Cite as: Bobrowska-Korzeniowska M, Jerzyńska J, Podlecka D, Polańska K, Gwardys M, Paciorek M, Stelmach W: Impact of environmental pollutants – particulate matter $PM_{2.5}$, PM_{10}, ozone and nitrogen dioxide on asthma and allergy in preschoolers. Pediatr Med Rodz 2023; 19 (3): 195–206. DOI: 10.15557/PiMR.2023.0035.

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Received: 21.07.2023
Accepted: 29.08.2023
Published: 05.12.2023

Impact of environmental pollutants – particulate matter $PM_{2.5}$, PM_{10}, ozone and nitrogen dioxide on asthma and allergy in preschoolers

Wpływ zanieczyszczeń środowiska – pyłu zawieszonego $PM_{2,5}$, PM_{10}, ozonu i dwutlenku azotu na rozwój astmy i alergii u dzieci w wieku przedszkolnym

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https://doi.org/10.15557/PiMR.2023.0035

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Abstract

Introduction and objective: This study evaluated the impact of day-to-day air pollutants, such as particulate matter $PM_{2.5}$, PM_{10}, nitrogen dioxide (NO_2), and ozone (O_3) on the development on asthma and allergy in preschool children. **Materials and methods:** A total of 284 5-year-old children participated in the study. The questionnaires were completed by the caregivers. For each child, the visit involved skin prick testing and the distribution of backpacks containing GilAir Plus Basic personal aspirators for 24-hour measurement of $PM_{2.5}$ and PM_{10}. The dust samples were analysed in the laboratory. Air pollutants in the place of residence have been assessed for 5 years using the CALMET/CALPUFF modelling system. **Results:** Significantly higher levels of $PM_{2.5}$ (as measured by personal aspirators), 2015 $PM_{2.5}$ [μg/m^3] and 2015 PM_{10} [μg/m^3] were observed in the allergy group in comparison to the no-allergy group. 2018 and 2019 ozone levels differentiated the analysed groups. In multivariate analysis, passive smoking was associated with a 3-fold increase in the risk of allergy (odds ratio – OR = 2.76, 95% confidence interval – 95% CI [1.21;6.53], $p = 0.017$). Among the analysed pollutants, 2017 PM_{10} [μg/m^3] increased the odds of allergy with the highest strength. An increase in its concentration by 1 μg/m^3 resulted in 14% higher odds of allergy (OR = 1.14, 95% CI [1.02;1.28], $p = 0.021$). Among the assessed pollutants, 2017 ozone increased the odds of asthma, with an increase in its concentration by 1 unit resulting in 41% higher odds (OR = 1.41, 95% CI [1.02;2.04], $p = 0.049$). **Conclusions:** Exposure to $PM_{2.5}$, PM_{10} and ozone seem to increase the overall risk of allergy and asthma. Passive smoking showed the strongest correlation with an increase in the risk of allergy.

Keywords: children, asthma, environmental exposure, allergic diseases, air pollutants

Streszczenie

Wprowadzenie i cel: Badanie miało na celu ocenę wpływu zanieczyszczeń powietrza $PM_{2,5}$, PM_{10}, dwutlenku azotu (NO_2) oraz ozonu (O_3) na rozwój astmy i alergii u dzieci. **Materiał i metody:** W badaniu wzięło udział 284 dzieci w wieku 5 lat. Kwestionariusze zostały wypełnione przez opiekunów. W przypadku każdego dziecka wizyta obejmowała wykonanie punktowych testów skórnych oraz wydanie plecaka z aspiratorem GilAir Plus Basic do 24-godzinnego pomiaru zanieczyszczenia powietrza ($PM_{2,5}$ i PM_{10}). Próbki pyłu zebrane przez osobiste aspiratory przeanalizowano w laboratorium. Zanieczyszczenia powietrza w miejscu zamieszkania oceniano przez 5 lat, korzystając z systemu modelowania CALMET/CALPUFF. **Wyniki:** W grupie dzieci z alergią w porównaniu z dziećmi bez alergii zaobserwowano znacząco wyższe stężenia $PM_{2,5}$ (mierzone przez aspiratory osobiste), 2015 $PM_{2,5}$ [μg/m^3] i 2015 PM_{10} [μg/m^3]. Analizowane grupy różniły się pod względem stężeń ozonu 2018 i ozonu 2019. W analizie wieloczynnikowej bierne palenie tytoniu zwiększało ryzyko alergii trzykrotnie, iloraz szans (*odds ratio*, OR) wynosił 2,76, 95-procentowy przedział ufności (*95% confidence interval*, 95% CI): 1,21–6,53, $p = 0,017$. Spośród czynników zanieczyszczających ryzyko alergii zwiększał najsilniej 2017 PM_{10} [μg/m^3]. Dwukrotne zwiększenie stężenia PM_{10}

skutkowało 14-procentowym zwiększeniem ryzyka alergii, OR = 1,14, 95% CI: 1,02–1,28, p = 0,021. Ozon 2017 zwiększał prawdopodobieństwo astmy – zwiększenie jego stężenia o jedną jednostkę skutkowało o 41% wyższym ryzykiem, OR = 1,41, 95% CI: 1,02–2,04, p = 0,049. **Wnioski:** Obserwacje autorów sugerują, że zanieczyszczenia mogą wpływać na alergię i astmę dopiero po przekroczeniu dozwolonych stężeń. Narażenie na $PM_{2.5}$, PM_{10} i ozon wydaje się zwiększać ogólne ryzyko alergii i astmy. Palenie bierne miało najsilniejszy związek ze wzrostem ryzyka rozwoju alergii.

Słowa kluczowe: dzieci, astma, ekspozycja środowiskowa, choroby alergiczne, zanieczyszczenia powietrza

INTRODUCTION

Allergies have become a serious public health concern since their prevalence has increased over the last two decades, affecting 30–40% of the world population[1]. Asthma and other atopic diseases arise as a result of an interplay between individual genetic factors and environmental exposures during pregnancy and in the first years of life[2,3]. Air quality and other environmental factors are gaining importance in public health policies. Urban residents, especially children, are disproportionately exposed to different environmental factors, including air pollution, which trigger asthma symptoms[4,5]. Particulate matter ($PM_{2.5}$ and PM_{10}) produced during combustion, by traffic and industrial activities, is believed to be responsible for harmful health effects[6]. It can irritate the nasal and bronchial mucosa, causing the release of inflammatory mediators, which may aggravate the symptoms of atopic diseases.

Due to its high water solubility, sulfur dioxide (SO_2) severely damages the upper airways in contrast to nitrogen dioxide (NO_2) and ozone (O_3), which are less soluble and therefore penetrate deeper into the lungs[7].

According to the European Environment Agency (EEA), in spite of the efforts done towards meeting the air quality standards European Union urban population is exposed to air pollutant concentrations above World Health Organization (WHO) reference values in 2016–2018 is as follows: 74–78% to $PM_{2.5}$, 43–48% to PM_{10}, 96–99% to O_3, 4–7% to NO_2, 75–90% to benzo(a)pyrene (BaP), 19–31% to SO_2[8]. An overview of air quality in the 27 member countries of the EU and the United Kingdom showed that the rising O_3 levels became a major public health issue in the EU[9].

Conflicting results have been reported despite of the high number of investigations on the association between asthma and air pollution. Some studies in children showed that the exposure to NO_2 was positively associated with a higher prevalence of asthma, whereas exposure to PM and SO_2 was positively correlated with a higher incidence of wheeze[10]. Exposure to traffic-related air pollution were weakly associated with respiratory symptoms in an Italian birth cohort study[11]. In contrary, no evidence of an association between long-term exposure to PM_{10} and NO_2 and the prevalence of wheezing and asthma was found in the Manchester Asthma and Allergy Study (MAAS)[12].

The aim of the present study was to evaluate the impact of day-to-day air pollutants, such as particulate matter $PM_{2.5}$, PM_{10}, NO_2, and O_3 on the development of allergy and asthma in preschool children living in urban or rural areas since birth.

METHODS

The study group consisted of 284 5-year-old children attending kindergartens in urban and rural areas of the city of Łódź and the Łódź Voivodeship. The research team (medical doctor and assistant) arranged meetings with caregivers/parents after receiving approvals from headmasters of the twelve randomly chosen kindergartens. The aim and procedures of the study were explained. Written consent forms for children's participation in the study were collected and the questionnaires were completed by the parents/caregivers. For each child, this visit included skin prick testing (SPT), wherever it was possible (if they did not take anti-histamine drugs) and the distribution of backpacks containing GilAir Plus Basic personal aspirators for 24 h measurement of air pollution (12 h $PM_{2.5}$ and 12 h PM_{10}). The backpacks were collected the next day. The Aerosol Laboratory of the Nofer Institute of Occupational Medicine in Łódź was responsible for the analysis of the collected dust samples. The air pollutants in the place of residence have been monitored for 5 years, since birthday of the participants, using the CALMET/CALPUFF modelling system. The complete description of the methodological assumption has been published elsewhere[13]. The Ethics Committee of the Medical University of Lodz approved the study (decision No. RNN/390/17/KE). The participants had the right to withdraw their consent for further participation in the study at any time. They were informed that the obtained data was used only for research purposes.

Questionnaire

The questionnaire was addressed to the parents/caregivers of the child and included data on the child's health (e.g. doctor-diagnosed allergy and asthma, the severity and frequency of diseases), exposure to air pollution and socio-economic data. The diagnosis of atopic disease (food allergy, asthma, allergic rhinitis or atopic dermatitis)

DOI: 10.15557/PiMR.2023.0035

was made by the allergist; asthma was diagnosed based on Global Initiative for Asthma (GINA) guidelines[14]. The survey was developed on the basis of the International Study of Asthma and Allergies in Childhood (ISAAC) and the European Community Respiratory Health Survey (ECRHS)[15].

Skin prick testing

Skin prick testing was performed according to the guidelines of the European Academy of Allergy and Clinical Immunology[16,17] with the most common inhalant allergens (published elsewhere[13]).

Environmental assessment

The measurement of air pollution was done using GilAir Plus Basic (Clearwater, FL, USA) personal aspirators (published elsewhere[13]). The collected dust samples were analysed in the Aerosol Laboratory of the Noffer Institute of Occupational Medicine in Łódź. The filters were placed in containers with individual identifiers, and then weighed before and after collecting dust samples to determine the dust concentration which was calculated as the ratio of the mass of dust retained in the filter to the volume of the air passed through the filter. Prior to weighing, clean filters were placed in a silica gel desiccators and conditioned for at least 20 hours, and then weighed on the same weight.

The analysis of air quality for the years 2015–2019 (the period since the birthday year of subjects till the year of participation in the study) was performed using CALPUFF and CAMx models. In selected locations in the Łódź Voivodeship, mathematical modelling of spatial distribution of concentrations of the following substances: PM_{10} and $PM_{2.5}$, SO_2, NO_2, benzene, BaP and O_3 was performed.

The CALMET/CALPUFF modelling system was developed in Earth Tech, Inc. in California. It consists of two basic tools: a preprocessor preparing meteorological data (CALMET v 6.5.0) and a basic dispersion model (CALPUFF v 7.2.1). The software code written in Fortran is available at http://www.src.com/calpuff/download/download.htm. It is fully compilable, which allows for its customisation to a specific case.

The CAMx model was used to determine the O_3 concentration distribution. The CAMx model performed calculations in a three-dimensional grid. Meteorological parameters for modelling dispersion of pollutants in the Łódź region were provided by the numerical weather prediction system WRF (Weather Research and Forecasting). The input source for the model was UCAR (University Corporation for Atmospheric Research) and NCAR (National Center for Atmospheric Research – NCAR Research Data Archive – https://rda.ucar.edu/).

The emission information was delivered to the model in the form of a cadastre (grid) part of the CALMET/CALPUFF modelling system, responsible for preparing the original field information and meteorological data for the input of the CALPUFF model is the CALMET preprocessor. Meteorological calculations were performed in a regular grid (grid), including area with emissions. For the purpose of this study, detailed field information in appropriate resolutions were created. The terrain elevation information has been developed on the basis of SRTM-3 data, which is publicly available on the server: ftp://edcftp.cr.usgs.gov/pub/data/srtm/Eurasia/.

The emission bases for the years 2015–2019 used in the calculations in this paper were collected, supplemented and verified by Office of Pro-ecological Studies and Measurements Ekometria, among others, on the basis of data provided by the National Centre for Emissions Management (KOBIZE, Poland).

Statistical analysis

Categorical traits were described with absolute and percentage frequency. Numerical traits were described with mean and standard deviation or median and interquartile range, depending on the normality of distribution. Normality of distribution was verified with Shapiro–Wilk test as well as skewness and kurtosis. Variance homogeneity was checked with Levene's test. Groups were compared using Fisher's exact test, Pearson's chi-square test, *t*-Student or *t*-Welch independent test, as appropriate. Logistic regression analysis was executed in two steps. Univariate models were applied for each trait and pollution variable. Initial variables filtering for multivariate models was based on $p = 0.25$ cut-off (out of univariate models' outcomes)*. Correlation analysis for pollution variables was employed to support variable selection. Further, stepwise approach based on AIC (Akaike information criterion) indicator was applied to design final multivariate models. Multivariate outcomes were verified with Nagelkerke R^2 and Hosmer–Lemeshow goodness of fit (GOF) test. VIF (variance inflation factor) indicators were used to avoid multicollinearity. All statistical calculations assumed significance at $p < 0.05$. The analysis was run in R statistical software, version 4.1.2.

RESULTS

Study participants

Two hundred eighty-four children completed the study. All participants were reported being Caucasians

* Hosmer DW, Lemeshow S: Applied Logistic Regression. John Wiley & Sons, 2000.

 DOI: 10.15557/PiMR.2023.0035

with a mean age of 5 years (standard deviation, *SD* 2.8 months). Study participants included 51.8% females. Atopy was noticed in 48.6% of families. Allergy of any type was present in 42.3% of children, whereas about 23% of the subjects were diagnosed with asthma. Parental smoking and at least 1 pet were reported for 33.5% and 43.1% of children. Baseline characteristic of the study population, including comparisons between any allergy/no allergy and asthma/no asthma groups, is presented in Tab. 1.

Allergies

Any allergy was present in 42.3% of participants, with 23% of them diagnosed with asthma, 14.5% with allergic rhinitis, 12.4% with atopic dermatitis, and 10.6% with food allergy. Skin prick tests were positive in 31.7% of children, with 12.5% sensitised to house dust mites, 16% to grass pollens, 12.5% to trees, 3.1% to mugwort, 6.6% to moulds, 5.9% to cat dander and 1.0% to dog dander. The proportion of children exposed to household smoking was significantly higher in the allergy group (39.2% vs. 28.8% within no-allergy group), $p = 0.045$. A family history of atopy was observed significantly more frequently in the allergy group (57.1% vs. 42.0% in no-allergy group), $p = 0.013$. Groups of children with and without any allergy were significantly differentiated in terms of having other type of animal at home: a lower proportion was observed in the allergy group (5.8%, 7 patients vs. 12.3% in no-allergy group), $p = 0.017$ (Tab. 1).

Asthma

Asthma was diagnosed in 66 children (23%). Economic status significantly differentiated groups of asthmatic vs. non-asthmatic children ($p = 0.023$). No children whose economic status was low and only one child with very low economic status belonged to the asthma group, while their proportion in the no-asthma group was 3.7% (8 patients) and 11.6% (25 patients), respectively. Both average and high economic status were more common in the asthma group (average: 74.2% vs. 65.3%, high: 24.2% vs. 19.4%). Household pets were significantly less common in the asthma group (30.8% vs. 46.8% in no-asthma group), $p = 0.032$ (Tab. 1).

Air pollution level at measurement stations

In the analysed period exceeding of the limit level of average annual concentrations of particulate matter PM_{10} (40 μg/m³) and $PM_{2.5}$ occurred at measurement stations in Łódź and Łódź region. In each of the analysed years, the target concentrations of the annual mean BaP were exceeded at all stations where measurements were carried out. No exceedances of the annual mean concentrations of benzene, NO_2, SO_2 have been recorded at any of the measuring stations.

The results of 8-hour O_3 concentrations showed exceeded target level (120 μg/m³).

A comparison of air pollution level between the study groups (calculated using CALPUFF and CAMx models)

Allergy group

Significantly higher levels of $PM_{2.5}$ (measured by personal aspirators), 2015 $PM_{2.5}$ [μg/m³] and 2015 PM_{10} [μg/m³] were observed in the allergy group vs. the no-allergy group (mean or median difference, MD = 4.00, 95% confidence interval – 95% CI [0.00;8.00], $p = 0.019$; MD = 1.01, 95% CI [0.13;1.90], $p = 0.025$ and MD = 1.28, 95% CI [0.09;2.46], $p = 0.035$, respectively). The 2018 and 2019 O_3 levels differentiated the analysed groups (MD = 0.10, 95% CI [0.00;0.10], $p = 0.034$ and MD = 0.00, 95% CI [0.00;0.10], $p = 0.009$, respectively) (Tab. 2).

Asthma group

The 2018 PM_{10} [μg/m³] and 2015 $PM_{2.5}$ [μg/m³] levels differed depending on the asthma status. Their significantly higher levels were noted in the asthma group (MD = 1.13, 95% CI [0.46;1.80], $p = 0.001$ and MD = 1.24, 95% CI [0.20;2.28], $p = 0.019$, respectively). Furthermore, the distribution of 2017 NO_2 [μg/m³] was significantly different depending on the prevalence of asthma (MD = 0.00, 95% CI [0.00;0.00], $p = 0.003$). The 2017 to 2019 O_3 levels also differentiated the groups, with higher levels in asthmatic children (MD = 1.03, 95% CI [0.30;1.75], $p = 0.006$; MD = 0.10, 95% CI [0.00;0.10], $p = 0.017$ and MD = 0.10, 95% CI [0.00;0.20], $p = 0.006$, respectively) (Tab. 2).

Allergy group – univariate logistic regression

A family history of atopy would double the odds of allergy – odds ratio, OR = 1.84, 95% CI [1.14;2.99], $p = 0.012$. The levels of $PM_{2.5}$ contributed to the risk of allergy. An increase in the level of $PM_{2.5}$ measured with personal aspirators [μg/m³] by 1 μg/m³ would increase the odds of allergy by 1% (OR = 1.01, 95% CI [1.00;1.01], $p = 0.020$). A significant increase in the odds of allergy was observed for 2015 $PM_{2.5}$ [μg/m³] and 2015 PM_{10} [μg/m³]. An increase in their levels by 1 μg/m³ resulted in 7% and 5% increased odds of allergy, respectively (OR = 1.07, 95% CI [1.01;1.15], $p = 0.026$ and OR = 1.05, 95% CI [1.00;1.11], $p = 0.033$, respectively) (Tab. 3).

Allergy group – multivariate logistic regression

In multivariate analysis step, passive smoking (PS) increased the allergy odds 3 times (OR = 2.76, 95% CI

DOI: 10.15557/PiMR.2023.0035

Tab. 1. Study group characteristics and comparisons between allergy/no allergy group and asthma/no asthma group

Variable	Total group	Allergy	No allergy	*p*	Asthma	No asthma	*p*
N	284	120	163*		66	218	
Sex, female, *n* (%)	147 (51.8)	60 (50.0)	86 (52.8)	0.762	33 (50.0)	114 (52.3)	0.852[1]
Mother's age, mean ± *SD* [years]	35.83 ± 4.75	35.67 ± 4.75	35.92 ± 4.76	0.678[3]	35.52 ± 5.08	35.93 ± 4.66	0.559[3]
Mother's education, *n* (%)							
Primary school	12 (4.3)	5 (4.2)	7 (4.3)	0.940	3 (4.5)	9 (4.2)	0.779
Vocational school	13 (4.6)	6 (5.0)	7 (4.3)		4 (6.1)	9 (4.2)	
High school	91 (32.4)	36 (30.3)	55 (34.2)		23 (34.8)	68 (31.6)	
University	165 (58.7)	72 (60.5)	92 (57.1)		36 (54.5)	129 (60.0)	
Father's age, median (IQR) [years]	37.00 (35.00;41.00)	36.50 (34.00;41.00)	38.00 (35.00;41.00)	0.430[2]	37.00 (34.00;40.00)	38.00 (35.00;41.00)	0.268[2]
Father's education, *n* (%)							
Primary school	18 (6.7)	9 (7.8)	9 (6.0)	0.648	6 (9.5)	12 (5.9)	0.357[1]
Vocational school	31 (11.6)	14 (12.2)	17 (11.3)		10 (15.9)	21 (10.3)	
High school	103 (38.6)	39 (33.9)	64 (42.4)		20 (31.7)	83 (40.7)	
University	115 (43.1)	53 (46.1)	61 (40.4)		27 (42.9)	88 (43.1)	
Type of housing, *n* (%)							
Tower block	13 (4.6)	6 (5.0)	7 (4.3)	0.273	2 (3.0)	11 (5.0)	0.811[1]
Block	89 (31.3)	42 (35.0)	47 (28.8)		19 (28.8)	70 (32.1)	
Tenement house	30 (10.6)	16 (13.3)	14 (8.6)		9 (13.6)	21 (9.6)	
House	146 (51.4)	52 (43.3)	93 (57.1)		35 (53.0)	111 (50.9)	
Other	6 (2.1)	4 (3.3)	2 (1.2)		1 (1.5)	5 (2.3)	
Household heating, *n* (%)							
Central	102 (35.9)	50 (41.7)	52 (31.9)	0.361	21 (31.8)	81 (37.2)	0.729
Oil	2 (0.7)	0 (0.0)	2 (1.2)		0 (0.0)	2 (0.9)	
Gas	54 (19.0)	22 (18.3)	32 (19.6)		16 (24.2)	38 (17.4)	
Coal-fired	71 (25.0)	27 (22.5)	43 (26.4)		16 (24.2)	55 (25.2)	
Tiled stove	16 (5.6)	8 (6.7)	8 (4.9)		5 (7.6)	11 (5.0)	
Other	39 (13.7)	13 (10.8)	26 (16.0)		8 (12.1)	31 (14.2)	
Living conditions, *n* (%)							
Steam on windows	141 (49.6)	65 (54.2)	76 (46.6)	0.230	36 (54.5)	105 (48.2)	0.443[1]
Signs of moisture	46 (16.2)	18 (15.0)	27 (16.6)	0.162	11 (16.7)	35 (16.1)	>0.999[1]
Signs of mould	59 (20.8)	24 (20.0)	35 (21.5)	0.907	13 (19.7)	46 (21.1)	0.942[1]
Cockroaches	4 (1.4)	1 (0.8)	3 (1.8)	0.646		4 (1.8)	0.577
Carpets	150 (52.8)	68 (56.7)	81 (49.7)	0.305	36 (54.5)	114 (52.3)	0.857[1]
Frequency of house cleaning, *n* (%)							
<1 × week	7 (2.5)	5 (4.2)	2 (1.2)	0.234	3 (4.5)	4 (1.8)	0.465[1]
1 × week	51 (18.0)	16 (13.3)	35 (21.5)		9 (13.6)	42 (19.3)	
2 × week	88 (31.0)	40 (33.3)	48 (29.4)		22 (33.3)	66 (30.3)	
>2 × week	138 (48.6)	59 (49.2)	78 (47.9)		32 (48.5)	106 (48.6)	
Financial status, *n* (%)							
Very low	9 (3.2)	6 (5.0)	3 (1.9)	0.406	1 (1.5)	8 (3.7)	**0.023[1]**
Low	25 (8.9)	7 (5.8)	18 (11.2)		0 (0.0)	25 (11.6)	
Average	190 (67.4)	82 (68.3)	107 (66.5)		49 (74.2)	141 (65.3)	
High	58 (20.6)	25 (20.8)	33 (20.5)		16 (24.2)	42 (19.4)	
Pets at home, *n* (%)	122 (43.1)	44 (37.0)	77 (47.2)	0.068	20 (30.8)	102 (46.8)	**0.032[1]**
Dog	71 (25.0)	27 (22.5)	44 (27.0)	0.556	15 (22.7)	56 (25.7)	0.746[1]
Cat	32 (11.3)	9 (7.5)	23 (14.1)	0.193	4 (6.1)	28 (12.8)	0.192[1]
Other	28 (9.9)	7 (5.8)	20 (12.3)	**0.017**	2 (3.0)	26 (11.9)	0.059[1]
Rodents	20 (7.0)	9 (7.5)	11 (6.7)	0.831	4 (6.1)	16 (7.3)	0.935[1]
Birds	3 (1.1)	0 (0.0)	3 (1.8)	0.272	0 (0.0)	3 (1.4)	>0.999
Parental smoking, *n* (%)	95 (33.5)	47 (39.2)	47 (28.8)	**0.045**	27 (40.9)	68 (31.2)	0.188[1]
Passive smoking, median (IQR) [hours]	0.00 (0.00;0.00)	0.00 (0.00;0.00)	0.00 (0.00;0.00)	0.443[2]	0.00 (0.00;0.00)	0.00 (0.00;0.00)	0.079[2]
Specialist clinic, *n* (%)	91 (32.2)	58 (48.3)	33 (20.4)	**<0.001**	30 (45.5)	61 (28.1)	**0.013[1]**
Medicines taken permanently, *n* (%)	57 (20.1)	40 (33.3)	17 (10.5)	**<0.001**	22 (33.3)	35 (16.1)	**0.004[1]**
Atopy in family, *n* (%)	137 (48.6)	68 (57.1)	68 (42.0)	**0.013**	34 (52.3)	103 (47.5)	0.587[1]

* Sum of "any allergy" and "no allergy" groups does not equal 284 due to missing data on incidence of atopic dermatitis for one patient, thus the patient was excluded from the subgroup analysis; **IQR** – interquartile range; ***SD*** – standard deviation. Groups compared with Fisher's exact test, Pearson's chi-square test[1], Mann–Whitney *U* test[2] or *t*-Student test for independent groups[3]. Sum of patients in some groups may be not equal to total group due to missing data.

DOI: 10.15557/PiMR.2023.0035

Tab. 2. Comparison of the mean annual pollution levels between allergy/no allergy group vs. asthma/no asthma group

Variable	Allergy	No allergy	MD (95% CI)	*p*	Asthma	No asthma	MD (CI 95%)	*p*
$PM_{2.5}$ [μg/m³]	23.80 (15.90;35.70)	19.80 (11.90;31.70)	4.00 (0.00;8.00)	**0.019**[1]	23.80 (15.90;31.70)	19.80 (11.90;31.70)	–	0.360[1]
PM_{10} [μg/m³]	41.70 (27.80;70.43)	47.60 (27.80;69.45)	–	0.764[1]	39.70 (27.80;71.40)	47.60 (27.80;67.50)	–	0.685[1]
2018 $PM_{2.5}$ [μg/m³]	23.07 ± 3.18	22.49 ± 3.12	–	0.128	23.03 ± 3.28	22.62 ± 3.13	–	0.355
2018 PM_{10} [μg/m³]	34.22 ± 2.52	33.92 ± 2.44	–	0.325	34.92 ± 2.23	33.78 ± 2.48	1.13 (0.46;1.80)	**0.001**
2017 $PM_{2.5}$ [μg/m³]	24.50 ± 3.30	23.95 ± 3.26	–	0.171	24.11 ± 3.59	24.17 ± 3.22	–	0.885
2017 PM_{10} [μg/m³]	29.91 ± 5.00	29.18 ± 4.87	–	0.214	29.10 (25.00;35.00)	29.10 (25.00;35.00)	–	0.128[1]
2016 $PM_{2.5}$ [μg/m³]	21.47 ± 2.46	21.04 ± 2.63	–	0.160	21.60 ± 2.57	21.08 ± 2.56	–	0.150
2016 PM_{10} [μg/m³]	27.66 (25.00;35.00)	27.66 (25.00;33.74)	–	0.221[1]	27.66 (25.00;35.00)	27.66 (25.00;33.74)	–	0.101[1]
2015 $PM_{2.5}$ [μg/m³]	22.52 ± 3.78	21.50 ± 3.73	1.01 (0.13;1.90)	**0.025**	22.87 ± 4.15	21.63 ± 3.62	1.24 (0.20;2.28)	**0.019**
2015 PM_{10} [μg/m³]	29.86 ± 5.18	28.58 ± 4.75	1.28 (0.09;2.46)	**0.035**[2]	30.07 ± 5.36	28.80 ± 4.84	–	0.070
2014 $PM_{2.5}$ [μg/m³]	22.02 ± 4.52	21.53 ± 4.51	–	0.368	21.84 ± 4.63	21.68 ± 4.48	–	0.804
2014 PM_{10} [μg/m³]	26.54 ± 4.13	26.01 ± 4.28	–	0.299	25.98 ± 4.39	26.29 ± 4.19	–	0.598
2018 NO_2 [μg/m³]	17.46 ± 3.44	17.72 ± 2.90	–	0.486	17.18 ± 3.80	17.74 ± 2.89	–	0.272[2]
2017 NO_2 [μg/m³]	22.06 ± 1.62	21.96 ± 1.60	–	0.588	22.50 (22.50;22.50)	22.50 (22.50;22.50)	0.00 (0.00;0.00)	**0.003**[1]
2017 BaP [μg/m³]	2.90 ± 0.48	2.88 ± 0.41	–	0.696	2.98 ± 0.51	2.86 ± 0.41	–	0.068
BaP 2015	3.76 ± 1.18	3.63 ± 1.14	–	0.373	3.69 ± 1.35	3.67 ± 1.09	–	0.939[2]
BaP 2016	4.74 ± 1.47	4.63 ± 1.45	–	0.539	4.62 ± 1.68	4.69 ± 1.39	–	0.737
BaP 2017	3.94 ± 1.13	3.85 ± 1.10	–	0.488	3.85 ± 1.29	3.89 ± 1.06	–	0.774
BaP 2018	2.81 ± 1.01	2.70 ± 0.92	–	0.316	2.76 ± 1.11	2.74 ± 0.91	–	0.877[2]
BaP 2019	2.21 ± 0.71	2.14 ± 0.69	–	0.403	2.16 ± 0.81	2.16 ± 0.66	–	0.982
Benzene 2015	0.42 ± 0.25	0.38 ± 0.24	–	0.181	0.39 ± 0.28	0.39 ± 0.24	–	0.845
Benzene 2016	0.32 ± 0.18	0.29 ± 0.17	–	0.209	0.30 ± 0.20	0.30 ± 0.17	–	0.816
Benzene 2017	0.23 ± 0.14	0.21 ± 0.13	–	0.402	0.21 ± 0.15	0.22 ± 0.13	–	0.643
Benzene 2018	0.34 ± 0.20	0.31 ± 0.19	–	0.146	0.32 ± 0.21	0.32 ± 0.18	–	0.786
Benzene 2019	0.24 ± 0.16	0.23 ± 0.15	–	0.417	0.23 ± 0.18	0.23 ± 0.15	–	0.762
$PM_{2.5}$ 2015	18.27 ± 3.31	17.85 ± 3.13	–	0.276	17.96 ± 3.71	18.03 ± 3.05	–	0.870
$PM_{2.5}$ 2016	17.95 ± 3.19	17.62 ± 3.03	–	0.368	17.59 ± 3.57	17.79 ± 2.95	–	0.644
$PM_{2.5}$ 2017	18.48 ± 3.19	18.13 ± 3.04	–	0.351	18.12 ± 3.55	18.30 ± 2.96	–	0.675
$PM_{2.5}$ 2018	24.68 ± 4.18	24.13 ± 3.78	–	0.254[2]	24.26 ± 4.56	24.37 ± 3.78	–	0.856
$PM_{2.5}$ 2019	23.84 ± 3.58	23.41 ± 3.33	–	0.292	23.43 ± 3.96	23.62 ± 3.28	–	0.702
PM_{10} 2015	26.21 ± 4.62	25.61 ± 4.38	–	0.261	25.76 ± 5.18	25.87 ± 4.27	–	0.868
PM_{10} 2016	25.80 ± 4.46	25.31 ± 4.25	–	0.341	25.29 ± 4.98	25.56 ± 4.14	–	0.661
PM_{10} 2017	26.45 ± 4.48	25.95 ± 4.25	–	0.343	25.93 ± 4.99	26.21 ± 4.15	–	0.653
PM_{10} 2018	25.23 ± 4.92	24.57 ± 4.47	–	0.250[2]	24.75 ± 5.36	24.85 ± 4.46	–	0.880
PM_{10} 2019	23.64 ± 4.11	22.87 ± 3.57	–	0.094	23.39 ± 4.49	23.18 ± 3.65	–	0.733[2]
NO_2 2015	13.60 ± 3.80	13.02 ± 3.52	–	0.186	13.20 ± 4.18	13.27 ± 3.48	–	0.898
NO_2 2016	13.52 ± 3.76	12.96 ± 3.49	–	0.193	13.12 ± 4.13	13.21 ± 3.44	–	0.857
NO_2 2017	13.25 ± 3.71	12.72 ± 3.44	–	0.213	12.85 ± 4.09	12.96 ± 3.40	–	0.830
NO_2 2018	13.18 ± 3.90	12.58 ± 3.59	–	0.179	12.76 ± 4.26	12.84 ± 3.56	–	0.874
NO_2 2019	12.77 ± 3.60	12.22 ± 3.32	–	0.192	12.38 ± 3.95	12.46 ± 3.29	–	0.865
O_3 2015	119.95 (116.10;124.20)	121.70 (117.15;124.20)	–	0.338[1]	120.93 ± 4.34	120.20 ± 3.95	–	0.199
O_3 2016	113.64 ± 3.88	114.02 ± 3.41	–	0.394[2]	114.45 ± 3.96	113.70 ± 3.49	–	0.143
O_3 2017	105.85 (103.30;109.20)	106.40 (103.30;108.90)	–	0.788[1]	107.25 ± 2.61	106.23 ± 2.61	1.03 (0.30;1.75)	**0.006**
O_3 2018	116.90 (116.80;117.00)	116.80 (116.80;117.00)	0.10 (0.00;0.10)	**0.034**[1]	116.90 (116.80;117.00)	116.80 (116.80;117.00)	0.10 (0.00;0.10)	**0.017**[1]
O_3 2019	117.30 (115.20;117.50)	117.30 (115.20;117.40)	0.00 (0.00;0.10)	**0.009**[1]	117.40 (115.20;117.50)	117.30 (115.20;117.40)	0.10 (0.00;0.20)	**0.006**[1]

CI – confidence interval; **MD** – mean or median difference (disease vs. no disease). Data presented as mean ± standard deviation or median (interquartile range), depending on normality of distribution. Groups compared with Mann–Whitney *U* test[1], *t*-Student test for independent groups or *t*-Welch test for independent groups[2].

[1.21;6.53], $p = 0.017$). Among pollution factors, 2017 PM_{10} [μg/m^3] increased the odds of allergy with the highest strength. An increase in its concentration by 1 μg/m^3 resulted in 14% higher odds of allergy (OR = 1.14, 95% CI [1.02;1.28], $p = 0.021$). $PM_{2.5}$ measured by personal aspirators [μg/m^3] had significant, yet minor, impact on the risk of allergy (OR = 1.03, 95% CI [1.01;1.05], $p = 0.030$) (Tab. 4). Model fit was verified with Nagelkerke R^2, which was equal to 39.5% as well as with Hosmer and Lemeshow GOF test with the outcome of $p = 0.834$.

Asthma group – univariate logistic regression

2018 PM_{10} [μg/m^3] influenced the odds of asthma. An increase in its level by 1 μg/m^3 resulted in 21% higher odds of developing asthma (OR = 1.21, 95% CI [1.08;1.37], $p = 0.001$). An increase in the level of 2016 PM_{10} [μg/m^3] by 1 μg/m^3 would increase the odds of asthma by 8% (OR = 1.08, 95% CI [1.01;1.15], $p = 0.020$). Significant increase in the odds of asthma was observed for 2015 $PM_{2.5}$ [μg/m^3]. An increase in its levels higher by 1 unit resulted in 9% increased odds of asthma (OR = 1.09, 95% CI [1.01;1.17], $p = 0.021$). An increase in 2017 NO_2 [μg/m^3] by 1 μg/m^3 resulted in an increase in the risk of asthma by 37% (OR = 1.37, 95% CI [1.12;1.72], $p = 0.004$). The levels of 2017 and 2019 O_3 impacted the odds of asthma by 16% and 39%, respectively; when higher by 1 unit (OR = 1.16, 95% CI [1.04;1.30], $p = 0.006$ and OR = 1.39, 95% CI [1.08;1.82], $p = 0.013$, respectively).

Having a pet at home decreased the risk of asthma almost by half (OR = 0.51, 95% CI [0.28;0.90], $p = 0.023$). Having an animal other than a dog, a cat, a bird or a rodent was associated with 77% lower odds of asthma (OR = 0.23, 95% CI [0.04;0.80], $p = 0.049$) (Tab. 3).

Asthma group – multivariate logistic regression

In multivariate analysis, household pets were identified as a factor influencing the odds of asthma. Household pets decreased the risk of asthma by 68% compared to not having a pet (OR = 0.32, 95% CI [0.12;0.82], $p = 0.022$). Among pollution factors, 2017 O_3 increased the odds of asthma; an increase in its concentration by 1 unit resulted in 41% higher odds (OR = 1.41, 95% CI [1.02;2.04], $p = 0.049$ (Tab. 4). Model fit was verified with Nagelkerke R^2, which was equal to 34.2% as well as with Hosmer and Lemeshow GOF test with the outcome of $p = 0.286$.

DISCUSSION

The global geographic range of allergy and asthma cases has increased significantly. Urban children are highly exposed to air pollution, and other environmental factors that trigger asthma symptoms[18,19]. Tobacco smoke is one of the most common indoor pollutants, although the relationship between PS and the development of allergic sensitisation in children is still unclear.

Our study showed a strong relationship between environmental pollutants, $PM_{2.5}$, PM_{10}, O_3 and NO_2, and asthma, as well as between PS and higher levels of $PM_{2.5}$, PM_{10} and O_3 and allergy in Polish preschoolers. Environmental pollution may be a risk factor for the increasing prevalence of allergy and asthma. Health officials should take priority preventive measures to limit environmental pollution in order to combat allergic diseases.

A meta-analysis by Feleszko et al. revealed that PS can increase the risk of atopic sensitisation, as assessed by skin prick tests and/or specific IgEs, and this effect was most pronounced in preschoolers[20]. PS may increase the risk of sensitisation to food allergens and atopic dermatitis[21] as well as allergic rhinitis[22]. Regular maternal smoking seems to be a strong risk factor for allergic sensitisation and asthma in children with a family history of atopy[23]. These observations are in accordance with our results, which revealed that PS was associated with 3-fold increase in the risk of allergy.

In 2016, the WHO reported that 92% of the world's population lived in areas where air quality levels exceed the WHO's ambient air quality guidelines for annual mean of $PM_{2.5}$[6,24] and the effects may be particularly harmful for children[6]. Two years later, air pollution (both indoor and outdoor) was recognised by the WHO as the single largest environmental health risk in Europe[25]. In our study, significantly higher levels of $PM_{2.5}$ measured by personal aspirator (which sampled mainly indoor dust), 2015 $PM_{2.5}$ and 2015 PM_{10} were found in the allergy group compared with the no-allergy group. All our study participants were born in 2015. In the asthmatic group, higher levels of 2015 $PM_{2.5}$, 2018 PM_{10}, 2017 NO_2 as well as 2017 and 2019 O_3 were reported. A meta-analysis based on birth cohorts data including almost 15,000 children from three European countries clearly showed that exposure to increased $PM_{2.5}$ since birth was associated with increased asthma prevalence through adolescence[26]. Bowatte et al. shared similar observations regarding increased longitudinal exposure to $PM_{2.5}$ from traffic-related air pollution (TRAP) when analysing North American cohorts of children till the age of 12 years[27]. After adjusting for potential confounding factors in our study, $PM_{2.5}$ measured by personal aspirators and 2017 PM_{10} were significantly more often associated with allergy, whereas increased levels of O_3 (2017 O_3) were associated with asthma. In univariate regression $PM_{2.5}$ and PM_{10} were correlated with allergy, while $PM_{2.5}$, PM_{10}, NO_2 and O_3 were correlated with asthma. Similar findings were reported by Nishimura et al., who showed associations between asthma and PM_{10} as well as an increased risk for subsequent asthma in children exposed to NO_2 during infancy[3]. By far, there is no clear evidence for the impact of O_3 exposure on the development of asthma. Some studies collected in a review by Zu et al. revealed statistically significant positive correlations

 DOI: 10.15557/PiMR.2023.0035

Tab. 3. Univariate logistic regression models for allergy group vs. asthma group

Variable	Allergy			Asthma		
	OR	95% CI	*p*	OR	95% CI	*p*
Sex, female (vs. male)	0.90	0.56–1.44	0.646	0.91	0.52–1.58	0.744
Mother's education, vocational school (vs. primary school)	1.20	0.24–6.05	0.821	1.33	0.23–8.48	0.749
Mother's education, high school (vs. primary school)	0.92	0.27–3.30	0.889	1.01	0.28–4.86	0.984
Mother's education, university (vs. primary school)	1.10	0.34–3.84	0.880	0.84	0.24–3.92	0.798
Father's education, vocational school (vs. primary school)	0.82	0.25–2.66	0.744	0.95	0.28–3.40	0.938
Father's education, high school (vs. primary school)	0.61	0.22–1.69	0.335	0.48	0.16–1.52	0.191
Father's education, university (vs. primary school)	0.87	0.32–2.38	0.782	0.61	0.22–1.90	0.371
Type of housing, block (vs. tower block)	1.04	0.32–3.47	0.944	1.49	0.36–10.18	0.621
Type of housing, tenement house (vs. tower block)	1.33	0.36–5.07	0.666	2.36	0.49–17.26	0.322
Type of housing, house (vs. tower block)	0.65	0.21–2.12	0.463	1.73	0.44–11.55	0.487
Type of housing, other (vs. tower block)	2.33	0.33–21.56	0.410	1.10	0.04–14.42	0.943
Housing heating, oil (vs. central)	0.00	–	0.981	0.00	–	0.989
Housing heating, gas (vs. central)	0.71	0.36–1.39	0.324	1.62	0.76–3.46	0.209
Housing heating, coal-fired (vs. central)	0.65	0.35–1.21	0.177	1.12	0.53–2.33	0.759
Housing heating, tiled-stove (vs. central)	1.04	0.36–3.03	0.942	1.75	0.51–5.41	0.343
Housing heating, other (vs. central)	0.52	0.24–1.11	0.096	1.00	0.38–2.41	0.992
Steam on windows	1.35	0.84–2.18	0.210	1.29	0.74–2.25	0.364
Signs of moisture	0.89	0.46–1.69	0.722	1.05	0.48–2.14	0.906
Signs of mould	0.91	0.51–1.63	0.763	0.92	0.45–1.79	0.805
Cockroaches	0.45	0.02–3.58	0.494	0.00	–	0.984
Carpets	1.32	0.83–2.13	0.246	1.09	0.63–1.91	0.748
Frequency of house cleaning, 1 × week (vs. <1 × week)	0.18	0.02–0.95	0.056	0.29	0.05–1.65	0.139
Frequency of house cleaning, 2 × week (vs. <1 × week)	0.33	0.05–1.64	0.203	0.44	0.09–2.40	0.312
Frequency of house cleaning, >2 × week (vs. <1 × week)	0.30	0.04–1.46	0.162	0.40	0.08–2.13	0.249
Financial status, low (vs. very low)	0.19	0.03–0.94	0.051	0.00	–	0.984
Financial status, average (vs. very low)	0.38	0.08–1.50	0.184	2.78	0.49–52.23	0.341
Financial status, high (vs. very low)	0.38	0.07–1.58	0.199	3.05	0.50–58.86	0.311
Pets at home	0.66	0.40–1.06	0.086	0.51	0.28–0.90	**0.023**
Dog	0.79	0.45–1.36	0.389	0.85	0.43–1.60	0.627
Cat	0.49	0.21–1.08	0.087	0.44	0.13–1.17	0.136
Other	0.44	0.17–1.04	0.075	0.23	0.04–0.80	**0.049**
Rodents	1.12	0.44–2.80	0.807	0.81	0.23–2.32	0.722
Birds	0.00	–	0.985	0.00	–	0.986
Parental smoking	1.59	0.96–2.62	0.069	1.53	0.86–2.69	0.144
Passive smoking [hours]	0.98	0.86–1.09	0.667	1.01	0.86–1.13	0.921
Specialist clinic	3.66	2.18–6.23	**<0.001**	2.13	1.20–3.76	**0.009**
Medicines taken permanently	4.26	2.31–8.18	**<0.001**	2.60	1.38–4.85	**0.003**
Atopy in family	1.84	1.14–2.99	**0.012**	1.21	0.70–2.12	0.494
PM_{10} [μg/m^3]	1.00	1.00–1.00	0.846	1.00	1.00–1.00	0.601
2018 $PM_{2.5}$ [μg/m^3]	1.06	0.98–1.15	0.129	1.04	0.96–1.14	0.354

Variable	Allergy			Asthma		
	OR	95% CI	*p*	OR	95% CI	*p*
2018 PM_{10} [µg/m^3]	1.05	0.95–1.16	0.324	1.21	1.08–1.37	**0.001**
2017 $PM_{2.5}$ [µg/m^3]	1.05	0.98–1.14	0.171	0.99	0.92–1.08	0.885
2017 PM_{10} [µg/m^3]	1.03	0.98–1.08	0.213	1.05	1.00–1.11	0.060
2016 $PM_{2.5}$ [µg/m^3]	1.07	0.97–1.18	0.160	1.09	0.97–1.23	0.151
2016 PM_{10} [µg/m^3]	1.04	0.98–1.10	0.184	1.08	1.01–1.15	**0.020**
2015 $PM_{2.5}$ [µg/m^3]	1.07	1.01–1.15	**0.026**	1.09	1.01–1.17	**0.021**
2015 PM_{10} [µg/m^3]	1.05	1.00–1.11	**0.033**	1.05	1.00–1.11	0.071
2014 $PM_{2.5}$ [µg/m^3]	1.02	0.97–1.08	0.367	1.01	0.95–1.07	0.803
2014 PM_{10} [µg/m^3]	1.03	0.97–1.09	0.298	0.98	0.92–1.05	0.597
2018 NO_2 [µg/m^3]	0.97	0.90–1.05	0.485	0.94	0.86–1.03	0.203
2017 NO_2 [µg/m^3]	1.04	0.90–1.21	0.586	1.37	1.12–1.72	**0.004**
2017 BaP [µg/m^3]	1.11	0.65–1.91	0.694	1.81	0.96–3.47	0.069
BaP 2015	1.10	0.89–1.35	0.372	1.01	0.79–1.28	0.931
BaP 2016	1.05	0.89–1.24	0.538	0.97	0.80–1.17	0.736
BaP 2017	1.08	0.87–1.33	0.486	0.96	0.75–1.23	0.774
BaP 2018	1.13	0.89–1.45	0.316	1.03	0.77–1.36	0.862
BaP 2019	1.16	0.82–1.62	0.402	1.00	0.66–1.47	0.982
Benzene 2015	1.93	0.74–5.07	0.181	0.89	0.28–2.72	0.844
Benzene 2016	2.35	0.62–9.04	0.209	0.83	0.16–3.89	0.816
Benzene 2017	2.17	0.35–13.30	0.401	0.60	0.07–4.94	0.641
Benzene 2018	2.50	0.73–8.68	0.146	0.82	0.19–3.43	0.785
Benzene 2019	1.87	0.41–8.47	0.416	0.76	0.12–4.34	0.761
$PM_{2.5}$ 2015	1.04	0.97–1.12	0.275	0.99	0.91–1.08	0.869
$PM_{2.5}$ 2016	1.04	0.96–1.12	0.367	0.98	0.89–1.07	0.642
$PM_{2.5}$ 2017	1.04	0.96–1.12	0.350	0.98	0.90–1.07	0.674
$PM_{2.5}$ 2018	1.04	0.98–1.10	0.246	0.99	0.93–1.06	0.855
$PM_{2.5}$ 2019	1.04	0.97–1.11	0.291	0.98	0.91–1.07	0.701
PM_{10} 2015	1.03	0.98–1.09	0.260	0.99	0.93–1.06	0.867
PM_{10} 2016	1.03	0.97–1.08	0.340	0.99	0.92–1.05	0.660
PM_{10} 2017	1.03	0.97–1.08	0.342	0.99	0.92–1.05	0.652
PM_{10} 2018	1.03	0.98–1.08	0.242	1.00	0.94–1.06	0.880
PM_{10} 2019	1.05	0.99–1.12	0.095	1.01	0.94–1.09	0.702
NO_2 2015	1.04	0.98–1.11	0.186	1.00	0.92–1.07	0.897
NO_2 2016	1.04	0.98–1.12	0.193	0.99	0.92–1.07	0.857
NO_2 2017	1.04	0.98–1.11	0.212	0.99	0.92–1.07	0.829
NO_2 2018	1.04	0.98–1.11	0.179	0.99	0.92–1.07	0.874
NO_2 2019	1.05	0.98–1.12	0.192	0.99	0.91–1.07	0.865
O_3 2015	0.97	0.92–1.03	0.329	1.05	0.98–1.12	0.199
O_3 2016	0.97	0.91–1.04	0.383	1.06	0.98–1.15	0.144
O_3 2017	1.01	0.92–1.11	0.820	1.16	1.04–1.30	**0.006**
O_3 2018	1.14	0.94–1.38	0.170	1.11	0.89–1.37	0.330
O_3 2019	1.21	0.98–1.50	0.079	1.39	1.08–1.82	**0.013**

CI – confidence interval; **OR** – odds ratio.

Tab. 3. Univariate logistic regression models for allergy group vs. asthma group (cont.)

DOI: 10.15557/PiMR.2023.0035

Variable	OR	95% CI	*p*
Any allergy			
Pets at home	0.48	0.21–1.04	0.068
Parental smoking	2.76	1.21–6.53	**0.017**
Specialist clinic	2.64	1.15–6.21	**0.024**
Medicines taken permanently	4.71	1.59–15.19	**0.007**
$PM_{2.5}$ [µg/m^3]	1.03	1.01–1.05	**0.030**
2018 $PM_{2.5}$ [µg/m^3]	0.85	0.70–1.03	0.103
2017 PM_{10} [µg/m^3]	1.14	1.02–1.28	**0.021**
O_3 2018	1.43	0.93–2.29	0.111
Asthma			
Pets at home	0.32	0.12–0.82	**0.022**
Parental smoking	2.10	0.81–5.47	0.126
Medicines taken permanently	10.18	3.35–35.06	**<0.001**
2015 PM_{10} [µg/m^3]	0.98	0.85–1.12	0.717
2017 PM_{10} [µg/m^3]	1.06	0.89–1.25	0.502
2018 PM_{10} [µg/m^3]	0.96	0.57–1.60	0.878
O_3 2017	1.41	1.02–2.04	**0.049**
O_3 2019	1.12	0.67–1.89	0.668

CI – confidence interval; **OR** – odds ratio.

Tab. 4. Multivariate logistic regression models for allergy group and asthma group

between the risk of childhood asthma and exposure to O_3, whereas others found a null association[28]. In their review on oxidative stress and its impact on asthma, Enweasor et al. showed O_3 was found to be a significant contributor to respiratory illness[29]. In particular, O_3 induced airway hyperreactivity in mouse models of asthma[30], in Th2-low asthma in macaques[31] and in patients with asthma and allergic comorbidities[32–35]. However, further research is needed to fully understand the role of O_3 in the development of asthma in paediatric patients.

The fact that only a single measurement of environmental pollution over 24 hours was performed was a limitation of our study, but according to suggestions from previous epidemical studies, every attempt at assessing indoor and personal concentrations in life environments is necessary to evaluate the total exposure to air pollution[36,37]. It is therefore suggested that further similar studies may be conducted to reach better conclusions.

Our observations indicate that in the case of children, air pollution is likely to have stronger effect on triggering asthma than allergy, perhaps due to induction of non-allergic inflammation. It is in accordance with data collected by Gruzieva et al., indicating that exposure to ambient air pollution did not increase the risk of sensitisation to common allergens up to school age[38]. The value of our study is that the air pollutants have been monitored for 5 years, that is, the whole life of the participants. An additional advantage is that the contamination was assessed by a modelling method, which, compared to other methods, allows to determine the concentration of pollutants throughout the studied area, takes into account the contribution of individual emission sources in total concentrations, as well as meteorological and geographical data. Additionally, attention should be paid to the fact that the limit of the mean annual concentrations of $PM_{2.5}$, PM_{10} and BaP (included in PM_{10}) was exceeded at the measuring stations in each of the years analysed (2015–2019). The target levels of 8-hour O_3 were also exceeded. No exceedances of the annual mean concentrations of air pollutant for which we found no relationship with allergy and asthma in the study group of children have been recorded, such as benzene, NO_2, or SO_2. Our observations suggest that pollutants may affect allergy and asthma only after exceeding the permitted concentrations. Further research is needed to confirm our findings.

DOI: 10.15557/PiMR.2023.0035

Conflict of interest

The authors have no conflicts of interest regarding this manuscript.

Funding/Support and role of the sponsor

This study was supported by the National Science Centre (grant No. 2017/25/B/NZ7/00161).

Author contributions

Original concept of study: JJ, WS. Collection, recording and/or compilation of data: MBK, DP, MG. Analysis and interpretation of data: MBK, KP, MP. Writing of manuscript: MBK. Critical review of manuscript: JJ, KP, WS. Final approval of manuscript: MBK, WS.

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DOI: 10.15557/PiMR.2023.0035 PEDIATR MED RODZ Vol. 19 No. 3, p. 195–206