

Metanalysis: Respiratory Effects in the General Population Exposed to Urban Pollution

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Received March 30th, 2011; revised July 14th, 2011; accepted August 24th, 2011.

ABSTRACT

Aim: The purpose of this study was to evaluate spirometric lung function parameters in the general population exposed to urban pollution and confirm the existence of an association between exposure to environmental pollutants and effects from these products and which respiratory parameters are associated to urban pollution in general population.

Methods: This study is a systematic research of all articles on the assessment of respiratory effects on general population exposed to urban pollution, excluding studies on adolescents and children. The research included articles from January 2008 to May 2009. In the articles included in our meta-analysis, the exposed group is represented by general population aged between 15 and 75 years for both genders, resident in very polluted urban areas, while the control group is represented by general population resident in rural and suburban areas, where pollution is lower.

Results: The results confirm the presence of statistically significant effects of urban pollution on the respiratory system for cough, phlegm, shortness of breath/ breathlessness, wheezing, FVC, FEV1, PEFr, chronic bronchitis, bronchial asthma, rhinitis, emphysema.

Keywords: Urban Pollution, Respiratory Symptoms on General Population, Lung Disease, Environmental Exposure

1. Introduction

On the basis of the assessment conducted by WHO, urban pollution is a very significant public health problem. Studies of assessment have invariably shown that adverse effects of air pollution on health are substantial.

Measures of containment have produced a reduction of urban pollution levels, but results of recent studies continue to emphasize acute and chronic effects on health, even when these levels are low.

Epidemiological research has consistently documented a wide range of adverse effects to human health for exposure to pollutants: it has been documented a wide range of adverse health outcomes due to short and long term exposure to air pollutant concentration levels for urban populations.

Numerous clinical and toxicological studies have provided significant information about the specific effects of pollutants and the possible mechanisms of action these effects have.

General population is daily exposed to pollutants of various kinds. These pollutants are made up of chemical and physical (*i.e.* noise).

Furthermore, the presence of high levels of air pollution is connected to respiratory symptoms and disease in various European and non European countries: in the U.S., the presence of chronic bronchitis was significantly associated with fine particle pollution [1].

In Italy it was shown that residents in a rural area have prevalence rates of respiratory symptoms lower than children who live in urban areas [21,22].

Another important European study has shown, in adults, an increased risk of developing a framework of bronchitis resulting from exposure to traffic pollution from motor vehicles [2]. The respiratory symptoms of 40 - 59 year old women, who spent much of their time at home, was related to the proximity of their residence to high motor vehicle traffic areas [15]. Other studies to remember in this context are conducted on a population of at least 10 years in metropolitan areas or rural areas, where there has been a significant association between symptoms of chronic obstructive respiratory disease and high levels of particulates [3].

The purpose of this study was to evaluate spirometric lung function parameters in the general population exposed to urban pollution and confirm the existence of an

association between exposure to environmental pollutants and effects from these products and which respiratory parameters are associated to urban pollution in general population.

2. Materials and Methods

This study has been conducted with a systematic research of all articles on the assessment of respiratory effects in the general population exposed to urban pollution, excluding studies on adolescents and children. The research included articles from January 2008 (year of enactment of Presidential Decree 203/88: "Implementation of directives CEE n. 80/779, 82/884, 84/360 and 85/203 that pertain to air quality directives, relatively to specific pollutant and the pollution produced by industry, pursuant to art. 15 of Law of April 16, 1987, number 183") to May 2009.

We used the following electronic search engines, available online:

- Biomedcentral
- MEDLINE/ PubMed
- MEDLINE/ National Library of Medicine (NLM)
- MEDLINE Plus
- Nioshtic-2
- Scopus
- TOXNET/Toxline

Furthermore we examined the acts of national congresses organized by S.I.M.L.I.I. (Italian Society of Occupational Health and Industrial Hygiene) and by A.I.D.I.I. (Italian Association of Industrial Hygienist) and many books of Environmental health.

For all search engines we used the following key words:

- Air pollution (or pollutant) and urban (or rural or general) population
- Urban pollution (or pollutants) and urban (or rural or general) population
- Urban air pollution and urban (or rural or general) population
- Urban atmospheric pollution (or pollutant) and urban (or rural or general) population
- Ambient air (pollution) and urban (or rural or general) population
- Ambient exposure and urban (or rural or general) population
- Environmental exposure and urban (or rural or general) population
- Particulate matter (PM) and urban (or rural or general) population
- Urban particulate matter (UPM) and urban (or rural or general) population
- Ultrafine particulate matter and urban (or rural or general) population

- Ultrafine particles (fine particles) and urban (or rural or general) population
- Concentrated ambient fine particles (CAP) and urban (or rural or general) population
- Volatile organic compounds (VOCs) and urban (or rural or general) population
- Suspended particulate matter (SPM) and urban (or rural or general) population
- Total suspended particulate matter (TSPM) and urban (or rural or general) population
- Traffic emissions (air pollution) and urban (or rural or general) population
- Urban traffic and urban (or rural or general) population
- Road traffic (pollution) and urban (or rural or general) population
- High (or heavy) traffic density and urban (or rural or general) population

Of the 976 publications we found, 85 turned out to be inherent with the aim of our study and only 22 responded to the following inclusion criteria:

1) Case-Control studies, studies in which the experimental group was composed of subjects vulnerable to urban pollution and the control group was made up of subjects exposed to a lower degree of urban pollution;

2) Studies that reported results in numerical terms of media and standard deviation (for the continues variables) or of frequency (for not continue variables); their main characteristics are reported in **Tables 1-3**.

We tried to contact the authors of publications in which the results were expressed in unvailable numeric form, to obtain substantial data but did not have any answer.

3. Description of Participants

In the articles included in our meta-analysis, the exposed group is represented by general population aged between 15 and 75 years for both gender, resident in most polluted urban areas, while the control group is represented by general population resident in rural and suburban areas, where pollution is lower.

The numbers of participants included in this meta-analysis is 48.848; the number of cases is 22.414 while the number of controls is 26.434.

In the studies, where is specified, total number of male subjects in the case group is 10.357 while the number of female is 10.965. In the studies, where is specified, total number of male subjects in the control group is 13.457 while the number of female is 12.136.

4. Data Organization

After a careful analysis of selected studies we have identified the most frequently studied variables on the

Table 1. Distribution of studies included in the variables.

Class	Variables	Authors	To t
Prevalence of respiratory symptoms	Cough	Van der Zee <i>et al.</i> 2000; Bjornsson <i>et al.</i> 1994; Viegi <i>et al.</i> 1991; Viegi <i>et al.</i> 1999; Viegi <i>et al.</i> 2004; Sunyer <i>et al.</i> 2006; Devereux <i>et al.</i> 1996; Walraven <i>et al.</i> 2001; Kumar <i>et al.</i> 2000; Sekine <i>et al.</i> 2004; Jedrychowski <i>et al.</i> 1989.	11
	Phlegm	Van der Zee <i>et al.</i> 2000; Viegi <i>et al.</i> 1991; Viegi <i>et al.</i> 1999; Viegi <i>et al.</i> 2004; Sunyer <i>et al.</i> 2006; Kumar <i>et al.</i> 2000; Sekine <i>et al.</i> 2004; Jedrychowski <i>et al.</i> 1989.	8
	Dyspnea	Viegi <i>et al.</i> 1991; Viegi <i>et al.</i> 1999; Viegi <i>et al.</i> 2004; Jedrychowski <i>et al.</i> 1989.	4
	Short breath/breathlessness	Devereux <i>et al.</i> 1996; Wieringa <i>et al.</i> 1997; Sekine <i>et al.</i> 2004;	3
	Wheezing	Van der Zee <i>et al.</i> 2000; Bjornsson <i>et al.</i> 1994; Viegi <i>et al.</i> 1991; Viegi <i>et al.</i> 1999; Devereux <i>et al.</i> 1996; Wieringa <i>et al.</i> 1997; Wieringa <i>et al.</i> 1998; Wieringa <i>et al.</i> 2001; Walraven <i>et al.</i> 2001; Burr <i>et al.</i> 2004; Sekine <i>et al.</i> 2004; Jedrychowski <i>et al.</i> 1989	12
Lung Function	Fev ₁	Boezen <i>et al.</i> 1998; Kumar <i>et al.</i> 2000; Chattopadhyay <i>et al.</i> 2007; Sichletidis <i>et al.</i> 2005; Vanderjagt <i>et al.</i> 2004; Lubinski <i>et al.</i> 2005; Sekine <i>et al.</i> 2004; Heydarpour <i>et al.</i> 2007	8
	Fvc	Kumar <i>et al.</i> 2000; Chattopadhyay <i>et al.</i> 2007; Sichletidis <i>et al.</i> 2005; Vanderjagt <i>et al.</i> 2004; Lubinski <i>et al.</i> 2005; Sekine <i>et al.</i> 2004; Heydarpour <i>et al.</i> 2007	7
	Fev ₁ /Fvc	Chattopadhyay <i>et al.</i> 2007; Sichletidis <i>et al.</i> 2005; Vanderjagt <i>et al.</i> 2004; Lubinski <i>et al.</i> 2005; Heydarpour <i>et al.</i> 2007	5
	PEFR	Kumar <i>et al.</i> 2000; Chattopadhyay <i>et al.</i> 2007; Vanderjagt <i>et al.</i> 2004; Lubinski <i>et al.</i> 2005	4
	Fef ₂₅₋₇₅	Chattopadhyay <i>et al.</i> 2007; Sichletidis <i>et al.</i> 2005; Vanderjagt <i>et al.</i> 2004; Heydarpour <i>et al.</i> 2007;	4
	Fef ₇₅₋₈₅	Chattopadhyay <i>et al.</i> 2007	1
	Chronic Bronchitis	Viegi <i>et al.</i> 1991; Viegi <i>et al.</i> 1999; Viegi <i>et al.</i> 2004; Kumar <i>et al.</i> 2000	4
Prevalence of respiratory disease	Asthma	Van der Zee <i>et al.</i> 2000; Bjornsson <i>et al.</i> 1994; Viegi <i>et al.</i> 1991; Viegi <i>et al.</i> 1999; Viegi <i>et al.</i> 2004; Wieringa <i>et al.</i> 1997; Wieringa <i>et al.</i> 1998; Wieringa <i>et al.</i> 2001; Walraven <i>et al.</i> 2001; Burr <i>et al.</i> 2004; Sekine <i>et al.</i> 2004	11
	Rinitis	Sichletidis <i>et al.</i> 2005; Viegi <i>et al.</i> 1991; Burr <i>et al.</i> 2004; Kumar <i>et al.</i> 2000	4
	Emphisema	Viegi <i>et al.</i> 1991; Viegi <i>et al.</i> 1999; Viegi <i>et al.</i> 2004; Wieringa <i>et al.</i> 2001	4
	Drug use for asthma	Van der Zee <i>et al.</i> 2000; Devereux <i>et al.</i> 1996; Wieringa <i>et al.</i> 1998; Wieringa <i>et al.</i> 2001; Burr <i>et al.</i> 2004	5

Table 2. Statistical Analysis for variables.

Classe	Variabile	Campione	Risultato	Indici della meta-analisi		
				P	I ² %	ES
Prevalence of respiratory symptoms	Cough	GE: 11389 GC: 16009	Increased in the exposed group	Z = 3.556 P = 0.000	72.890	OR 1.277 [1.116; 1.461]
	Phlegm	GE: 4971 GC: 7837	Increased in the exposed group	Z = 2.030 P = 0.042	66.369	OR 1.239 [1.007; 1.524]
	Dyspnea	GE: 3499 GC: 5590	NS	Z = -0.144 P = 0.885	99.985	OR 0.948 [0.459; 1.959]
	Short breath/breathlessness	GE: 2834 GC: 3979	Increased in the exposed group	Z = 2.623 P = 0.009	78.948	OR 2.058 [1.200; 3.528]
	Wheezing	GE: 16322 GC: 21040	Increased in the exposed group	Z = 4.657 P = 0.000	82.950	OR 1.402 [1.216; 1.617]
	FEV ₁	GE: 2282 GC: 1810	decreased in the exposed group	Z = -2.904 P = 0.004	79.272	SMD -0.282 [-0.472; 0.092]
	FVC	GE: 2191 GC: 1712	decreased in the exposed group	Z = -4.062 P = 0.000	21.996	WMD -0.139 [-0.206; 0.072]
Lung Function	FEV ₁ /FVC	GE: 2033 GC: 1536	NS	Z = -1.512 P = 0.131	87.879	SMD -0.230 [-0.528; 0.068]
	PEFR	GE: 336 GC: 736	decreased in the exposed group	Z = -4.779 P = 0.000	0.000	SMD -0.320 [-0.452; -0.189]
	Fef ₂₅₋₇₅	GE: 1883 GC: 1207	NS	Z = -1.644 P = 0.100	26.788	WMD -0.064 [-0.139; 0.012]
	Fef ₇₅₋₈₅	GE: 94 GC: 289	NS	Z = -1.673 P = 0.094	46.843	WMD -0.199 [-0.433; 0.034]
Prevalence of respiratory disease	Chronic Bronchitis	GE: 4252 GC: 7602	Increased in the exposed group	Z = 4.147 P = 0.000	52.391	OR 2.256 [1.536; 3.313]
	Asthma	GE: 15643 GC: 21176	Increased in the exposed group	Z = 3.864 P = 0.000	77.540	OR 1.573 [1.250; 1.958]

Rinitis	GE: 2862 GC: 4630	Increased in the exposed group	Z = 2.123 P = 0.034	97.217	OR 2.824 [1.083; 7.361]
Emphisema	GE: 5639 GC: 9390	Increased in the exposed group	Z = 5.057 P = 0.000	78.247	OR 2.976 [1.950; 4.541]
Drug use for asthma	GE: 6307 GC: 6903	NS	Z = 1.474 P = 0.140	60.016	OR 1.201 [0.941; 1.533]

GE: group of exposed; GC: group of control; NS: Not Significant; P: Probability; I²: Inconsistence Index; ES: Effect Size; SMD: Standardized Mean Difference; WMD: Weighted Mean Difference; OR: Odds Ratio.

Table 3. Statistical Analysis for class.

Classe	Campione	Risultato	Indici della meta-analisi		
			P	I ² %	ES
Prevalence of respiratory symptoms	GE: 16,509 GC: 21,319	High in the exposed group	Z = 5.26 P = 0.00	85.23	OR 1.30 [1.17; 1.43]
Lung Function	GE: 2282 GC: 1810	Low in the exposed group	Z = -4.08 P = 0.00	85.58	SMD -0.21 [-0.31; -0.11]
Prevalence of respiratory disease	GE: 20,009 GC: 24,419	High in the exposed group	Z = 4.89 P = 0.00	92.16	OR 1.66 [1.35; 2.04]

GE: group of exposed; GC: group of controls; P: Probability; I²: Inconsistence Index; ES: Effect Size; SMD: Standardized Mean Difference; OR: Odds Ratio.

assessment of the effects on the respiratory systems and of the factors related to the onset of lung disease.

The following variables contained data expressed as means and standard deviations:

- FEV1
- FVC
- PEFR
- FEV1/FVC (Tiffeneau index)
- FEF 25 - 75
- FEF 75 - 85

The following variables gave data expressed in frequency:

- cough
- sputum
- wheezing
- dyspnea
- shortness of breath / breathlessness
- Chronic bronchitis
- Asthma
- emphysema
- use of medications for asthma

5. Statistical Analysis

The Effect Size (ES) is a value that expresses the magnitude of the association strength between two variables and was used to express the result of our meta-analysis.

The confidence interval of the effect size was also calculated, that expresses the accuracy with which the effect size was estimated in our study.

In our study the confidence interval corresponds to 95% of the observations, P value was set equal to $P < 0.05$. The P value that is necessarily correlated to the confidence interval allows to express the significance of the ES.

When studies reported data expressed as mean and standard deviation, the ES was expressed in Standardized Mean Difference or in Weighted Mean Difference depending on the value of the Index of Inconsistency (I²).

The Inconsistency Index was used as a measure of heterogeneity. In the systematic review, the heterogeneity relates to the variability or the difference among the studies on the evaluation of the effects.

With the I², we calculated the percentage of variance due to real heterogeneity rather than to the blind chance.

If the I² value is near to the zero, the observed variance is referable to the blind chance while if the I² value is high the variance is referable to different factors that must be better investigated.

The calculation of heterogeneity was used for the choice of the statistical model to calculate the ES.

In the presence of a high Inconsistency Index (I² > 50%), the ES was evaluated with the Random Effects Model (REM) that is a statistical model in which the confidence interval is influenced by the sampling error internal to the study and by the variability among the studies included in the meta-analysis. In this case, the REM is more robust because it provides wider confidence intervals than those obtained from another model which the Fixed Effects Model (FEM). The quantification of the ES was calculated with the Standardized Mean Difference (SMD) that shows the relationship between the difference of two averages and an estimator of standard deviation inside the group.

Without a high inconsistency between the studies (I² < 50%), the ES was calculated with the FEM. In this model, only the variations inside the study can influence the confidence interval. So, the quantification of the ES is made with the Weighted Mean Difference (WMD), which allows to combine the measures of a continue

scale when average, standard deviation and numerosity of the sample are known parameters. The importance that is given to each study is determined by the precision of the estimator of the effect provided that all studies have measured the variable with the same rating scale. A difference of 0.0 showed lack of difference among studied groups, for the measures of ES based on the differences (*i.e.* SMD and WMD). The ES was expressed in terms of Odds Ratio (OR), when the studies reported data expressed in frequency.

As described above, in the presence of a high degree of heterogeneity ($I^2 > 50\%$), the ES was assessed with the Random Effects Model (REM) while in the absence of high heterogeneity among studies ($I^2 < 50\%$), the calculation of the ES was performed with the Fixed Effects Model (FEM).

A ratio of 1.0 indicates no difference among the studied groups, for measures of ES based on the reports (*i.e.* OR) [4].

The results are been detected on the basis of the activity of research after a convention between the University of Rome "Sapienza" (Unit of Occupational Medicine) and INAIL (National Institute for the Insurance against the Accident at Work).

6. Results

From the elaboration of the data inherent to each class we had the following results [1-26]:

- The prevalence of respiratory symptoms, evaluated in 15 studies, on a total sample of 16.509 cases and 21.319 controls, appears significantly higher in the group of subjects exposed to urban pollution than in the group of less exposed subjects (OR 1.30 [1.18; 1.43]) the value of heterogeneity among the studies was I^2 85.23.
- The lung function, evaluated in 8 studies, on a total sample of 2.282 cases and 1.810 controls, appears significantly lower in the group of less exposed subjects to urban pollution than in the group of less exposed subjects (SMD -0.21 [-0.31 ; -0.11]) the value of heterogeneity among the studies was I^2 85.58.
- The prevalence of respiratory disease, evaluated in 13 studies, on a total sample of 20.009 cases and 24.419 controls, appears significantly higher in the group of subjects exposed to urban pollution than in the group of less exposed subjects (OR 1.66 [1.35; 2.04]) the value of heterogeneity among the studies was I^2 92.16.

The statistical analysis of data expressed as mean and standard deviation gave the following results (**Table 2**):

- The average FVC, evaluated in 7 studies, on a total sample of 2.191 cases and 1.712 controls, appears significantly lower in the group of subjects exposed to

urban pollution than in the group of less exposed subjects (WMD -0.139 [-0.206 ; -0.072]) the value of heterogeneity among the studies was I^2 21.996 ($P = 0.000$).

- The average FEV₁, evaluated in 8 studies, on a total sample of 2.282 cases and 1.810 controls, appears significantly lower in the group of subjects exposed to urban pollution than in the group of less exposed subjects (SMD -0.282 [-0.472 ; -0.092]) the value of heterogeneity among the studies was I^2 79.272 ($P = 0.004$).
- The PEF average evaluated in 4 studies, on a total sample of 336 cases and 736 controls, appears significantly lower in the group of subjects exposed to urban pollution than in the group of less exposed subjects (SMD -0.320 [-0.452 ; -0.189]) the value of heterogeneity among the studies was (I^2 0.00; $P = 0.000$).
- For all other evaluated parameters (FEV1/FVC, FEF25-75, FEF75-85) there were no statistically significant differences in the comparison of values found in the exposed group and in the group of less exposed.

The statistical treatment of data expressed in frequency gave the following results (**Table 2**):

- Prevalence of respiratory symptoms: cough (OR 1.277 [1.116 to 1.461]; I^2 72.890, P 0.000), phlegm (OR 1.239 [1.007 to 1.524]; I^2 66.369, P 0.042), wheezing (OR 1.402 [1.216 to 1.617]; I^2 82.950, P 0.000), shortness of breath/breathlessness (OR 2.058 [1.200 to 3.528]; I^2 78.948, P 0.009), increased significantly in the group of subjects exposed to urban air pollution compared to the group of less exposed;
- Prevalence of respiratory disease: chronic bronchitis (OR 2.256 [1.536 to 3.313]; I^2 52.391, P 0.000), bronchial asthma (OR 1.573 [1.250 to 1.980]; I^2 77.540, P 0.000), rhinitis (OR 2.824 [1.083 to 7.361]; I^2 97.217, P 0.034) and emphysema (OR 2.976 [1.954 to 4.541]; I^2 78.247, P 0.000) increased significantly in the group of subjects exposed to urban air pollution compared with subjects less exposed.

As to the other parameters no statistically significant differences appeared in the comparison of the frequencies found in the two groups.

7. Discussion

The results of our meta-analysis allow to confirm the evidence of the effects of urban pollution on the respiratory system, although the mechanisms of action have been clarified only in part (Committee the Environmental and Occupational Health Assembly of the American Thoracic Society 1996).

The effects of pollutants on the respiratory system de-

pend on the type of pollutant, its concentration in the environment, on the duration of exposure and ventilatory capacity [11].

Our results in line with other studies in the scientific literature support the hypothesis that the reduction of some parameters of lung function such as FVC, FEV1 and PEFr found in the general population living in urban areas, or in areas with high pollution, could be due to exposure to urban pollutants and depict a framework of obstructive syndrome [12].

In one study [5], conducted in the USA, there was a significant association between lung function and levels of suspended particles in the non-smoking adults examined (NHANES I). The study showed that the increase of particulate was associated with a decrease of FVC: this association ceased approximately below $60 \mu\text{g}/\text{m}^3$ of particulate [10].

A reduction of respiratory capacity (FVC and FEV1) is also observed in the study of Pope III *et al.* 1993: in particular a decrease of 2% of FEV 1 for rise of PM10 of $100 \text{ g}/\text{m}^3$ in the subjects examined resulted statistically significant, all the subjects were smokers with mild or moderate chronic obstructive lung disease [13].

Another study [7] that must be remembered was made in Cracovia on 1414 persons who hadn't changed residence in the last 8 years: a faster decrease of respiratory function was observed in persons living in an areas with SO_2 and PM10 pollution compared people living in other areas.

An association between previous chronic exposition to high levels of air pollution and respiratory function was found in non-smokers living in 2 areas characterized by different levels of pollution [16,17].

In an Italian study among subjects with BPCO and asthma, the results of the analysis show that an increase of environmental concentrations of PM10 and PM2,5 causes a decrease of respiratory function during the following 24 h - 48 h [18-20].

A Dutch study shows a reverse correlation between FEV1 and nearness to a very busy freeway or between FEV1 and the number of heavy vehicles in a day [8].

The availability of controlled studies in the literature is rather small but the number of subjects studied is quite high. Many studies [22-24] show a disparity between the number of those who constitute the experimental group and the size of the control group with the resulting distortion of the results.

However, the processing of the data available, grouped by the three classes, allowed us to demonstrate an impairment of the respiratory system characterized by:

- 1) reduction in lung function (I^2 98.23);
- 2) increased prevalence of the class of symptoms (I^2 55.65);

3) increase in respiratory disease (I^2 51.31).

Among the class of respiratory symptoms, the results on the short breath/ breathlessness and wheezing were the most sensitive.

With an ES of 2.06 and 1.40 respectively, the meta-analysis indicates that within the population most exposed, shortness of breath/ breathlessness and wheezing were observed more frequently than in individuals belonging to the group of less exposed.

Studies concerning the variable shortness of breath/ breathlessness and wheezing, show a low homogeneity represented by their high value of I^2 (respectively 78.95 and 82.95) [25].

Among respiratory disease, variables with a high sensitivity that can be attributed to the effects of pollution include chronic bronchitis, emphysema with an ES of 2.25 and 2.97 respectively.

The heterogeneity among the studies which analysed for the presence of chronic bronchitis is 52.39 instead of 78.24 for the variable emphysema [9]. Among the statistically significant results related to lung function the parameters FVC, FEV1 and PEFr are all reduced, indicating obstructive deficit as chronic effect in the exposed population. The ES of the FVC and FEV1 PEFr is respectively -0.14 and -0.28 -0.32 while the value of I^2 is respectively 21.99, 79.27 and zero.

The meta-analysis identified the variables that can be used as indicators of the effects of pollution on the respiratory system. Variables with low heterogeneity and high sensitivity as the PEFr and bronchitis may be considered useful indicators of exposure to urban pollutants [26].

The variables with high sensitivity and high heterogeneity such as emphysema and shortness of breath/ breathlessness should be further explored in future studies to verify their usefulness as indicators of exposure to urban pollution.

8. Conclusions

In the light of the results is clear that the health of the population exposed to urban pollution must necessarily include a careful evaluation of the respiratory system through targeted surveys to the variables mentioned above with high sensitivity.

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