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Association of Postpartum Temperature Exposure with Postpartum Depression: A Retrospective Cohort Study in Southern California

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Background: Postpartum depression (PPD) has been associated with biological, emotional, social, and environmental factors. However, evidence regarding the effect of temperature on PPD is extremely limited.

Objectives: We aimed to examine the associations between postpartum temperature exposure and PPD.

Methods: We conducted a retrospective cohort study using data from Kaiser Permanente Southern California electronic health records from 1 January 2008 through 31 December 2018. PPD was first assessed using the Edinburgh Postnatal Depression Scale (score $\geq$10) during the first year of the postpartum period and further identified by using both diagnostic codes and prescription medications. Historical daily ambient temperatures were obtained from the 4-km resolution gridMET dataset (https://www.climatologylab.org/gridmet.html) and linked to participants' residential addresses at delivery. Postpartum temperature exposures were measured by calculating various temperature metrics during the period from delivery to PPD diagnosis date. A time-to-event approach with a discrete-time logistic regression was applied to estimate the association between temperature exposure and time to PPD. Effect modification by maternal characteristics and other environmental factors was examined.

Results: There were 46,114 (10.73%) PPD cases among 429,839 pregnancies (mean $\pm$ standard deviation age $= 30.22 \pm 5.75$ y). Increased PPD risks were positively associated with exposure to higher mean temperature [adjusted odds ratio (aOR) per interquartile range increment: 1.07; 95% confidence interval (CI): 1.05, 1.09] and diurnal temperature range (aOR = 1.08; 95% CI: 1.06, 1.10); the associations were stronger for maximum temperature compared with minimum temperature. The temperature-related PPD risks were greater among African American, Asian, and Hispanic mothers and among mothers $\geq$25 years of age compared with their counterparts. We also observed higher effects of temperature on PPD among mothers exposed to higher air pollution or lower green space levels and among mothers with lower air conditioning penetration rates.

Conclusion: Maternal exposure to higher temperature and diurnal temperature variability during the postpartum period was associated with an increased risk of PPD. Effect modification by maternal age, race/ethnicity, air pollution, green space, and air conditioning penetration was identified. https://doi.org/10.1289/EHP14783

Introduction

As the perinatal form of major depressive disorder, postpartum depression (PPD) is one of the most frequent childbirth complications.[1] Postpartum depressive symptoms affect ~10%–20% of new mothers worldwide.[1–4] Mothers with PPD experience symptoms, such as feelings of anhedonia, worthlessness or guilt, depressed mood, insomnia, or hypersomnia, that can last for up to 1 y postpartum, and infants born to mothers with PPD are at a greater risk of developing behavioral and emotional problems, learning disabilities, and obesity later in life.[2,5–7] PPD also influences mother–infant bonding, which impacts the child throughout their childhood and later life.[6,8]

High ambient temperatures and heat stress have been shown to impact mental health in previous epidemiological studies, focusing on outcomes such as increased hospital admissions and emergency department visits for mental health concerns and exacerbation of symptoms for mental and behavioral disorders.[9–14] For example, a study in the United States, in California, found that hospital admissions for mental health concerns increased by a daily mean average of 9.8% during heat–health events.[13] Rising temperatures and heat stress could affect mental health by causing increases in cortisol levels[15] and initiating systemic inflammatory responses[11]; in addition, extreme heat events can exacerbate everyday life stresses.[14] There has also been research regarding the impact of heat stress on the blood–brain barrier (BBB)[16,17] and the hypothalamic–pituitary–adrenal (HPA) axis,[16,18] which results in a subsequent poor stress response. For example, increased BBB permeability due to heat stress can lead to increased infiltration of immune cells, inflammatory cytokines, and reactive oxygen species into the brain,[16,17] which have been associated with a dysregulated HPA axis.[19,20] A dysregulated HPA axis, the structure responsible for the body's stress response, can alter cortisol levels, resulting in

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poor stress responses.[5,7,21] Although the relationship between high temperatures and mental health in the general population has been documented,[9–13,22] a limited number of studies have investigated the relationship between high-temperature exposure and mental health among pregnant women. Only one previous study, in Shanghai, China, reported that extremely high temperatures may induce emotional stress during pregnancy.[23] No past study has explored the impact of temperature on postpartum maternal mental health. Moreover, biological and hormonal changes during the postpartum period affect the body's ability to regulate core body temperature,[24,25] so temperature variation during this period may be more uncomfortable for mothers and adversely affect pregnancy outcomes. Research into the impact of temperature on PPD is important to provide insight into etiological mechanisms, identify women who are at the greatest risk, and implement targeted prevention and intervention strategies.

Maternal exposure to antepartum and postpartum air pollution[26] and low levels of green space[27] have been demonstrated as potential environmental factors for PPD in our previous work. The frequency and intensity of climate sensitive events[9,15] are increasing over time. Associations between heat stress and mental health might be modified by exposure to air pollutants that can potentially amplify the vulnerability of people to high temperatures.[28] In addition, effective adaptation measures to climate change, such as the development of green infrastructure and air conditioning (AC) use, may help reduce vulnerability to heat.[28] In the context of climate change, investigating the effect modification of environmental and climate adaptation factors could help develop preventive measures against adverse pregnancy outcomes.

In this study, we aimed to examine the relationships between maternal exposure to ambient postpartum temperature and PPD within the time-to-event framework in a large pregnancy cohort in southern California and to explore potential effect modifiers. The primary hypothesis was that there are associations between postpartum temperature exposure and time to PPD; the temperature–PPD relationship varies according to air pollution levels, climate adaptation factors, and maternal characteristics.

Methods

Study Population

This retrospective cohort study included >430,000 women who had singleton births between 1 January 2008 and 31 December 2018 at Kaiser Permanente Southern California (KPSC) facilities. KPSC is a large integrated health care organization serving ~19% of the population in Southern California[29,30]; KPSC archives detailed information on demographics, individual lifestyles, medical records, fetal characteristics, and residential addresses. All maternal residential addresses at delivery in KPSC electronic health records (EHRs) were geocoded. Gestational age was determined by self-reported last menstrual period date and confirmed by ultrasonography, with ultrasound assessment considered to be the gold standard.[31] Participants who were not KPSC members at the time of pregnancy, had a gestational age of <20 or >47 wk ($n = 8{,}912$), were without a known residential address ($n = 680$), had had multiple births (e.g., twins and triplets, $n = 7{,}454$), or had had a stillbirth ($n = 1{,}961$) were excluded from the study. This study was approved by the KPSC Institutional Review Board with an exemption of informed consent.

Outcome: PPD

The clinical practice adopted by KPSC is based on the depression screening guidelines for all participants.[32] PPD was first assessed using the Edinburgh Postnatal Depression Scale (EPDS) for each participant during postpartum visits.[32] Patients who screened positive on the EPDS (score ≥10, suggesting minor or major depression) were referred to a clinical interview for further assessment and follow-up care, including diagnosis and treatment.[33] PPD ascertainment in this study was defined by using a combination of International Classification of Diseases, Clinical Modification (Ninth and Tenth Revisions) depression diagnostic codes and pharmacy records (Table 1) during the first 12 months postpartum.[34]

Exposure Assessment

Historical daily ambient temperature data (in degrees Celsius) from 2008 to 2018 at 4 km × 4 km resolution was obtained from the gridMET dataset (https://www.climatologylab.org/gridmet.html), which provides validated and publicly available daily surface fields of maximum temperature (T_{max}) and minimum temperature (T_{min}) covering the contiguous United States.[35] For creating individual postpartum temperature exposure, we assigned daily temperature values to each participant based on the geocoded residential address at delivery. We defined mean temperature as $T_{mean}[(T_{max} + T_{min})/2]$ and diurnal temperature range as $T_{range}(T_{max} - T_{min})$. We then calculated long-term postpartum temperature exposure by averaging temperatures during the period from delivery to the date of PPD diagnosis. Given that ~64% of PPD diagnoses occurred within the first 3 months after delivery, shorter exposure windows were not taken into account for the long-term analysis. We also examined relatively short-term postpartum temperature exposures by averaging temperatures during the 1-month and 2-wk periods prior to PPD diagnosis.

Potential Environmental Effect Modifiers

The effect modification of potential environmental and climate adaptation factors was examined at different levels (low <50th vs. high ≥50th percentile) of exposure to air pollution, green space, and AC penetration.

Historical daily ambient air pollution data for the years 2008–2018 were obtained from the US Environmental Protection Agency's monitoring stations, including particulate matter with an aerodynamic diameter of ≤2.5 μm ($PM_{2.5}$), and ozone (O_3) (an 8-h window of 1000 hours to 1800 hours). Monthly averaged concentrations were then calculated and spatially interpolated between stations using empirical Bayesian kriging (EBK). The cross-validation R^2 values of the EBK estimates for $PM_{2.5}$ and

Table 1. International classification of diseases (ICD) 9/10 diagnostic codes and medications used to ascertain postpartum depression diagnosis.

ICD-9-CM code	ICD-10-CM code	Medications
300.4	F32.9	Bupropion
309.0	F33.0	Celexa
311	F33.2	Citalopram
—	F33.3	Cymbalta
—	F33.41	Desvenlafaxine
—	F33.9	Duloxetine
—	F34.1	Effexor
—	F43.21	Escitalopram
—	F53.0	Fluoxetine
—	—	Lexapro
—	—	Paroxetine
—	—	Paxil
—	—	Pristiq
—	—	Prozac
—	—	Sertraline
—	—	Venlafaxine
—	—	Wellbutrin
—	—	Zoloft

Note: —, not applicable; ICD-9-CM, *International Classification of Diseases, Ninth Revision, Clinical Modification*; ICD-10-CM, *International Classification of Diseases, Tenth Revision, Clinical Modification*.

O_3 were 0.65 and 0.72, respectively.[36] Air pollution levels during the entire pregnancy were determined by averaging monthly measurements based on the residential histories (address, start date, and end date) of each participant; air pollution levels during the postpartum periods (from delivery to PPD diagnosis date) were estimated based on the residential address at delivery in KPSC EHRs.

A validated machine learning model developed in our previous work was applied to estimate continuous green space variables based on street view images within a 500-m radius around the residential address at delivery.[27,37] The accuracy of the model was high, with a 92.5% mean intersection over union.[37] Briefly, entire streetscape images from four different directions at locations every 200 m along the road network in the study region were obtained from the Microsoft Bing Maps Application Programming Interface. Approximately 99% of Bing streetside images used in this study were randomly captured in 2014–2015. The amount of green space was measured by averaging the proportion of greenery pixels in all street view images within the 500-m buffer zone for each residential address. We also used satellite-based green space measures, including the Normalized Difference Vegetation Index (NDVI), land-cover green space, and tree canopy cover. NDVI data were obtained from the Terra (MOD13Q1) satellite instrument of Moderate Resolution Imaging Spectroradiometer products from NASA. The annual mean NDVI was calculated by averaging the NDVI values (250 m × 250 m, every 16 d) in all grids within a 500-m circular buffer. Land-cover and tree canopy data (30 m × 30 m) were obtained from the National Land Cover Database (NLCD; 2013 version).[38] Natural environment-related categories from the NLCD were aggregated as one measure of land-cover–based green space, including forest, shrubland, herbaceous, wetlands, and developed open space (i.e., >80% vegetation cover). The percentages of the area within a 500-m circular buffer were calculated as land-cover green space and tree canopy exposures. Further details of the green space measurements have been previously described.[27]

AC penetration rates at the census tract level from 2015 to 2016 were derived according to hourly residential electricity records and daily average ambient temperature data.[39] Given that the AC datasets were only created for partial areas in California, only participants in census tracts with available AC penetration data were included in the AC analysis ($n = 241{,}136$). We further explored the impact of AC penetration among mothers living in climate zones[40] with more extreme heat episodes (i.e., California Climate Zones 9–16 among our study population),[41] who tended to use AC more often during hot weather.[42]

Statistical Analysis

The distribution of selected population characteristics and temperature metrics were assessed, and their association with PPD was assessed using chi-square tests for categorical variables and one-way analysis of variance for continuous variables. The association between PPD and temperature exposure was examined using time-to-event models, and non-PPD was censored at 12 months postpartum. To estimate time-varying associations between postpartum temperature exposure and PPD, we applied a discrete-time approach with pooled logistic regressions,[43] which can be used to relax the proportional hazards assumption and handle large datasets with time-dependent variables. In discrete-time logistic regression models, we included time (i.e., birth month) as a covariate in a flexible manner (polynomials), as suggested by prior research.[44] Adjusted odds ratios (aORs) and 95% confidence intervals (CIs) of PPD associated with per interquartile range (IQR; the 75th percentile minus the 25th percentile) increment of the temperature variable were estimated. Temperature metrics were also categorized into quintiles to test the assumption of linearity. To examine the longer-term temperature variability,[45] we used the standard deviation (SD) of daily temperature to capture temperature anomalies during the postpartum period.

Pregnancy-related covariates and potential confounders were selected *a priori* based on the existing literature.[46–48] In the primary analysis, we included maternal age, self-reported race/ethnicity (African American, Asian, Hispanic, non-Hispanic White, and others including Pacific Islanders, Native American/Alaska Native, and multiple race/ethnicities specified), self-reported educational level (≤8th grade, 9th grade to high school, college <4 y, college ≥4 y), census block group-level median household income in 2013 (categorized as quartiles),[49] season of conception (warm season: May–October, and cool season: November–April), and year of infant birth (2008–2018). Potential covariates were obtained from KPSC EHRs. Self-reported maternal race and ethnicity information was extracted from both the birth certificate records (primary source) and KPSC EHRs (secondary source to supplement the missing data). County was fitted as a random effect to account for potential PPD spatial clustering.

In the sensitivity analysis, we examined the influence of adjusting for other potential covariates, including insurance type (Medicaid or other, as an additional proxy for socioeconomic status), preterm birth and pregnancy complications (preeclampsia, gestational hypertension, and gestational diabetes),[50,51] method of delivery (cesarean section and vaginal delivery),[52] parity (primiparous and multiparous),[53] prepregnancy body mass index (BMI; underweight: <18.5 kg/m^2, normal: 18.5–24.9 kg/m^2, overweight: 25.0–29.9 kg/m^2, and obese: ≥30.0 kg/m^2, as a proxy for obesity),[54] and season of delivery (warm season: May–October, and cool season: November–April, as an alternative to season of conception). We also performed sensitivity analyses, adding air pollution and green space (as continuous exposure variables) and restricting the analyses to climate zones with more extreme heat episodes. Furthermore, we conducted subgroup analyses to examine the effect modification by maternal characteristics, including age, race/ethnicity, educational attainment levels, household income, prepregnancy BMI, parity, method of delivery, preterm birth, and maternal comorbidities. The heterogeneity among subgroups was assessed using Cochran's Q tests. Participants with missing or invalid data for the exposure (e.g., living in regions where temperature exposure could not be calculated) or confounding variables were excluded from the analysis. All analyses were conducted with SAS (version 9.4; SAS Institute, Inc.).

Results

A total of 429,839 singleton births (Figure S1) with 45,073 (10.49%) PPD cases at 1 y postpartum were included in our study. Among PPD cases, 15,443 (34.26%) had both PPD diagnosis and prescription medications, 11,433 (25.37%) were identified solely by diagnostic codes, and 18,191 (40.36%) were identified by supplemental pharmacy use records. The mean maternal age of the study population was 30.22 ± 5.75 y at delivery. Hispanic mothers accounted for 51.15% of the total population, followed by non-Hispanic White (26.10%), Asian (12.58%), African American (7.54%), and multiple races or other races and ethnicities (Multiple/other; 2.62%) mothers. Table 2 presents the distribution of population characteristics by PPD groups. As compared with women without the diagnosis of PPD, women with the diagnosis of PPD were more likely to be from non-Hispanic White, African American, and Multiple/other racial/ethnic groups. They also tended to have <4 y of college education or more than a college education, to live in middle- and high-income neighborhoods, to be overweight or obese, to have Medicaid insurance, to

Table 2. Population characteristics by postpartum depression status; KPSC cohort, 2008–2018.

Characteristics	PPD $n = 45{,}073$	Non-PPD $n = 384{,}766$	Total births $n = 429{,}839$	p-Value
Maternal age [y (mean+SD)]	30.93 ± 5.63	30.14 ± 5.76	30.22 ± 5.75	<0.001
Maternal race/ethnicity [*n* (%)]				
African American	3,667 (8.14)	28,752 (7.47)	32,418 (7.54)	<0.001
Asian	2,717 (6.03)	51,366 (13.35)	54,083 (12.58)	
Hispanic	22,157 (49.16)	197,705 (51.38)	219,862 (51.15)	
Non-Hispanic White	15,169 (33.66)	97,009 (25.21)	112,178 (26.10)	
Multiple/other races[a]	1,362 (3.02)	9,890 (2.57)	11,252 (2.62)	
Missing	1 (0.01)	45 (0.01)	46 (0.01)	
Maternal education [*n* (%)]				
<College	12,908 (28.63)	121,265 (31.51)	134,173 (31.22)	<0.001
<4 y college	11,836 (26.26)	85,269 (22.16)	97,105 (22.59)	
≥4 y college	13,317 (29.55)	120,612 (31.35)	133,929 (31.16)	
>College	6,101 (13.54)	50,052 (13.01)	56,153 (13.06)	
Missing	911 (2.02)	7,568 (1.97)	8,479 (1.97)	
Census block group-level maternal household income [*n* (%)]				
<$43,696	9,978 (22.14)	97,310 (25.07)	107,279 (24.96)	<0.001
$43,697–$55,962	11,254 (24.97)	95,712 (24.78)	106,966 (24.89)	
$55,963–$71,602	11,820 (26.22)	95,292 (24.66)	107,112 (24.92)	
≥$71,603	11,885 (26.37)	95,233 (24.65)	107,118 (24.92)	
Missing	136 (0.30)	1,228 (0.32)	1,364 (0.32)	
Prepregnancy BMI (kg/m^2) in categories [*n* (%)]				
Underweight (<18.5)	770 (1.71)	9,736 (2.53)	10,506 (2.44)	<0.001
Normal (18.5–24.9)	16,336 (36.24)	166,408 (43.25)	182,744 (42.51)	
Overweight (25.0–29.9)	12,700 (28.18)	107,052 (27.82)	119,752 (27.86)	
Obese (≥30.0)	15,031 (33.35)	98,317 (25.55)	113,348 (26.37)	
Missing	236 (0.52)	3,253 (0.85)	3,489 (0.81)	
Parity [*n* (%)]				
Primiparous	17,188 (38.13)	160,693 (41.76)	177,881 (41.38)	<0.001
Multiparous	27,811 (61.70)	223,566 (58.10)	251,377 (58.48)	
Missing	74 (0.16)	507 (0.13)	581 (0.14)	
Insurance type [*n* (%)]				
Medicaid	5,112 (11.34)	35,831 (9.31)	40,943 (9.54)	<0.001
Other	39,521 (87.68)	342,100 (88.91)	381,621 (88.88)	
Missing	440 (0.98)	6,835 (1.78)	7,275 (1.59)	
Method of delivery [*n* (%)]				
Cesarean section	14,889 (33.03)	110,981 (28.84)	125,870 (29.28)	<0.001
Vaginal delivery	30,147 (66.88)	273,589 (71.11)	303,736 (70.66)	
Missing	37 (0.08)	196 (0.05)	233 (0.05)	
Preterm birth or pregnancy-related comorbidities [*n* (%)][b]				
Yes	11,573 (25.68)	83,936 (21.81)	95,509 (22.22)	<0.001
No	33,500 (74.32)	300,830 (78.19)	334,330 (77.78)	
Season of conception [*n* (%)]				
Warm season (May–October)	21,825 (48.42)	190,269 (49.45)	212,094 (49.34)	<0.001
Cool season (November–April)	23,248 (51.58)	194,497 (50.55)	217,745 (50.66)	
Year of infant birth [*n* (%)]				
2008	2,897 (8.19)	32,489 (8.44)	35,386 (8.23)	<0.001
2009	2,924 (8.63)	30,966 (8.05)	33,890 (7.88)	
2010	2,937 (8.70)	30,824 (8.01)	33,761 (7.85)	
2011	3,129 (8.79)	32,466 (8.44)	35,595 (8.28)	
2012	3,309 (8.70)	34,745 (9.03)	38,054 (8.85)	
2013	3,987 (10.33)	34,626 (9.00)	38,613 (8.98)	
2014	4,656 (11.62)	35,410 (9.20)	40,066 (9.32)	
2015	4,821 (11.51)	37,048 (9.63)	41,869 (9.74)	
2016	4,749 (10.93)	38,696 (10.06)	43,445 (10.11)	
2017	4,986 (11.33)	39,013 (10.14)	43,999 (10.24)	
2018	6,678 (14.79)	38,483 (10.00)	45,161 (10.51)	
Climate zones [*n* (%)]				
Zones with extreme heat episodes	27,427 (60.85)	226,158 (58.78)	253,585 (59.00)	<0.001
Other	16,891 (37.47)	152,586 (39.66)	169,477 (39.43)	
Missing	755 (1.68)	6,022 (1.57)	6,777 (1.58)	
Postpartum temperature exposures [°C (mean ± SD)]				
Maximum	25.40 ± 4.54	25.11 ± 2.07	25.14 ± 2.45	<0.001
Minimum	12.49 ± 3.96	12.36 ± 1.82	12.38 ± 2.15	<0.001
Mean	18.94 ± 4.02	18.73 ± 1.49	18.76 ± 1.92	<0.001
Range	12.89 ± 2.96	12.67 ± 2.93	12.77 ± 2.56	<0.001

Note: Categorical variables are presented as *n* (%); continuous variables are presented as mean+SD. BMI, body mass index; KPSC, Kaiser Permanente Southern California; postpartum period, from delivery to the date of PPD diagnosis; PPD, postpartum depression; SD, standard deviation.

[a]Multiple/other: Pacific Islanders, Native American/Alaska Native and mothers with multiple race/ethnicities specified.

[b]Pregnancy-related comorbidities: preeclampsia, gestational hypertension, and gestational diabetes.

have a cesarean section delivery, to have a preterm birth or maternal comorbidities, and to be multiparous. In addition, temperature exposures during the postpartum period were higher among those with a PPD diagnosis than those without the diagnosis. Table 3 shows the summary statistics of the temperature metrics during the postpartum period. Maternal postpartum exposures to T_{max}, T_{min}, T_{mean}, and T_{range} [median (IQR)] were 24.18 (7.80), 11.84 (7.46), 18.06 (7.61), and 12.59 (4.23) °C, respectively. Table S1 shows the summary statistics of air pollutants, green space, and AC penetration rates.

Associations between Temperature and PPD

The associations between postpartum exposure to temperature and the risk of PPD are shown in Table 4. Elevated PPD risks were associated with postpartum exposure to higher temperatures and temperature ranges. For example, we observed a 7% increase in PPD risks per IQR rise in mean temperature exposure during the postpartum period aOR = 1.07; 95% CI: 1.05, 1.09] after controlling for confounders. The magnitude of associations was relatively stronger for maximum temperatures compared with minimum temperatures. Moreover, greater risks of PPD were observed to be associated with each IQR increase in temperature range (aOR = 1.08; 95% CI: 1.06, 1.10). The associations of temperature exposures with PPD were slightly decreased in magnitude in unadjusted models. We also examined temperature exposures during the 1-month and 2-wk periods prior to PPD diagnosis. The results were similar for the long-term temperature exposure from delivery to PPD diagnosis (Table S2). The results using categorized temperature metrics did not show nonlinear relationships; exposure to extreme low temperature (<5th percentile) was not associated with an elevated risk of PPD. We found no significant effect of postpartum temperature variability on PPD (Table S3).

In the sensitivity analyses (Table S3), associations of temperature exposures with PPD were slightly stronger after further adjusting for insurance type, pregnancy-related comorbidities (preeclampsia, gestational hypertension, and gestational diabetes), preterm birth, method of delivery, parity, prepregnancy BMI, residential surrounding green space, or antepartum and postpartum air pollution.

Effect Modification by Environmental Factors and Maternal Characteristics

Results of effect modification by environmental and climate adaptation factors are presented in Table 5. The observed positive associations between high temperature/temperature range and PPD were significantly stronger among mothers living in areas with higher air pollution exposure levels (Table S1; air pollutant concentrations ≥50th percentile: $PM_{2.5}$ ≥11.54 $\mu g/m^3$, O_3 ≥43.56 ppb), lower green space exposure (<50th percentile: NDVI <0.26, land-cover green space <5.06%) or lower AC penetration rates (<50th percentile: AC penetration rate <0.76). For instance, the adjusted OR of PPD per IQR increase in mean temperature was 1.09 (95% CI: 1.06, 1.13) for people who were exposed to the high $PM_{2.5}$ level and 1.04 (95% CI: 1.02, 1.07) for the low $PM_{2.5}$ level. Notably, we observed higher associations between exposure to an increased temperature range and PPD among subgroups living in census tracts with lower AC penetration rates, but not for mean temperature. Restricting the results to climate zones with high temperatures revealed a pattern similar to that of the entire study region (Table 5). We found effect modification by satellite-based total amount of green space (i.e., NDVI and land-cover green space), but not by residential street green space or tree canopy cover. Furthermore, the stratified analysis by maternal characteristics (Table 6; Table S4) showed higher associations between exposure to high temperature and PPD among mothers ≥25 years of age and racial/ethnic minority groups (African American, Asian, or Hispanic mothers). Cochran's Q tests did not reveal any significant heterogeneity across other maternal subgroups.

Table 3. Summary statistics of temperature exposure metrics during the postpartum period; KPSC cohort, 2008–2018.

Temperature (°C)	25th percentile	Median	75th percentile	IQR
Maximum	20.55	24.18	28.35	7.80
Minimum	8.51	11.84	15.97	7.46
Mean	14.57	18.06	22.18	7.61
Range	10.58	12.59	14.81	4.23

Note: IQR, interquartile range; KPSC, Kaiser Permanente Southern California; postpartum period, from delivery to the date of PPD diagnosis.

Table 4. Adjusted odds ratios (aORs) and 95% confidence intervals (CIs) of postpartum depression associated with postpartum ambient temperature exposure from delivery to PPD diagnosis date; KPSC cohort, 2008–2018.

Temperature exposure	aORs	95% CIs
Unadjusted models		
Maximum temperature	1.06	1.05, 1.08
Minimum temperature	1.02	1.01, 1.04
Mean temperature	1.05	1.03, 1.07
Temperature range	1.06	1.05, 1.08
Adjusted models		
Maximum temperature	1.08	1.06, 1.10
Minimum temperature	1.04	1.02, 1.06
Mean temperature	1.07	1.05, 1.09
Temperature range	1.08	1.06, 1.10

Note: aORs and 95% CIs were calculated for per interquartile range increment for temperature; models were adjusted for maternal age, race/ethnicity, education, household income, season of conception, and year of birth; county was fitted as a random effect. KPSC, Kaiser Permanente Southern California.

Discussion

In this large pregnancy cohort in southern California from 2008 to 2018, we found that maternal postpartum exposure to high temperature and diurnal temperature variability was associated with increased PPD risks during the first year of the postpartum period. We also observed the effect modification by maternal age, race and ethnicity, air pollution levels, green space, and AC penetration rates.

Temperature exposure, especially heat, has long been known to have an association with adverse pregnancy outcomes, including preterm birth, gestational diabetes mellitus, severe maternal morbidity, and premature rupture of membranes.[41,55–58] For maternal mental health, only one study, with 1,931 pregnant women in Shanghai,[23] found that exposure to extremely low (<5th percentile) and high (≥95th percentile) temperatures and an increased temperature range were associated with emotional stress levels during pregnancy; a U-shaped relationship was observed between daily mean temperatures and maternal stress scores. To our knowledge, no previous study has specifically investigated temperature exposure and PPD. Our results are partially consistent with previous findings, where high ambient temperature and temperature range were found to adversely impact maternal mental health. However, we did not observe a nonlinear relationship between postpartum exposure to temperature and PPD risk or a significantly elevated risk of PPD associated with extreme low temperature in our analysis. Although both the study by Lin et al.[23] and the present study assessed maternal mental health and ambient temperature exposure, the difference in results might be explained by many reasons. For example, we focused on the postpartum period and defined PPD based on clinical diagnosis rather than antepartum emotional stress from a

Table 5. Adjusted odds ratios (aORs) and 95% confidence intervals (CIs) of postpartum depression associated with postpartum ambient temperature exposure stratified by green space, air pollution, and air conditioning penetration; KPSC cohort, 2008–2018.

Description	Low (<50th percentile)		High (≥50th percentile)		*p*-Value for Cochran's *Q* test
	aORs	95% CIs	aORs	95% CIs	
Air pollution					
$PM_{2.5}$ (median = 11.54 μg/m^3)					
Maximum temperature	1.06	1.03, 1.09	1.10	1.07, 1.13	0.04
Minimum temperature	1.02	0.99, 1.04	1.07	1.03, 1.10	0.02
Mean temperature	1.04	1.02, 1.07	1.09	1.06, 1.13	0.03
Temperature range	1.07	1.05, 1.10	1.11	1.07, 1.14	0.09
O_3 (median = 43.56 ppb)					
Maximum temperature	1.09	1.06, 1.13	1.07	1.04, 1.09	0.24
Minimum temperature	1.03	1.00, 1.07	1.07	1.04, 1.10	0.03
Mean temperature	1.03	1.00, 1.07	1.09	1.07, 1.11	<0.01
Temperature range	1.08	1.05, 1.11	1.06	1.03, 1.09	0.20
Green space within 500 m					
Street green space (median = 24.37%)					
Maximum temperature	1.08	1.05, 1.11	1.08	1.05, 1.11	0.52
Minimum temperature	1.05	1.02, 1.08	1.03	1.00, 1.06	0.45
Mean temperature	1.07	1.04, 1.10	1.07	1.04, 1.10	0.74
Temperature range	1.08	1.05, 1.11	1.09	1.06, 1.12	0.46
NDVI (median = 0.26)					
Maximum temperature	1.11	1.08, 1.14	1.05	1.03, 1.08	<0.01
Minimum temperature	1.05	1.02, 1.08	1.03	1.00, 1.06	0.35
Mean temperature	1.09	1.06, 1.12	1.05	1.02, 1.08	0.04
Temperature range	1.10	1.08, 1.13	1.06	1.03, 1.09	0.03
Land-cover green space (median = 5.06%)					
Maximum temperature	1.11	1.08, 1.15	1.04	1.02, 1.07	<0.01
Minimum temperature	1.05	1.01, 1.09	1.04	1.01, 1.06	0.58
Mean temperature	1.09	1.06, 1.13	1.04	1.02, 1.07	0.02
Temperature range	1.10	1.07, 1.13	1.03	1.01, 1.06	<0.01
Tree canopy cover (median = 1.27%)					
Maximum temperature	1.07	1.04, 1.10	1.10	1.07, 1.13	0.17
Minimum temperature	1.03	1.00, 1.06	1.05	1.02, 1.08	0.35
Mean temperature	1.06	1.03, 1.09	1.09	1.05, 1.12	0.24
Temperature range	1.09	1.06, 1.12	1.10	1.07, 1.13	0.14
Air conditioning penetration (median = 0.76)					
All regions					
Maximum temperature	1.08	1.06, 1.11	1.07	1.03, 1.10	0.34
Minimum temperature	1.04	1.01, 1.06	1.06	1.03, 1.10	0.13
Mean temperature	1.06	1.04, 1.09	1.08	1.04, 1.11	0.49
Temperature range	1.09	1.07, 1.11	1.01	0.97, 1.05	<0.01
Zones with extreme heat episodes					
Maximum temperature	1.05	1.02, 1.08	1.06	1.02, 1.09	0.69
Minimum temperature	1.02	0.99, 1.06	1.06	1.03, 1.09	0.14
Mean temperature	1.03	1.00, 1.07	1.07	1.03, 1.10	0.18
Temperature range	1.11	1.07, 1.15	1.01	0.96, 1.06	<0.01

Note: aORs and 95% CIs were calculated for per interquartile range increment for temperature; models were adjusted for maternal age, race/ethnicity, education, household income, season of conception, and year of birth; county was fitted as a random effect. KPSC, Kaiser Permanente Southern California; NDVI, Normalized Difference Vegetation Index; O_3, ozone; $PM_{2.5}$, particulate matter with an aerodynamic diameter of ≤2.5 μm.

one-time interview/questionnaire. We measured the effect of relatively chronic exposure (up to 1 y postpartum and monthly exposure) rather than acute exposure (weekly and daily exposure). In addition, the population, climate, season, adaptation to environments in local inhabitants, and the utilization of AC are very different in Shanghai compared with our study region of southern California.[59] Furthermore, with the effects of climate change, exposure to extreme temperature values may negatively impact PPD without major differences in mean temperatures. We explored the relationship between extreme temperature (the proportion of days above the 90th percentile for maximum temperature from delivery to the PPD diagnosis date) and PPD, and we found that exposure to extremely high temperature was associated with an increased risk of PPD (aOR = 1.04; 95% CI: 1.02, 1.06, per IQR increase). Epidemiological evidence for temperature-related PPD risk is sparse, and the effects of temperature on maternal mental health are still unclear, warranting future studies.

Our results suggest that temperature's impact on PPD was modified by air pollution exposure. Temperature-associated PPD risks were significantly higher among mothers with more severe exposure to $PM_{2.5}$ and O_3. Previous research has evaluated the associations of air pollution with mental health[46,60] and reported that air pollutants may modify the association between temperature exposure and mental disorders.[61] However, existing results are mixed. Recently, there is increasing evidence that concurrent heat stress and air pollution may exacerbate adverse pregnancy outcomes, including preterm birth and premature rupture of membranes.[41,62] Air pollution and heat may plausibly have synergistic harmful impacts on health through common biological pathways, including oxidative stress and systemic inflammation in the body. In addition, raising ambient temperature may stimulate a greater exposure to hazardous air pollutants through heat-induced sweating, increased skin blood flow, and pulmonary ventilation.[63] To our knowledge, no prior study has explored whether air pollution could modify the association between temperature and PPD. Our findings suggest that better air quality may not only reduce air pollution-induced PPD risks but also alleviate adverse impacts of temperature on PPD.

Table 6. Adjusted odds ratios (aORs) and 95% confidence intervals (CIs) of postpartum depression associated with postpartum ambient temperature exposure stratified by maternal age and race/ethnicity; KPSC cohort, 2008–2018.

Characteristics	aORs per IQR air pollutant metrics	95% CIs	*p*-Value for Cochran's *Q* test
Maternal age			
Maximum temperature			0.37
<25	1.05	1.01, 1.08	
25–34	1.07	1.05, 1.10	
≥35	1.08	1.05, 1.12	
Minimum temperature			0.04
<25	1.03	1.00, 1.07	
25–34	1.06	1.03, 1.08	
≥35	1.05	1.01, 1.08	
Mean temperature			0.34
<25	1.04	1.00, 1.08	
25–34	1.07	1.05, 1.10	
≥35	1.07	1.04, 1.11	
Temperature range			0.04
<25	1.05	1.01, 1.09	
25–34	1.07	1.04, 1.09	
≥35	1.09	1.06, 1.13	
Maternal race/ethnicity			
Maximum temperature			0.03
African American	1.12	1.05, 1.19	
Asian	1.11	1.04, 1.18	
Hispanic	1.09	1.06, 1.12	
Non-Hispanic White	1.03	1.00, 1.07	
Multiple/other	1.05	0.95, 1.16	
Minimum temperature			<0.01
African American	1.02	0.95, 1.09	
Asian	1.10	1.02, 1.19	
Hispanic	1.08	1.04, 1.11	
Non-Hispanic White	0.99	0.96, 1.03	
Multiple/other	0.97	0.86, 1.08	
Mean temperature			
African American	1.08	1.01, 1.16	0.01
Asian	1.14	1.05, 1.24	
Hispanic	1.09	1.06, 1.12	
Non-Hispanic White	1.02	0.98, 1.05	
Multiple/other	1.02	0.91, 1.14	
Temperature range			0.05
African American	1.15	1.08, 1.21	
Asian	1.08	1.01, 1.15	
Hispanic	1.06	1.03, 1.09	
Non-Hispanic White	1.06	1.03, 1.09	
Multiple/other	1.09	1.01, 1.17	

Note: aORs and 95% CIs were calculated for per IQR increment for temperature; models were adjusted for maternal age, race/ethnicity, education, household income, season of conception, and year of birth; county was fitted as a random effect. IQR, interquartile range; KPSC, Kaiser Permanente Southern California; Multiple/other, Pacific Islanders, Native American/Alaska Native and mothers with multiple race/ethnicities specified.

It is noteworthy that although no effect modification by street view-based green space was observed, we found higher temperature–PPD risks among mothers with lower exposures to the total amount of green space based on the satellite imagery (i.e., NDVI and land-cover green space). Compared with street view-based eye-level green space or tree coverage that may directly impact PPD,[27] remote sensing images capture the total amount of green space within a certain area that may provide more cooling benefits for their surroundings.[64] Our findings also suggest that various green space metrics from different sources may reflect different facets of natural environments and should be carefully used and interpreted in environmental health studies.

Regarding AC use, previous studies have found that ownership and usage of AC significantly reduced the effect of (especially high) temperature on the risk of multiple diseases, including all-cause mortality, cardiovascular diseases, respiratory diseases, dehydration, and heat stroke.[65,66] For mental health, a study from California regarding temperature exposure and three mental health outcomes (i.e., suicides, emergency department visits for mental illness, and self-reported days of poor mental health) reported that the negative effects of high temperatures on mental health remain stable across AC adoption levels.[67] That study used county-level outcome and exposure rather than individual-level data and spanned a long period of time (1960–2016); during the more than five decades in which that study was performed, both climate change-related weather phenomena and AC usage may have changed substantially, potentially leading to ambiguous results. In addition, other studies did not consider the exposure to temperature range. In our study, analogously, we found no evidence of statistically significant effect modification by AC penetration on the identified relationship between high temperatures and PPD, but our results revealed that an increased diurnal temperature range might be more harmful to mothers with lower AC penetration rates. Therefore, AC use could be a potential adaptation strategy for climate change to regulate temperature variation and reduce the risk of PPD.

Previous studies have shown that high temperatures may increase HPA axis activity and subsequent hormone release.[16,18,68] An overactivated HPA axis leads to an increased release of cortisol, the body's stress hormone, which may result in a poor stress response.[7,69,70] In addition, the sympathetic nervous system (SNS) begins to increase vasodilation and sweating to cool both the mother and the fetus when the ambient temperature is greater than core body temperature.[58,71] The activated SNS also stimulates the HPA axis, triggering physical responses to stress.[5,7] Another potential mechanism may be due to the disruption of the BBB, which has been associated with several neurological disorders, such as depression.[19,72–74] The BBB separates the brain from the rest of the body in addition to regulating the transport of molecules into the brain. BBB dysfunction under heat stress has been reported.[16,17] This is potentially due to heat stress decreasing the levels of claudin-5, which is a BBB tight junction protein that plays a role in the BBB's structural integrity; decreased tight junction expression can lead to higher BBB leakage.[17,72–74] Higher levels of inflammatory cytokines during pregnancy also contribute to a weakened BBB.[19,75,76] Therefore, increased BBB permeability makes the brain more vulnerable to peripheral immune cells, pathogens, and other toxins[16,73,75] that can enter the brain and cause excessive activation of brain structures, including the HPA axis. Furthermore, core body temperature varies until 1 y postpartum.[25] Mothers already have difficulty regulating core temperature after delivery, so high temperature and large temperature differences may lead to an exaggerated physiological response in postpartum mothers (Figure S2).

Recently, a global study with 47,628 participants across 68 countries also suggested that climate change and warmer temperatures harm human mental health through behavioral changes; increases in temperatures and the diurnal temperature range can lead to disrupted sleep patterns and sleep loss, which may contribute to poor mental health, especially for vulnerable populations, such as the elderly and women.[77] Continued research into the mechanisms regarding temperature and PPD and the effect modification of environmental factors and climate adaptation strategies is warranted to help improve maternal mental health.

Strengths

To the best of our knowledge, this is the first study providing evidence of the associations between postpartum temperature exposure and the risk of PPD. We applied a series of temperature metrics for different time scales to reflect various aspects of temperature exposure and investigated their impacts on PPD risk that may provide insights into PPD etiology and prevention. Other strengths include the high-quality clinical data based on KPSC

EHRs, including PPD ascertainment and diagnosis dates, the large and diverse population from the KPSC pregnancy cohort, and the rich and detailed information on individual- and neighborhood-level covariates that allowed us to adjust for a wide range of potential confounders and extensively examine the effect modification by maternal characteristics, as well as modifiable environmental factors.

Limitations

Some limitations of this study need to be acknowledged. First, exposure misclassifications may have existed given that postpartum temperatures were estimated at a relatively coarse resolution (4 km), and based solely on the maternal address at delivery due to unavailable residential mobility data during the postpartum period. Nevertheless, the estimated exposure levels may not change substantially given that most of the women who moved may likely have relocated within the same subregion (median distance: 6 km) based on the residential history during pregnancy among this population.[78] Another source of potential exposure misclassification could be the lack of information on time–activity patterns and individual-level monitors to account for indoor or workplace exposures. Second, although a series of standard procedures were implemented to ensure accuracy in PPD ascertainment (e.g., unified screenings, clinical interviews, pharmacy records, multiple follow-ups), it is inevitable that a proportion of PPD cases may not have been identified and treated timely[8,79] or that patients' choices to not attend further assessment may have led to an underestimation of PPD, temporal mismatches in exposure, and biased association estimates. Third, we used the AC penetration data at the census tract level in 2015 and 2016 as a proxy of AC use, which cannot account for temporal changes and energy insecurity[80] or reflect individual accessibility of AC given that AC estimations were not specific to households. Highly resolved household-level data of AC usage (e.g., the frequency, duration, intensity of use) would enable a more accurate analysis of how AC usage might moderate health outcomes in future studies. Furthermore, although our analyses considered several covariates and modifiers, residual confounding, unmeasured modifiers, and PPD-related factors (e.g., psychiatric history, adverse life events) may also exist owing to data unavailability, biasing the estimates, which deserves future investigation. Finally, studies in other geographical settings and populations are warranted because PPD-related risk factors[81] and climate features could vary significantly across different regions.

Conclusions

In this comprehensive analysis of temperature effects on PPD, as well as the effect modification by environmental factors, climate adaptation measures, and maternal characteristics, we found positive associations between PPD and exposure to higher temperatures and temperature ranges during the postpartum period. Targeted interventions might be beneficial for mothers exposed to higher temperatures and temperature ranges, especially for African American, Asian, and Hispanic mothers, older mothers, and mothers living in regions with higher air pollution, lower green space levels, or lower AC penetration rates.

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