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Fine particulate matter exposure and incident atopic dermatitis: a birth cohort study

Lih-Hwa Lin,[1] Chung-Chin Lee,[2] Meng-Min Hwang,[3] Chau-Ren Jung,[3] I-Hsiu Lai,[4] Wei-Ting Chen[5] and Bing-Fang Hwang[2,6]

[1]Division of Chinese Medicine, An Nan Hospital, China Medical University, Tainan, Taiwan
[2]Department of Occupational Safety and Health, College of Public Health, China Medical University, Taichung, Taiwan
[3]Department of Public Health, College of Public Health, China Medical University, Taichung, Taiwan
[4]Department of Pediatrics, An Nan Hospital, China Medical University, Tainan, Taiwan
[5]Department of Atmospheric Sciences, National Taiwan University, Taipei, Taiwan
[6]Department of Occupational Therapy, College of Medical and Health Science, Asia University, Taichung, Taiwan
Correspondence: Bing-Fang Hwang. Email: bfhwang@mail.cmu.edu.tw

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Abstract

Background The association between exposure to fine particulate matter (PM2.5) from conception to 1 year after birth and the later development of atopic dermatitis (AD) has not been completely elucidated.

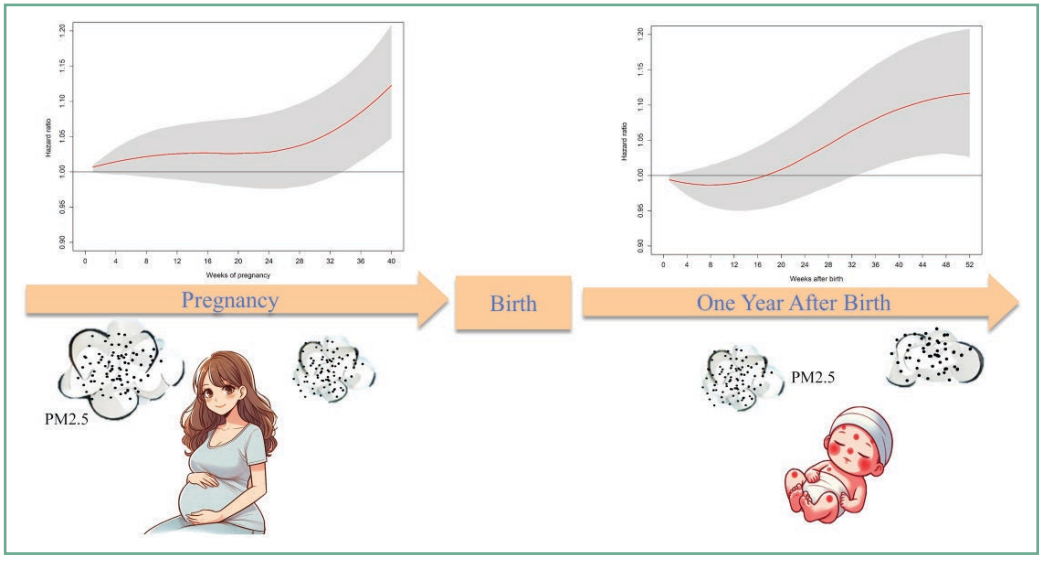
Objectives To investigate the effects of PM2.5 exposure during pregnancy and infancy on the later development of AD, and to explore vulnerable time periods to identify biologic pathways that may result in AD after exposure to PM2.5.

Methods We conducted a birth cohort study comprising 564 869 term births born between 2004 and 2013. The infants were followed-up until 5 years after birth. A satellite-based model was used to calculate PM2.5 exposure for each child. A Cox proportional hazards model combined with a distributed lag nonlinear model was created to examine the associations of AD with PM2.5, as well as the dose–response relationship.

Results The birth cohort comprised 76 944 infants diagnosed with AD. Increased cumulative exposure to PM2.5 from 34 weeks' gestation until birth, as well as from 33 weeks after birth, was significantly associated with a higher incidence of AD. With regard to the dose–response relationship, exposure to > 65 μg m^{-3} PM2.5 sharply increased the risk of AD.

Conclusions Prenatal and postnatal exposure to PM2.5 was related to later development of AD. The sensitive time periods may be late gestation and early life after birth.

Graphical Abstract



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Lay summary

Atopic dermatitis ('AD' for short) is a common skin condition. AD can be affected by a person's exposure to air pollution. However, the relationship between a child's exposure to a type of air pollution called 'fine particulate matter' ('PM2.5' for short) before they are born up to when they are 1 year old is still not completely clear. PM2.5 includes tiny particles of solids or liquids that can be breathed in through the lungs and enter the blood stream, causing health problems. Some examples include dust, ash, exhaust fumes and soot.

This study looked at the effects of exposure to PM2.5 during pregnancy and infancy to see if there was a relationship to AD. We also wanted to find out whether there is a particular window of time that might be critical for the development of AD after exposure to PM2.5. We studied data from 564,869 babies born in Taiwan from 2004 to 2013 and calculated each child's exposure to PM2.5. Using statistical techniques, we looked for an association between PM2.5 exposure and the development of AD. Out of a group of 76,944 children diagnosed with AD, we found that they had been exposed to higher levels of PM2.5 from 34 weeks of pregnancy until they were born. We also found that there were more cases of AD at 33 weeks after birth.

Our findings suggest that exposure to PM2.5 during pregnancy and infancy could be associated with the development of AD later in life. The most critical times may be mid-to-late pregnancy and the first few weeks after birth.

What is already known about this topic?

- Most previous studies of the association between exposure to fine particulate matter (PM2.5) and AD have been cross-sectional or case–control studies focusing on the prevalence of AD rather than its incidence, making it difficult to establish a causal relationship between PM2.5 exposure and AD in children.
- Only a few birth cohort studies have investigated the association of maternal exposure to PM2.5 during pregnancy and infancy and the subsequent development of AD.

What does this study add?

- It remains unclear whether there is a particularly critical time period during pregnancy and infancy where exposure to PM2.5 exposure may lead to the development of AD in the child.
- However, exposure to PM2.5 during critical periods, such as late gestation and early life after birth, is linked to adverse developmental outcomes, including the potential development of AD in children.
- Pregnant individuals and infants should minimize outdoor activities when PM2.5 levels exceed 65 μg m^{-3}, to reduce the risk of exposure during critical developmental windows.

Atopic dermatitis (AD), also known as atopic eczema, is a chronic disease characterized by inflammation, redness, irritation and – potentially – epidermal hyperplasia and thickening.[1] It is usually diagnosed during infancy or early childhood.[2] According to the Global Burden of Disease Study 2017, the global prevalence of AD is 2689.85 per 100 000 people and is highest in children aged 1–5 years.[2] The prevalence of AD has increased in recent years.[3] Genetic and environmental factors may play an important role in the development of AD. As genetic factors are less likely to change over time, the increasing incidence of AD is mainly due to environmental factors. There is an urgent need to implement AD prevention strategies by identifying modifiable factors and the most susceptible time windows for AD.

Genetic susceptibility, family history and environmental impacts are significant risk factors for AD.[1,4,5] The pathological mechanisms involved include dysfunction of the epidermal barrier (including abnormalities in epidermal structural proteins and lipids), cutaneous inflammation primarily driven by T helper (Th)2 and/or Th22 cells, and neuroimmune interactions triggered by pruritogens and sensory nerves of the skin.[1,4,6,7] Th cells are involved in the primary immune mechanisms of AD. Th2 cells elicit allergic responses and induce inflammation by releasing chemicals. Excess Th22 cells can compromise the epidermal barrier, allowing irritants and allergens to penetrate more easily. Overactive Th1 and Th17 responses can exacerbate chronic inflammation and the clinical manifestations of AD.[1,4,6,8]

Short- and long-term exposure to air pollutants exaggerates cutaneous oxidative stress, reduces epidermal barrier integrity, increases transepidermal water loss, disturbs inflammatory signalling cascades, alters stratum corneum pH and interferes with the skin's microbiota,[9,10] leading to erythema, oedema, scratch marks, oozing and crusting, and intensification of the itch–scratch cycle.[1,9,10] Various heavy metals present in PM2.5, such as silicon, lead, manganese, chromium, cadmium and arsenic, can cause cytotoxicity. The apoptosis pathway can be induced, along with a decrease in the level of catalase mRNA, highlighting the harmful effects of oxidative stress provoked *in vitro* by PM2.5.[7,11] Common allergy triggers in children with AD include metal and heavy metal compounds such as nickel sulfate and potassium dichromate.[12]

Most previous studies have been cross-sectional or case–control studies reporting on prevalence rather than incidence, where elucidating a causal relationship between PM2.5 and AD in children is difficult. Only a few birth cohort studies have reported that maternal exposure to higher amounts of PM2.5 (> 53.0 μg m^{-3}) during pregnancy is not significantly associated with the development of AD in the

infant in the first 12 months after birth.[13] However, the association between maternal exposure to PM2.5 and the later development of AD in their child, and whether there is a sensitive time window for exposure during pregnancy and infancy, have not been completely elucidated.

Using data from a large birth cohort, we applied a high temporal and spatial resolution satellite-based model to investigate the association between weekly average exposure to PM2.5 during pregnancy and infancy and AD, and to determine the sensitive time windows for developing AD. The study was designed to establish an appropriate temporal relationship between PM2.5 exposure and AD, and to clarify the possible mechanisms behind the relationship.

Materials and methods

Study area

Infants born in central and southern Taiwan were eligible for inclusion in the study (Figure S1; see Supporting Information). Primary sources of emissions comprised large coal-fired power plants, steel plants, industrial and scientific parks, incinerators and traffic.

Data sources

We conducted a population-based birth cohort study using data from the Taiwan Maternal and Child Health Database (TMCHD).[14] In the period 2004–13, the TMCHD encompassed nationwide databases, including the Taiwan Birth Registry (2004–13), the National Registration of Death (2004–18) and the National Health Insurance Research Database (NHIRD) for outpatient and inpatient visits (2003–18).[14] The NHIRD covers 99.9% of the Taiwanese population. Information on newborn babies (e.g. sex, birth date, birthweight, gestational age, single or multiple births, birth order and Apgar score), the pregnant individual (e.g. pregnancy complications, age at delivery and residential address) and history of visits to the doctor [e.g. outpatient visits, hospital admissions, prescriptions and diagnosis of disease based on the International Classification of Diseases, Ninth and tenth Revisions, Clinical Modification (ICD-9-CM) and (ICD-10-CM)] for the pregnant individuals and the infants was available from the TMCHD.

Study population

Between 1 January 2004 and 31 December 2013, the TMCHD held data on 630 660 live births in central Taiwan; the infants were followed from conception until the end of December 2018. All children were followed-up to the age of 5 years. We excluded children from subsequent analyses if they were part of a multiple birth (n=18 219), were stillborn (n=21), had congenital anomalies (n=3130) or were < 37 weeks' gestational age (n=44 421). The final birth cohort included 564 869 children (Figure 1). Children lost to follow-up (n=1508) were considered to have had health insurance withdrawn or to have died during the study period. The response rate was 99.7%.

Outcome of interest

A child was classified as having AD if they had received at least two consecutive AD diagnosis codes (ICD-9-CM:

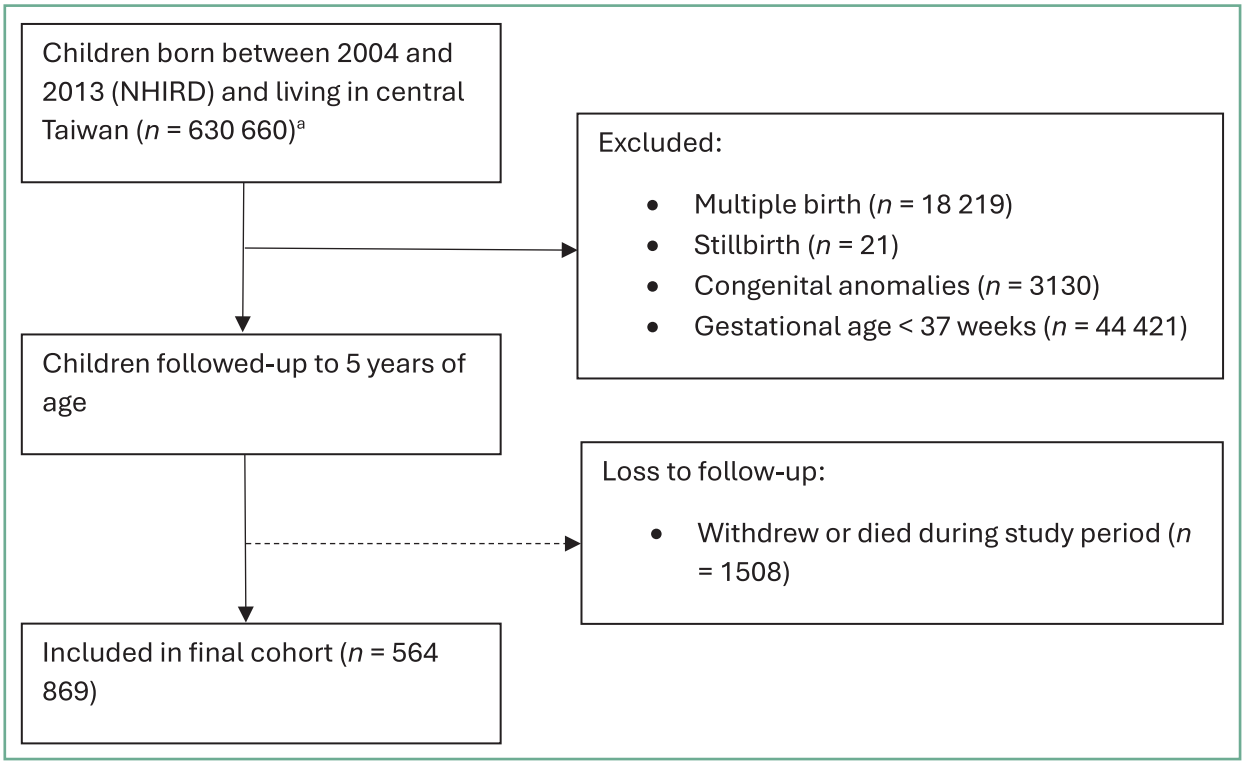

Figure 1 Flowchart of the birth cohort study. NHIRD, National Health Insurance Research Database. [a]Taichung City, Changhua County, Nantou County, Yunlin County, Chiayi County and Tainan County.

691.8; ICD-10: L20.89, L20.9) assigned by physicians, most often dermatologists, between their birth and the end of December 2018, at either outpatient or inpatient medical visits. Incident AD was identified as a child who received their first diagnosis of AD. Diagnosing AD involves identifying key features such as persistent itching and eczematous lesions, which follow a pattern of flare-ups and remission. Lesions typically appear on the face, neck and extensor surfaces. Supporting features include early onset, a family history of atopic diseases, elevated IgE levels and dry skin. Additional signs like facial pallor, white dermatographism and darkening around the eyes can further support the diagnosis. It is essential to rule out other conditions like seborrhoeic dermatitis or psoriasis, to ensure an accurate diagnosis. The diagnostic criteria for AD followed the 2014 American Academy of Dermatology Association clinical guidelines.[15]

Exposure assessments

A 1-km spatial satellite-based model that integrated aerosol optical depth (AOD), meteorological parameters and land-use data was used to calculate ground-level PM2.5 concentrations in Taiwan. The details of the model have been reported previously.[16] In the 10-fold cross-validation, the coefficient of determination was 0.78 and the root mean squared error was 8.1 $\mu g\ m^{-3}$.

Pre- and postnatal exposure to PM2.5 in children was determined from the satellite-based model, corresponding to their residential address at birth. The weekly average exposure to PM2.5 was calculated on the basis of the daily average for each child.

Covariates

Covariates included sex assigned at birth (male or female); birthweight (< 2500 g, 2500–4000 g or ≥ 4000 g); maternal household income derived from the amount of insurance paid (divided into four quartiles: $\leq Q_1$, Q_1–Q_2, Q_2–Q_3, $> Q_3$); maternal age (> 35 years, 35–20 years and ≤ 20 years); maternal anaemia; maternal heart disease; maternal lung disease; maternal chronic hypertension; maternal kidney disease; gestational hypertension; gestational diabetes mellitus and pre-eclampsia; smoking, prescribed medication intake or alcohol consumption during pregnancy (yes or no); and maternal atopy (defined as a diagnosis of asthma, allergic rhinitis or AD).

Statistical analyses

We first fitted Cox proportional hazard models to evaluate the association of PM2.5 exposure in the prenatal and postnatal periods with the later development of AD. Event time was considered from conception until 5 years after birth for each child. A child was treated as a censor if they did not develop AD by the end of follow-up or if they were withdrawn. We further introduced the time transform function (*tt* function) of *coxph* (*survival* package for R; R Foundation for Statistical Computing, Vienna, Austria), considering that PM2.5 may change over time.[17] If a covariate was associated with AD and was also associated with PM2.5 exposure, but was not a mediator of PM2.5, the covariate was considered to be a confounding factor and included in the final model. In the Cox proportional hazards models, we adjusted for sex, birth season, maternal household income, birthweight, maternal age, heart disease, atopy and smoking/alcohol consumption or prescribed medication taken during pregnancy.[18] The results are presented as hazard ratios (HRs) with 95% confidence intervals (CIs).

To determine sensitive time windows, we further estimated the cumulative HR using a distributed lag nonlinear model (DLNM) to explore the cumulative effects of pre- and postnatal weekly average exposure to PM2.5 on the development of AD separately.[19] The Akaike information criterion (AIC) was used to determine the type of spline and degrees of freedom for the exposure–response relationship, as well as lag effect.[20] Both exposure–response and lag–response relationships were fitted using a b-spline with six degrees of freedom and a natural cubic spline with three degrees of freedom, with the centring value set at 30 $\mu g\ m^{-3}$, representing the median of the PM2.5 distribution based on the minimum AIC. All statistical analyses were conducted using R version 3.4.3 (*dlnm* and *survival* packages).

Results

In our birth cohort from southern and central Taiwan, there were 76 944 cases of AD in 564 869 children (14%). Children with AD were more likely to be of male sex, have a higher maternal household income and be born to individuals with heart disease and atopy (Table 1).

The mean (SD) weekly PM2.5 concentrations in the pre- and postnatal periods were 34.24 (11.71) $\mu g\ m^{-3}$ (range 5.13–86.46) and 33.99 (11.76) $\mu g\ m^{-3}$ (range 4.01–86.46). The statistical models used demonstrated that increases of 10 $\mu g\ m^{-3}$ in PM2.5 during pre- and postnatal periods were positively associated with incident AD [adjusted HR (aHR) 1.07 (95% CI 1.06–1.08) and aHR 1.09 (95% CI 1.07–1.10), respectively] (Table 2). Cumulative exposure to PM2.5 during pregnancy and infancy (DLNM) is shown in Figures 2 and 3, respectively. We found that the cumulative HR, starting from gestational week 34 and continuing until birth (Figure 2), as well as from 33 weeks after birth, was positively associated with an increased risk of AD (Figure 3). The effects of exposure to PM2.5 from conception to 52 weeks after birth on incident AD (DLNM) are shown in Figure 4. In the exposure–response relationship, the HRs of AD increased sharply for PM2.5 exposure over 65 $\mu g\ m^{-3}$ (Figure 5).

Discussion

Our birth cohort study showed that increased exposure to PM2.5 during particular pre- and postnatal periods was positively associated with an increased incidence of AD. The susceptibility time windows start from gestational week 34 and continue until birth, as well as from 33 weeks after birth. Our results also revealed that the exposure of pregnant individuals to PM2.5 above 65 $\mu g\ m^{-3}$ might dramatically increase the effects of later development of AD in their children (Figure 3).

The biologic mechanisms by which pre- and postnatal exposure to PM2.5 may affect the incidence of AD potentially involve epidermal barrier dysfunction, immune

Table 1 Characteristics of the birth cohort from central Taiwan (2004–13)

Characteristic	Total (n=564 869)	AD (n=76 944)	PY (n=2 953 744)	Incidence density[a,b]	HR (95% CI)
Sex					
Female	272 406	35 067	1 435 133	2.44	Ref.
Male	292 463	41 877	1 518 611	2.76	1.13 (1.11–1.14)[c]
Birth season					
Spring	134 091	17 745	704 046	2.52	Ref.
Summer	139 204	19 034	727 684	2.62	1.04 (1.02–1.06)[c]
Autumn	155 983	21 658	812 938	2.66	1.06 (1.04–1.08)[c]
Winter	135 591	18 507	709 075	2.61	1.04 (1.02–1.06)[c]
Maternal household income (New Taiwan Dollar)					
≤ 15 840	101 104	11 428	536 473	2.13	Ref.
15 840 to ≤ 19 200	144 475	19 404	757 063	2.56	1.20 (1.17–1.23)[c]
19 200 to ≤ 27 600	161 481	21 503	845 008	2.54	1.19 (1.16–1.22)[c]
> 27 600	157 809	24 609	814 199	3.02	1.41 (1.38–1.44)[c]
Maternal age (years)					
≤ 20	12 335	1220	66 072	1.85	0.71 (0.67–0.75)[c]
20–35	484 714	66 347	2 533 738	2.62	Ref.
> 35	67 820	9377	353 933	2.65	1.01 (0.99–1.03)
Birthweight (g)					
< 2500	17 468	2202	91 337	2.41	0.92 (0.89–0.96)[c]
2500–4000	536 820	73 289	2 807 125	2.61	Ref.
≥ 4000	10 581	1453	55 281	2.63	1.01 (0.96–1.06)
Maternal anaemia					
Yes	3427	465	17 959	2.59	0.99 (0.91–1.09)
No	561 442	76 479	2 935 785	2.61	Ref.
Maternal heart disease					
Yes	492	82	2518	3.26	1.25 (1.00–1.55)[d]
No	564 377	76 862	2 951 226	2.60	Ref.
Maternal lung disease					
Yes	75	10	395	2.53	1.00 (0.54–1.84)
No	564 794	76 934	2 953 348	2.61	Ref.
Maternal chronic diabetes					
Yes	543	79	2788	2.83	1.09 (0.88–1.36)
No	564 794	76 865	2 950 955	2.60	Ref.
Gestational diabetes					
Yes	2588	362	13 300	2.72	1.05 (0.94–1.16)
No	562 311	76 582	2 940 444	2.60	Ref.
Maternal chronic hypertension					
Yes	457	50	2,418	2.07	0.80 (0.60–1.05)
No	564 412	76 894	2 951 326	2.61	Ref.
Gestational hypertension					
Yes	2847	410	14 777	2.77	1.07 (0.97–1.18)
No	562 022	76 534	2 938 967	2.60	Ref.
Pre-eclampsia					
Yes	1221	165	6375	2.59	0.99 (0.85–1.16)
No	563 648	76 779	2 947 369	2.61	Ref.
Maternal kidney disease					
Yes	87	13	448	2.90	1.11 (0.65–1.92)
No	564 575	76 931	2 953 295	2.60	Ref.
Maternal smoking, prescribed medication intake and alcohol consumption					
Yes	395	35	2129	1.64	0.64 (0.46–0.88)[c]
No	564 474	76 909	2 951 615	2.61	Ref.
Maternal atopy					
Yes	121 743	18 757	627 600	2.99	1.19 (1.17–1.21)[c]
No	443 126	58 187	2 326 144	2.50	Ref.

CI, confidence interval; HR, hazard ratio; PY, person-years. [a]Incidence density per 100 PY; [b]incidence density for the total cohort was 2.61; [c]$P<0.01$; [d]$P<0.05$.

dysregulation, oxidative stress, enhanced sensitization to allergens and inflammatory responses.[21] During pregnancy, PM2.5 can cross the placental barrier and indirectly affect the fetus, causing oxidative stress and inflammation. This process can trigger immune dysregulation, making the fetus more susceptible to irritants and allergens, and increasing the risk of developing AD.[22] During infancy, continued exposure to PM2.5 after birth can directly damage the skin barrier through increased oxidative stress, water loss, physicochemical injury and changes in the microbiota, leading to an increased risk of developing AD in childhood.[23]

Table 2 Atopic dermatitis (AD) and exposure to fine particulate matter in prenatal and postnatal periods

	Crude model, HR (95% CI)	Adjusted model, HR (95% CI)
Prenatal period exposure	1.07 (1.05–1.08)[a]	1.07 (1.06–1.08)[a]
Postnatal period exposure	1.09 (1.07–1.10)[a]	1.09 (1.07–1.10)[a]
Total exposure	1.09 (1.08–1.10)[a]	1.09 (1.08–1.10)[a]

The model was adjusted for sex, birth season, maternal household income, birthweight, maternal age, maternal smoking, prescribed medication intake and alcohol consumption, and maternal atopy (including asthma, allergic rhinitis and AD). CI, confidence interval; HR, hazard ratio. [a]$P<0.01$.

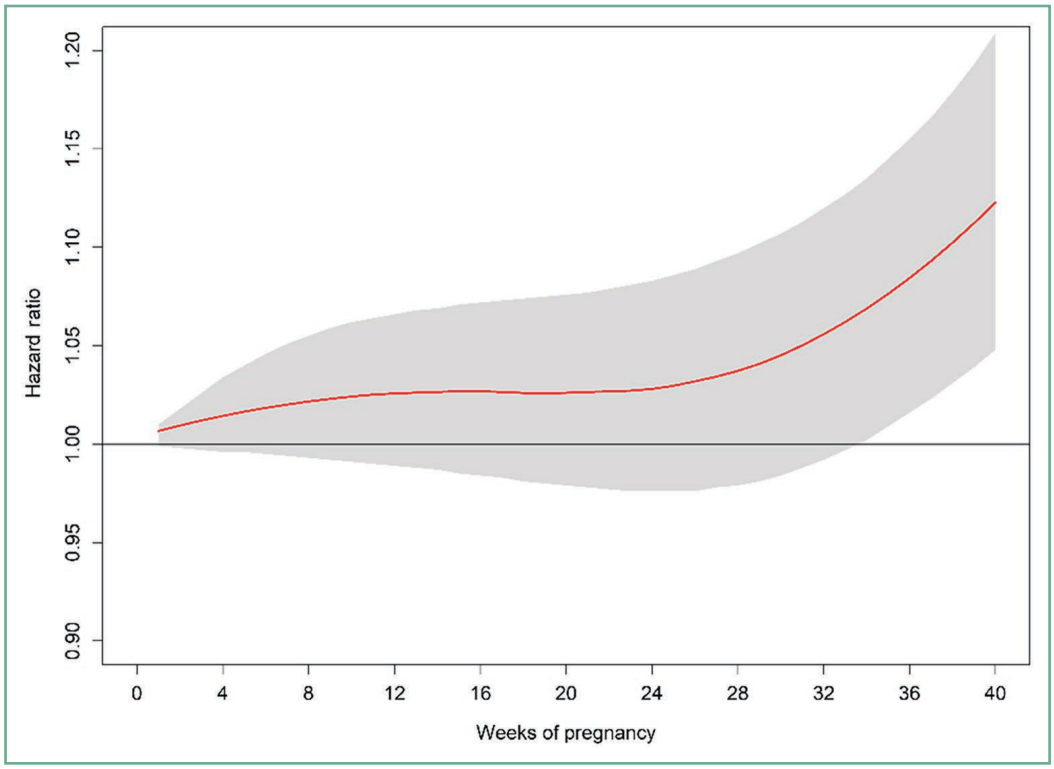

Figure 2 Cumulative exposure to fine particulate matter (PM2.5) and incident atopic dermatitis (AD) during pregnancy. Cumulative hazard ratio (red line) and 95% confidence interval (grey area) of AD were estimated using a distributed lag nonlinear model. The model was adjusted for sex, birth season, maternal household income, birthweight, maternal age, maternal smoking, prescribed medication intake and alcohol consumption, and maternal atopy (including asthma, allergic rhinitis and AD). All children were followed-up until the age of 5 years.

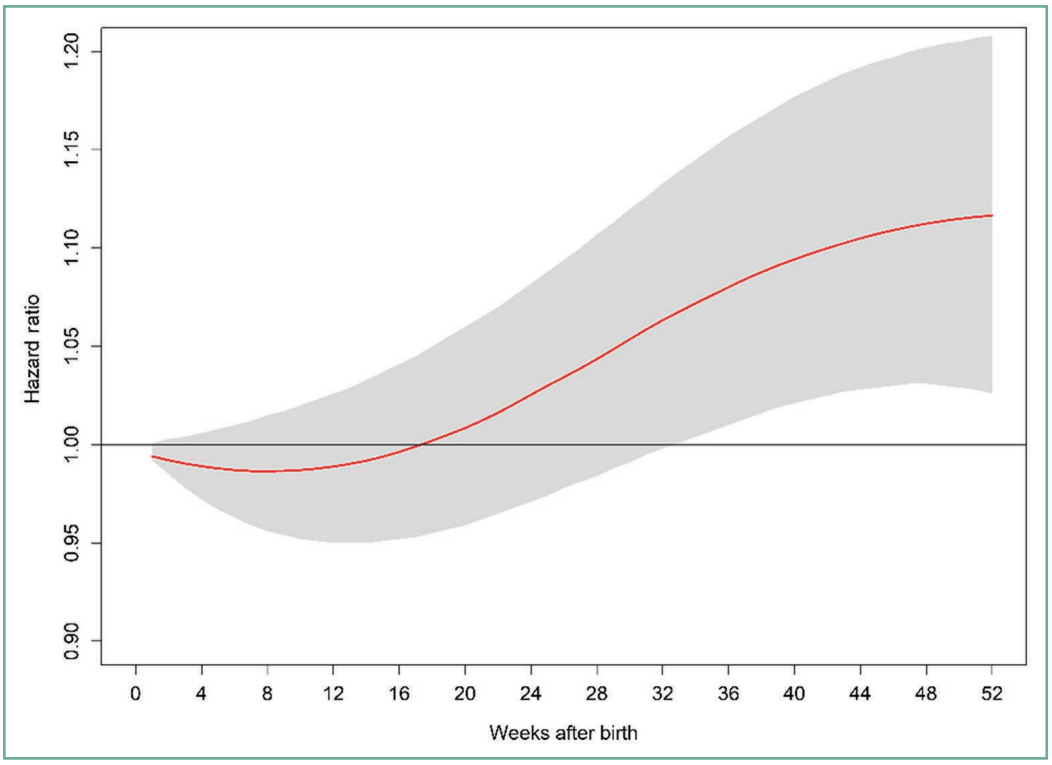

Figure 3 Cumulative exposure to fine particulate matter (PM2.5) and incident atopic dermatitis (AD) during infancy. Cumulative hazard ratio (red line) and 95% confidence interval (grey area) of AD were estimated using a distributed lag nonlinear model. The model was adjusted for sex, birth season, maternal household income, birthweight, maternal age, maternal smoking, prescribed medication intake and alcohol consumption, and maternal atopy (including asthma, allergic rhinitis and AD). All children were followed-up until the age of 5 years.

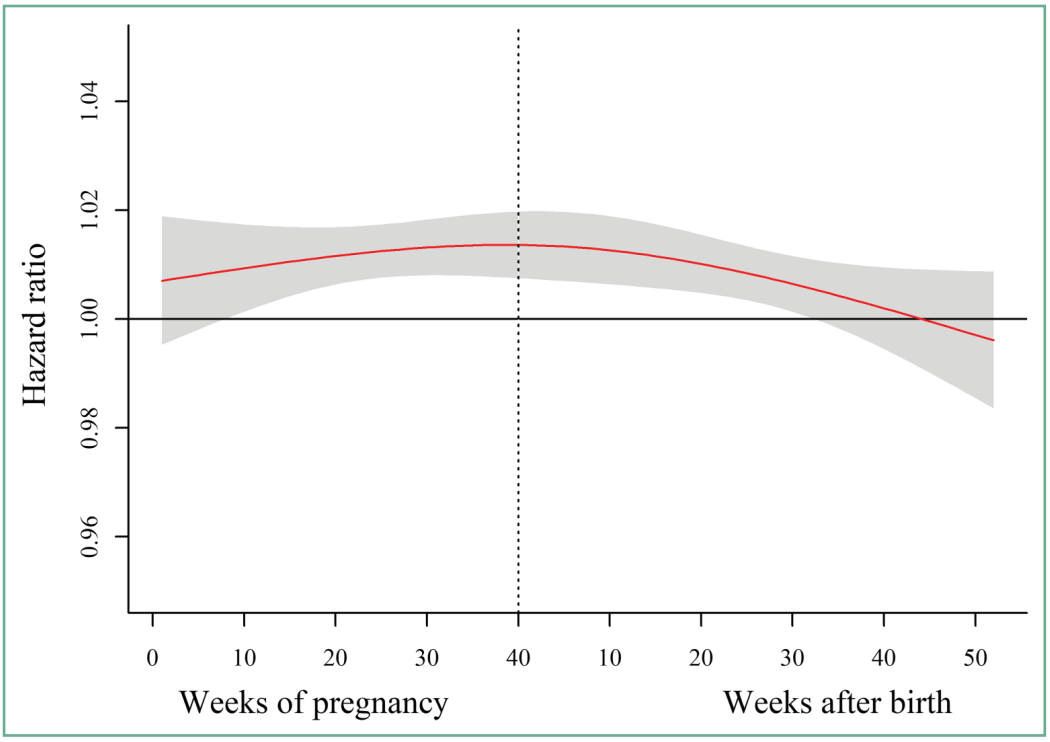

Figure 4 The lag–response relationship of fine particulate matter (PM2.5) with atopic dermatitis (AD) from conception to the first year after birth. Hazard ratio (red line) and 95% confidence interval (grey area) of AD were estimated using a distributed lag nonlinear model. The model was adjusted for sex, birth season, maternal household income, birthweight, maternal age, maternal smoking, prescribed medication intake and alcohol consumption, and maternal atopy (including asthma, allergic rhinitis and AD). All children were followed-up until the age of 5 years.

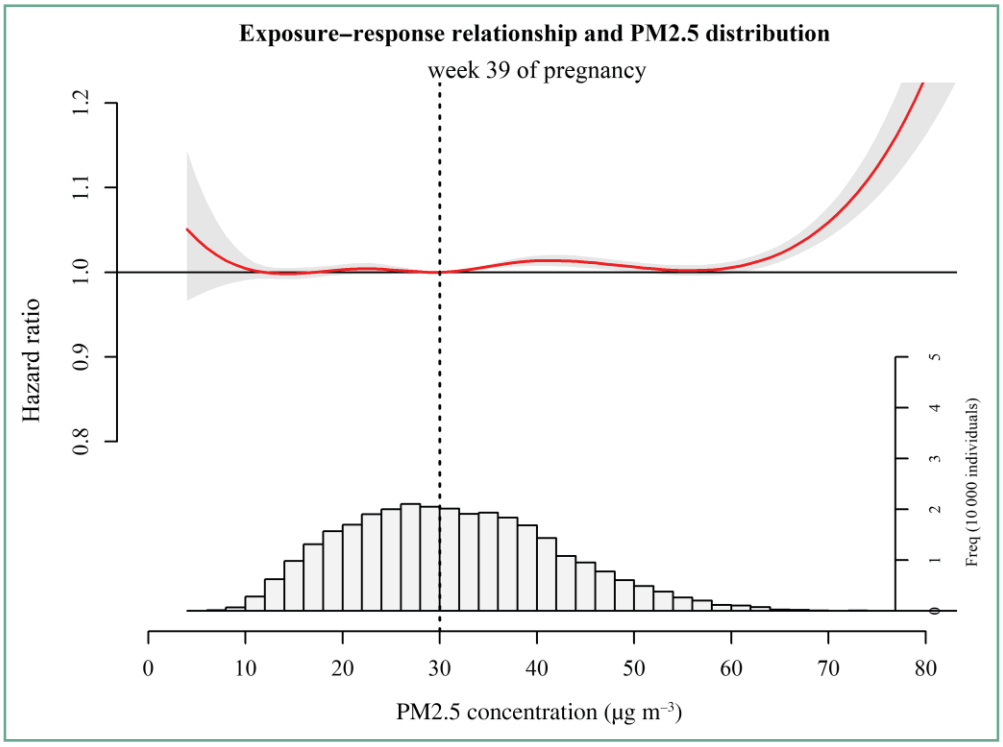

Figure 5 The dose–response relationship between atopic dermatitis (AD) and fine particulate matter (PM2.5) concentrations (in gestational week 39). The results are shown as hazard ratio (red line), 95% confidence interval (grey area) and exposure to PM2.5 concentration (histogram). The model was adjusted for sex, birth season, maternal household income, birthweight, maternal age, maternal smoking, prescribed medication intake and alcohol consumption, and maternal atopy (including asthma, allergic rhinitis and AD).

Previous epidemiological studies have predominantly been cross-sectional or case–control in nature, focusing on prevalent rather than incident cases. This makes it challenging to establish a causal relationship between PM2.5 exposure and AD in children. Only a limited number of birth cohort studies have explored the link between maternal exposure to PM2.5 during pregnancy and infancy, and the subsequent development of AD. One such study, involving 469 full-term infants in New York City and Krakow, found that the mean (SD) PM2.5 concentration pregnant individuals were exposed to in the second trimester was 44.4 (46.5) $\mu g\ m^{-3}$. The study reported that maternal exposure to higher PM2.5 levels ($> 53.0\ \mu g\ m^{-3}$) during pregnancy was not significantly associated with the development of AD in the first year of life.[13] The inconsistent results across studies can be attributed to several factors, including the varying diagnostic criteria for AD used in different countries, differences in age groups studied, and the diverse components and concentrations of PM2.5.

This study had two strengths. Firstly, our large birth cohort, comprised of 630 660 children, had sufficient power to allow us to explore the effects of exposure to PM2.5 during pregnancy and infancy on incident AD. Secondly, we applied a valid satellite-based model to integrate AOD, meteorological factors and land-use data with fine temporal and spatial resolution to predicted ground PM2.5 in Taiwan.[16] This model can identify sensitive time windows from conception up to the first year after birth.

Our study had several limitations. Air pollution is caused by complex mixtures of solid particles, liquid droplets and gaseous molecules. The toxicity of PM2.5 from different compositions, sources and sizes varies. However, we did not investigate the associations between exposure to specific PM2.5 and AD owing to a lack of information on the composition and source, and on ultrafine particles. Secondly, although we were able to adjust for important confounders, including sex, birth season, maternal household income, birthweight, maternal age, maternal heart disease, maternal smoking, prescribed medication intake, alcohol consumption and maternal atopy, we could not rule out potential bias from unmeasured or unknown confounding factors, such as maternal food allergy, increased total serum IgE, atopic stigmata, atopic comorbidities of AD (food allergy, asthma, allergic rhinitis and allergic conjunctivitis), climate change, allergens, time spent indoors and outdoors, and genetic and nutritional factors.[24] Thirdly, the duration of follow-up was only 5 years. It is likely that the first diagnosis of AD was lost within a short period of time.

In conclusion, our study indicates that pre- and postnatal exposure to PM2.5 is associated with later development of AD in children. The critical time periods may be during late gestation and early life after birth. Our findings suggest that, during these particular time periods, pregnant individuals and children should limit their outdoor activity when the ambient PM2.5 in the air is $> 65\ \mu g\ m^{-3}$.

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Conflicts of interest

The authors declare no conflicts of interest.

Data availability

The data underlying this article cannot be shared publicly due to data regulations of the Health and Welfare Data Science Center, Taiwan Ministry of Health and Welfare.

Ethics statement

The study was ratified by the Institutional Review Board of the China Medical University Hospital (CRREC-110-020) and followed the principles of the Declaration of Helsinki.

Patient consent

Not applicable.

Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website.

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