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Living Near Wildfires and the Risk of Fetal Congenital Heart Defects: Evaluating Critical Windows of Vulnerability

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Abstract

Background Wildfires produce air pollutants that have been associated with complications during pregnancy. This study examined the association between wildfire exposure before and during pregnancy and the odds of congenital heart defect (CHD) in the offspring.

Methods This retrospective cohort study used the California Linked Birth File and the Forestry and Fire Protection data between 2007 and 2010. Patients living within 15 miles of wildfire during pregnancy were considered exposed. Multivariate logistic regression models were used to estimate the association between wildfire exposure by these various exposure metrics and atrial septal defect (ASD) or ventricular septal defect (VSD) types of CHD compared to pregnancies without wildfire exposure.

Results Compared to births without wildfire exposure, those with first-, second-, and third-trimester exposure were associated with a higher risk of ASD with a first-trimester adjusted odds ratio (aOR) of 1.11 (95% confidence interval (CI): 1.04–1.18), second-trimester aOR of 1.12 (95% CI: 1.07–1.18), and third-trimester aOR of 1.08 (95% CI: 1.02–1.14). Wildfire exposure during the critical window of fetal heart development (weeks 3–8) was associated with aOR of 1.12 (95% CI: 1.02–1.23).

Conclusion Wildfire exposure during pregnancy appears to increase the risk of developing ASD.

Keywords
- wildfire
- congenital heart defect
- pregnancy
- air pollution

Key Points

- Wildfire exposure during critical periods in pregnancy are associated with congenital cardiac malformation.
- Pre-pregnancy exposure to wildfire is not associated with increased risk of congenital cardiac malformation.
- Pregnant individuals should avoid wildfire exposure.

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Introduction

The impact of climate change has expanded regions vulnerable to devastating wildfires and lengthened wildfire season rapidly across the world. In recent years, California wildfires have increased in frequency and widened their destructive impact on the environment and human health.[1] Many studies have demonstrated an association between exposure to toxic byproducts from wildfires and the development of a wide spectrum of adverse health outcomes, including respiratory and cardiovascular diseases as well as a few pregnancy-related complications. As a result, more and more reproductive-aged individuals are exposed to the toxic effects of wildfire during and immediately before pregnancy, demonstrating the need for a better understanding of the teratogenic consequences of wildfire exposures.[2]

There is limited published literature on the evaluation of wildfire exposure during pregnancy and its potential impact on birth defects specifically. For example, one study found that exposure to wildfire smoke in Brazil was associated with a slightly higher risk of cleft lip/palate and anomalies of the respiratory and nervous systems.[3] Our research group recently published findings on the association between California wildfire exposure and fetal gastroschisis risk and spina bifida.[4,5] Other than these two published studies on wildfire exposure, other environmental studies have reported on the association between specific environmental pollutants more broadly in ambient air pollution exposure and birth defects.[6]

One systematic review and meta-analysis found an association between particulate matter 10 microns (PM_{10}) and fetal atrial septal defects (ASDs).[7] The same study also reported an association between sulfur dioxide and nitrous dioxide exposure and risks of aortic coarctation and tetralogy of Fallot. Congenital heart disease is the most common birth defect, affecting approximately 1% of live births, and ASD and ventricular septal defect (VSD) are the most common types of congenital heart defect (CHD).[8] The etiology of CHDs remains multifactorial, with 15% attributed to genetic causes and up to 30% linked to environmental agents.[9]

Currently, it is unclear if these defects form only during organogenesis or if they could develop later in fetal life from possible cellular injuries from environmental exposure. Therefore, it is unclear how wildfire exposure could potentially impact the formation of CHD. During a wildfire event, large quantities of the same air pollutants associated with congenital cardiac defects are rapidly released into the surrounding environment. Therefore, we investigated whether prenatal exposure to wildfire was associated with a higher risk of congenital cardiac defects.

Materials and Methods

Cohort Selection

This retrospective cohort study used the California Office of Statewide Health Planning and Development (OSHPD) Linked Birth File linked to the California Department of Forestry and Fire Protection (CAL FIRE) data between 2007 and 2010. Due to the computational demand of the statistical analyses and the available computer hardware for this project, we limited our data range to 4 years, using the period of 2007 to 2010. This period included the highest recorded number of wildfire events in California with available Linked Birth File data. The OSHPD Linked Birth File captured all births among Californian residents. It included birth and death certificate information linked to patient discharge data up to 1 year from delivery. Furthermore, it included maternal and neonatal diagnoses and procedure codes based on the International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM). Analyses were limited to birth records with linked ICD-9-CM codes and primary residence zip codes within California. The CAL FIRE wildfire data included the start and end dates of every wildfire recorded in California as well as the corresponding zip codes affected by the fire.

Exposure

There are various components of wildfire pollution that could potentially lead to ASD and VSD development.[10] Wildfires can have different rates and ranges of dispersion influenced by local weather conditions. Therefore, we chose not to use spatial mapping of specific air quality indexes to determine exposure. Instead, exposure was defined more generally as the mother's primary residence zip code within 15 miles of the closest edge of a wildfire.

The exposure was further stratified into first-trimester exposure (>0 to 13 and 6/7 weeks), second-trimester exposure (14 and 0/7 to 27 and 6/7 weeks), and third-trimester exposure (≥28 weeks) based on the first wildfire exposure in pregnancy. The period of gestation was calculated based on the obstetric estimate of gestational age at delivery and date of delivery. Despite the first 2 weeks of pregnancy being the pre-conception period, we included this period in the exposure risk due to the possibility that any environmental chemical exposure may have prolonged tissue effects due to slow physiologic clearance. Additionally, we assessed the effect of pre-pregnancy wildfire exposure on ASD and VSD by comparing pregnancies with wildfire exposure within 30 days prior to pregnancy to those without any wildfire exposure. Because ASD and VSD formation is an early embryologic event, we counted the earliest wildfire exposure in stratification based on pregnancy epochs in cases where the pregnancy was exposed to multiple fires. Critical window of fetal heart development was defined as between 3 and 8 gestational weeks.[11]

Outcome

Pregnancies complicated by CHDs were identified by neonatal ICD-9-CM codes. Code 745.5 was used for ASD, and code 745.4 was used for VSD. The diagnosis of ASD and VSD was not mutually exclusive.

Statistical Analysis

Maternal demographic information was compared between pregnancies with and without ASD and VSD. Maternal BMI was compared as a continuous variable. Tobacco use during pregnancy was categorized as the trimester of earliest reported use. Race and ethnicity were based on birth records and categorized into non-Hispanic White, non-Hispanic

Black, Latino, or Asian/Pacific Islander. For continuous variables, normality was assessed by a histogram plot. The impact of seasonal climate changes, regional baseline pollution, and socioeconomic status variations were accounted for in a separate regression model incorporating the month of birth and data from the California Environmental Protection Agency's CalEnviroScreen data.[12]

The rates of VSD and ASD were compared between pregnancies with and without wildfire exposure. Multivariable log-binomial regression analyses were performed to estimate the association between wildfire exposure in each pregnancy epoch and CHDs. The regression models were adjusted for potential confounding variables. First, we determined a priori factors that were associated with wildfire and CHDs based on the previous literature and possible clinical mechanisms. We conducted univariate analyses with potential confounders with wildfire exposure and CHDs (ASD and VSD). Confounders were included in the model if univariate analyses of confounders with both wildfire exposure and CHDs were statistically significant ($P < 0.05$). Therefore, the final models were adjusted for maternal age, pre-pregnancy BMI, education level, race/ethnicity, maternal tobacco exposure, gestational age, regional baseline pollution level, Down's syndrome, and lupus. All analyses were performed using Stata 15 (College Station, TX).

This study was approved by California State University – Fullerton Institutional Review Board (IRB# HSR-19-20-548).

Results

Between 2007 and 2010, 2,127,970 births were registered and 1,205 wildfires recorded. Of these births, records missing either mom ID ($n = 10,425$) or child ID ($n = 24,360$) were excluded because they could not be linked to ICD-9-CM codes. Also, 10,425 birth records missing maternal diagnosis codes and 24,360 missing neonatal diagnosis codes were excluded. The remaining 2,093,185 births, including 12,790 ASD cases and 7,682 VSD cases, were included in the analysis (►**Fig. 1**).

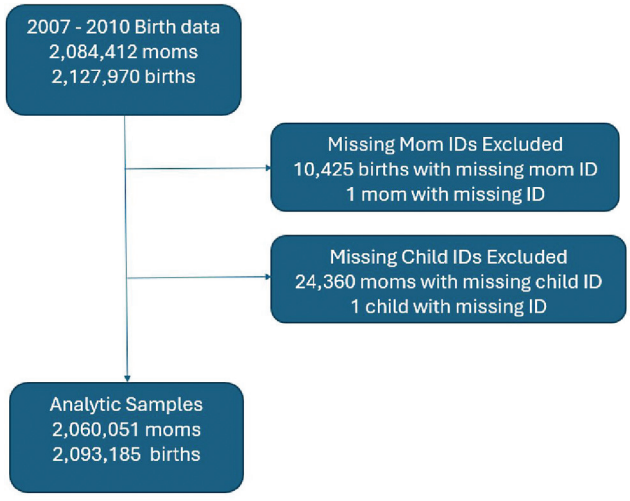

Fig. 1 Study participants and exclusion criteria.

Mothers of children with ASD and VSD had a lower rate of college education (26.7 and 27.5%) when compared to mothers of non-affected children (28.6%). These mothers also had higher rate of public health insurance use (53% and 51% versus 49%), and a higher mean pre-pregnancy BMI (25.8 and 26.5 versus 26.4) when compared to those mothers with children without ASD or VSD. In terms of medical risk factors, the differences in rates of pre-gestational and gestational diabetes, lupus, and Down's syndrome showed statistically significant between exposure and control groups (►**Table 1**).

There were 12,790 ASD cases and 7,682 VSD cases. The rate of ASD per 10,000 births was 59.5 in the group without wildfire exposure. The rates of ASD per 10,000 births were 78.4, 65.4, and 50.8 for first-, second-, and third-trimester exposures, respectively. When we examined wildfire exposure during the critical period (between gestational weeks 3 and 8), the rate of ASD was 83.7 per 10,000 births. Additionally, when women were exposed to wildfire within 30 days prior to pregnancy, the rate of ASD was 100.5 per 10,000 births (►**Fig. 2**), while the rate of VSD per 10,000 births was 36.6 in the group without wildfire exposure. The rates of VSD per 10,000 births were 40.3, 37.2, and 34.2 for first-, second-, and third-trimester exposures, respectively. The VSD rate was 40.9 among mothers who were exposed to wildfires during the critical period. Additionally, when women were exposed to wildfire within 30 days prior to pregnancy, the rate of VSD was 44.7 per 10,000 births (►**Fig. 2**).

Compared to births without wildfire exposure, pregnancies exposed to wildfire across pregnancy were associated with a higher risk of ASD in the first trimester (adjusted odds ratio [aOR] 1.11, 95% confidence interval [CI] 1.04, 1.18), second trimester (aOR 1.12, 95% CI 1.07, 1.18), and third trimester (aOR 1.08, 95% CI 1.02, 1.14). Narrowing births to those with wildfire exposure during the critical period of cardiovascular development consistently showed higher risk (aOR 1.12, 95% CI 1.02, 1.23). In contrast, wildfire exposure during pregnancy was not associated with VSD risk for any trimester (►**Table 2**).

Discussion

This large retrospective cohort study demonstrates that wildfire exposure during pregnancy significantly increased the risk of fetal ASD but not VSD for each trimester. Specifically, after controlling for potential confounders, wildfire exposure during the critical period in pregnancy was associated with a 12% higher risk of ASD compared to pregnancies without wildfire exposure. Furthermore, the risk of ASD was 11, 12, and 8% higher in pregnancies exposed to wildfire in the first, second, and third trimesters, respectively. This study indicates that the antenatal period is particularly susceptible to exposure and has long-term consequences on their future health.

Fetal cardiac structural and functional development is complex. While early morphogenetic stages of development show rapid structural formation after implantation during organogenesis period, the cardiac structures continue to undergo remodeling and maturation well into the third

Table 1 Participant characteristics

	n = 2,074,983		*n* = 12,790			*n* = 7,682		
	No Congenital heart defects		ASD		Test*	VSD		Test*
Variable	Mean	Std. dev.	Mean	Std. dev.		Mean	Std. dev.	
Maternal age	28.19	6.35	29.08	6.78	<0.001	29.23	6.80	<0.001
Paternal age	31.04	7.33	31.81	7.79	<0.001	31.86	7.64	<0.001
Birthweight (g)	3,302.37	564.67	2,812.51	1,044.85	<0.001	3,005.78	866.27	<0.001
Gestational age at birth	38.29	3.99	35.76	5.53	<0.001	36.96	4.78	<0.001
Maternal pre-pregnancy BMI	25.78	5.89	26.59	6.50	<0.001	26.39	6.49	<0.001
	%		%			%		
Male	51.22		51.94		0.005	46.68		<0.001
BMI category					<0.001			<0.001
≥19 to <25	43.44		39.38			41.12		
≥25 to <30	23.28		23.06			23.22		
≥30 to <35	11.15		12.49			11.25		
≥35 to <40	4.47		6.04			5.49		
≥40	11.97		13.67			13.71		
< 19	5.69		5.35			5.21		
Payment					<0.001			<0.001
MediCal/Govt	49.07		53.02			51.44		
No prenatal care	0.48		0.82			0.49		
Private/Self	48.78		45.15			46.76		
Other	1.67		1.01			1.3		
Prenatal smoke					0.004			0.93
No smoke	94.66		94.18			94.66		
Pre-pregnancy smoke	1.18		1.09			1.12		
Prenatal smoke	0.37		0.49			0.35		
Other	3.78		4.25			3.87		
Pre-gestational diabetes	0.76		3.32		<0.001	3.74		<0.001
Gestational diabetes	0.82		3.49		<0.001	3.91		<0.001
Lupus	0.11		0.33		<0.001	0.23		0.002
Downs	0.11		5.02		<0.001	5.71		<0.001
Mom education					<0.001			<0.001
< High school	25.35		26.77			27.51		
High school grad	42.67		41.67			40.82		
College grad & above	28.64		27.56			27.48		
Unknown	3.33		4.00			4.19		
Mom Race/Ethnicity					<0.001			<0.001
Non-Hispanic White	45.12		43.64			46.91		
Non-Hispanic Black	0.16		0.19			0.14		
Hispanic	34.89		37.11			35.45		
Asian	12		10.14			9.15		
Other	6.97		7.94			7.49		

Abbreviations: ASD, atrial septal defect; VSD, ventricular septal defect.
Note: *Compared to no congenital heart defect.

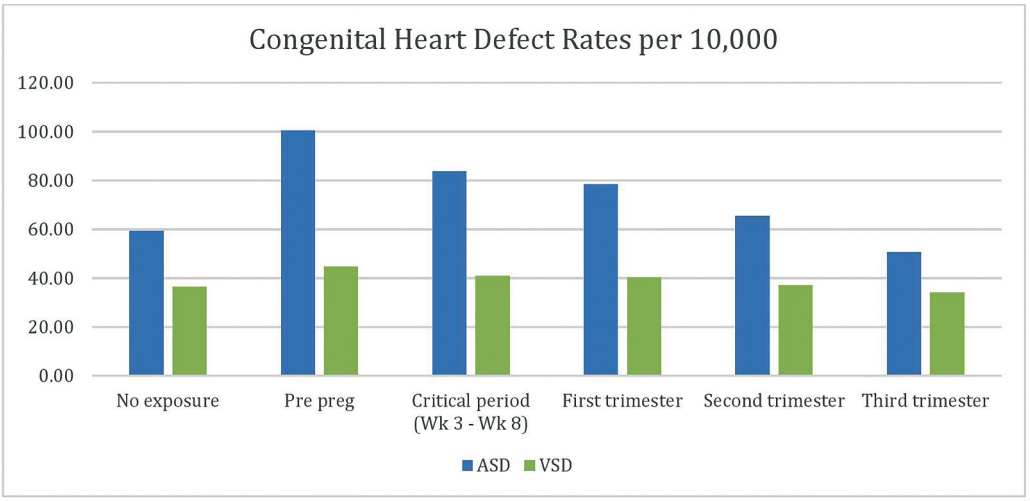

Fig. 2 Incidence of congenital heart defect by windows of vulnerability. ASD, atrial septal defect; VSD, ventricular septal defect.

Table 2 Pre-pregnancy and prenatal wildfire exposure and ASD and VSD risk adjusting for confounders

		OR	*P*	95% CI
Atrial septal defect (ASD)	Pre-pregnancy	1.02	0.79	0.86–1.23
	First trimester	1.11	0.00	1.04–1.18
	Second trimester	1.12	0.00	1.07–1.18
	Third trimester	1.08	0.01	1.02–1.14
	Critical period, week 3–week 8	1.12	0.02	1.02–1.23
Ventricular septal defect (VSD)	Pre-pregnancy	0.91	0.45	0.70–1.17
	First trimester	0.99	0.89	0.92–1.08
	Second trimester	0.99	0.83	0.93–1.06
	Third trimester	1.05	0.16	0.98–1.13
	Critical period, week 3–week 8	0.98	0.73	0.86–1.11

Note: *Adjusted for gestational age, pre-pregnancy BMI, maternal age, prenatal smoke, maternal education, maternal race, lupus, Downs, rubella, gestational diabetes, and pregestational diabetes.

trimester.[13] The complexity of cardiac development helps explain the current study's observation that exposure to wildfire during all three trimesters of pregnancy are associated with ASD detection at birth.

These findings also present many challenges for risk-reduction strategies in reproductive age individuals exposed to wildfire pollutants. Many women may not be aware of their pregnancy during this time frame. Due to the early nature of this embryological event and the lack of data on how long the effects of pollutants persist within maternal tissues after exposure, it is possible that damage inflicted by wildfire-related contaminants, or the toxins themselves, can persist in maternal respiratory and circulatory systems, causing congenital defects in future gestations.[14,15] This is consistent with the findings of increased rates of ASD in women exposed to wildfires in the 30 days prior to confirmation of pregnancy. Furthermore, it is also not feasible for many women to relocate in order to avoid wildfire exposure for the entire pregnancy.

With the development of cardiopulmonary bypass, congenital heart disease is no longer a fatal diagnosis, but the epidemiology of CHD will inform the study of disease etiology, its impact on global health outcomes, and the implementation of health policy.[16] According to the Global Burden of Disease Study, the incidence of CHD among males was 19.1 per 1,000 in 2017, while the incidence rate in females was 16.6 per 1,000 in 2017. CHD incidence rate trends vary across socio-demographic index (SDI) regions and disease subtype. From 1990 to 2017, incidence rates for CHD experienced a downward trend, except CHD incidence rates in high SDI regions, which increased from 12.4 per 1,000 to 12.6 per 1,000 from 1990 to 2017. The upward trend of CHD incidence in high SDI regions was attributed to the increased incidence rates of ASD and VSD.

The particularly harmful elements of wildfire smoke are related to the biomass and manmade structures that are burned. This creates a specific air composition of particulate matter. Wildfire smoke produces a toxic mixture of particles and chemicals, gases such as carbon monoxide and nitrogen oxide, and hydrocarbons and volatile organic matter.[17] These hazardous components have been linked to many negative

effects on affected populations' health, such as compromising respiratory health including asthma, cardiovascular disease, and toxic injury.[18] Among the cardiovascular and respiratory threats connected to wildfire smoke exposure and specific at-risk populations, further investigation has prompted a review of maternal health, and pregnancy studies have also proven there are adverse neonatal outcomes when prenatal exposure occurs. Particulate matter of 2.5 or less micrometers in diameter (PM2.5) is especially dangerous due to the fine nature of the pollutant that is easily inhaled, can enter the bloodstream, and lead to systemic adverse health outcomes.[19] Wildfire-generated particulate matter has been shown to negatively impact vulnerable populations more than the usual contaminants typically affecting the region.[20] The size of PM2.5 and its constituents allow the uptake and infiltration of the respiratory barrier and circulatory system.[21] Therefore, it could lead to compromise of the maternal–fetal exchange unit. Regional population studies have also explored different air pollutants such as PM2.5, PM10, and O_3 and their relationship to the development of various congenital malformations.[22] A large ecologic analysis conducted in China demonstrated increased risk ratios for birth defects when exposure to NO_2 and PM10 occurred within the first 3 months of pregnancy.[23] PM10 exposure—another small, easily inhaled pollutant—was linked to an increased risk of CHDs with specific impact during the first and second months of pregnancy. NO_2 was also studied as a contaminant of non-wildfire related ambient air and was linked to increased risk of several birth defects. Various mechanisms for this damage have been proposed but no consensus reached.

Wildfire exposure has been widely linked to adverse outcomes in pregnancy, fetal and neonatal development that could be mediated through the common pathway of inflammatory disruption of normal placentation. Particulate matter derived from wildfire smoke and its derivatives may acutely trigger a maternal inflammatory response with multiple downstream effects. This particulate matter can also accumulate within the placenta, compromising the placental barrier and function. Inflammation and oxidative stress can trigger endothelial dysfunction, leading to vasoconstriction and alteration of placental blood flow and maternal–fetal exchange.[19] Adequate placental perfusion is vital to fetal cardiac output and growth. This acute injury causing dysregulation of normal cell processes insults the tightly regulated uterine microenvironment that is necessary for successful implantation and cardiac development.[24] Early in gestation the process of implantation and cardiac development occur in tandem and are integral to successful placentation, linked through their common beta catenin signaling pathways. Many early embryologic pathways integral to neural, cardiac, and placental maturation share similar signaling cascades, which could explain why a singular environmental exposure could impact various tissues in the same embryo or have different penetrance dependent on the timing of exposure. The parallel development of cardiac and neural systems at overlapping timepoints within gestation underlies the data pointing to the protective role of folate supplementation on cardiac development. Folate supplementation throughout preconception and pregnancy has been widely recommended for the prevention of neural tube defects. Although further study is needed, it is plausible that folate supplementation could have clinical application in support of cardiac development and prevention of CHDs.

The strength of this study is that it is based on a large sample of over 2 million births. This allowed adjustments to be made for common risk factors of ASD, highlighting the distinct impacts of wildfires and the increased risk of ASDs. This study also relied on validated methods that have been previously published to further substantiate the findings. Additionally, the geographic risk of exposure in relation to these birth defects was able to be classified by the zip codes of pregnant individuals.

There are also limitations to this study that should be noted. The limitations of population-based data encompass data entry and coding errors. ICD-9 codes are generalizable and easily used for accurate data analysis; however, misclassification can occur when using these codes. Population-based data also neglect specific risk factors, like infection, alcohol, or substance use, which cannot be analyzed across a broad review. There is also the possibility that the recorded zip codes do not reflect the actual full-time residences of the pregnant patients in question. However, studies have shown that it is less likely for pregnant populations to change residence during pregnancy, making residential zip codes a reliable measure of full-term pregnancy exposure.[25]

Conclusion

This study demonstrates that climate change and the resulting wildfire exposures can have serious implications during pregnancy. Identifying the consequences of wildfire exposure can help clinicians, public health professionals, and policymakers better respond to future prenatal care needs of women and their fetuses as we face the oncoming environmental crisis.

Patient Consent

This was a population-based database; patient consent was not required. The study was approved by California State University – Fullerton Institutional Review Board (IRB# HSR-19-20-548).

Conflict of Interest

None declared.

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