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Research on Optim ization of $Pm_{2.5}$ Exposure Risk Based on the Relationship between Exposure Risk and Age

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ABSTRACT. *Air pollution affects people's health and daily life. In previous studies, mean exposure response coefficient was often used to study the impact of air pollution on health. However, using the mean exposure response coefficient weakens the impact of air pollution on children and the elderly. This paper optimized the use of exposure response coefficient, taking Beijing from 2014 to 2018 as an example, used Integrated Exposure-response (IER) model and life table to estimate the number of premature deaths and years of potential life lost (YPLL), and compared the results before and after optimization. Results show that with the reduction of $PM_{2.5}$ concentration, the number of premature deaths of Beijing in 2018 decreased by about 2,000 compared with 2014, and YPLL decreased by 0.2 y. By comparing the results before and after optimization, it was found that previous studies had weakened the impact of $PM_{2.5}$ on human health. Therefore, it is necessary to distinguish ages in air pollution studies. When the question involves the population of all ages, the result of mean exposure response coefficient could be used as the lower limit of the research results.*

KEYWORDS: *Air pollution, Integrated Exposure-response model, YPLL, Premature death, Exposure risk*

1. Introduction

A serious haze occurred in China and lasted for 26 days in January 2013. Analyzing the published data from over 20 cities affected by the haze of China, direct economic loss exceeded 23 billion RMB [1]. Actually, haze episodes

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happened all over the world since the 1930s, causing huge social losses. Frequent occurrence of haze episode made people around the world aware of the seriousness of haze. China, even the world, is facing with the problem of haze governance. It not only brings about great damage, but also threatens the health of people.

Numerous studies have shown that air pollution seriously affects people's daily life. These studies are divided into two research areas: some focused on pathological study, analyzing the pathogenic situation of air pollution to human body from epidemiological perspective. They reported significant associations between particulate matter pollution and adverse health effects including caused respiratory disease [2], cardiovascular and cerebrovascular disease [3, 4], affected body's immune system [5], and even affected fertility, causing fetal deformity [6, 7]. The others focused on economic loss caused by particulate matter pollution. Such studies estimated economic loss in two main ways. One was the decline in people's health, fatigue, illness and death, resulting in a decline of labour [8]. The second is the increase in diseases caused by haze, leading to an increase in medical and prevention costs [9, 10, 11].

Almost all of the above studies used exposure response coefficient. Mean exposure response coefficient and endpoints of exposure response coefficient interval were used to study related issues. The problem with the mean exposure response coefficient is that it weakens the exposure risk of children and the elderly. They are the group most vulnerable to air pollution [12], this approach is obviously biased. The problem with the endpoints is that it extends the calculated range too much. However, it is impractical to study the exposure risks of air pollutants at different ages. Therefore, how to optimize the use of exposure response coefficient is the focus of this study.

The objectives of the study were 1) to make several reasonable assumptions to optimize the use of exposure response coefficient, 2) to take Beijing from 2014 to 2018 as an example to compare the results in various assumptions, 3) to estimate years of potential life lost (YPLL) in Beijing from 2014 to 2018, and quantify the effects of different $PM_{2.5}$ concentrations.

2. Materials and Methods

3.1 Materials

3.1.1 Pollutants

In this study, we choose $PM_{2.5}$ (Fine particulate matter with aerodynamic diameter <2.5 μm) as the representative pollutant. One reason is because $PM_{2.5}$ has the greatest impact on the human body compared to other air pollutants. $PM_{2.5}$ is relatively small in size and will enter the human lungs and blood. What's more, it is difficult to be discharged [13]. Secondly, there are many studies on $PM_{2.5}$ and

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research scope is relatively wide , which is convenient for us to compare and analyze results.

3.1.2 Study Area

In this paper, we chose Beijing, the capital of China, as the study area. It has a long history, reflecting China's economic level and development. The data required for Beijing is also relatively complete and easy to obtain. The situation of haze in Beijing is also one of the main reasons why the study choose it as the research city.We obtained Population and disease data of Beijing from Beijing Statistical Yearbook and Beijing Municipal Health and Population Health Status Report released by Beijing municipal government from 2015 to 2019. Data of $PM_{2.5}$ could be obtained from Beijing Municipal Ecological and Environmental Monitoring Center [14].

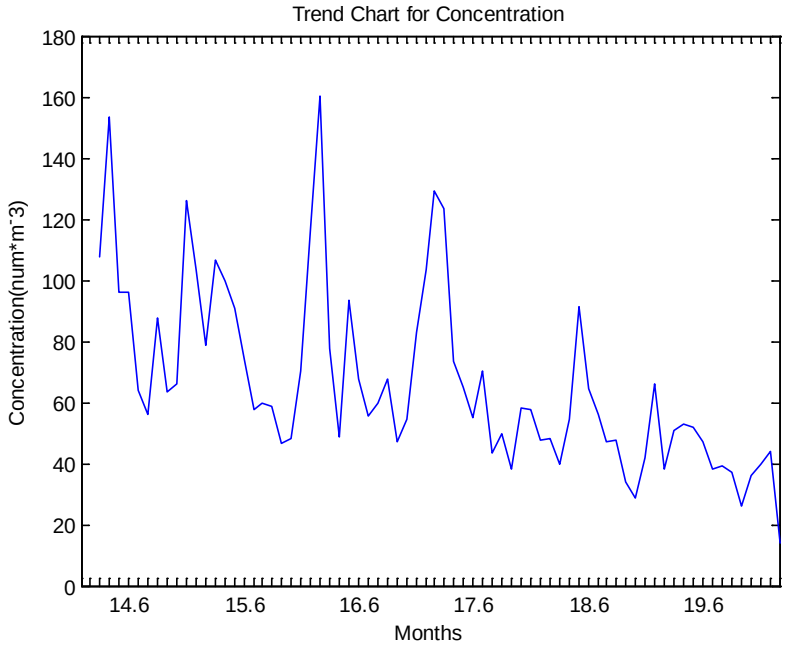

Fig.1 The Monthly Variation of $Pm_{2.5}$ Concentration in Beijing from 2014 to 2019. Blue Line is the Average Monthly Concentration of $Pm_{2.5}$ from 2014 to 2019.

Fig.1 showed the monthly variation of $PM_{2.5}$ concentration from January 2014 to September 2019. It could be clearly seen that $PM_{2.5}$ was maintained at a higher concentration from November of the previous year to January of the second year. In 2016, Beijing municipal government made a decision to deal with haze. From the

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

winter of 2017 to the beginning of 2018, it was obvious that peak concentration had decreased significantly compared with previous years. $PM_{2.5}$ concentration showed an overall downward trend, but still higher than international standards.

3.2 Methods

3.2.1 Integrated Exposure-Response (Ier) Model

Integrated Exposure-response (IER) model was proposed by Burnett et al., (2014) [15], which can describe several patterns of RR (Relative health risk). The model assumes when $PM_{2.5}$ falls below some concentration, there is no additional risk, and when $PM_{2.5}$ concentration is higher than the concentration, this model is presented as near-linear, sub-linear and other forms. The form of IER model is as follows:

$$RR_{p,r,s,y,m,\delta,g}(C) = \begin{cases} 1, C_{p,r,s,y} \le CO_z \\ 1 + CRF_{m,s,g}(C_{p,r,s,y} - CO_p), \\ \quad C_{p,r,s,y} > CO_p, linear \quad function \\ 1 + \lambda\left(1 - \exp\left(-\gamma(C_{p,r,s,y} - CO_p)^s\right)\right), \\ \quad C_{p,r,s,y} > CO_p, nonlinear \quad function \end{cases} \tag{1}$$

$$EP_{p,r,s,y,m,s,g} = \begin{cases} P_{r,y,m}\left(RR_{p,r,s,y,m,s,g}(C) - 1\right), for \quad linear \quad morbidity \quad function \\ P_{r,y,m} I_{r,^*allcause^*}\left(RR_{p,r,s,y,m,s,g}(C) - 1\right), for \quad linear \quad morbidity \quad function \\ P_{r,y,m}\hat{I}_{r,s}\left(RR_{p,r,s,y,m,s,g}(C) - 1\right), for \quad nonlinear \quad morbidity \quad function \\ function, where \quad \hat{I}_{r,s} = \dfrac{I_{r,\delta}}{RR_{r,\delta}} \end{cases} \tag{2}$$

Where, *EP* is defined as health terminal. C is defined as actual exposure concentration of $PM_{2.5}$, CO is defined as exposure concentration with assuming no additional risk. *CRF* is concentration effect equation. *P* is defined as exposed population. $\hat{I}$ is defined as disease incidence or mortality without the health risk caused by $PM_{2.5}$. I is defined as average annual disease incidence or mortality.

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

$I_{r,^{*}allcause^{*}}$ is defined as average annual natural mortality. α , γ and δ are mortality nonlinear equation parameters. The subscripts, *p, r, s, y, m, e, g* , represent pollutants ($PM_{2.5}$), region, scenario, year, health terminal classification (treatment rate or mortality), health terminal, value range (Medium, low and high).

In the study, all-cause mortality was taken as the health endpoint to evaluate YPLL. α , γ and δ were obtained from IHME (2016) [16], which provided 1000 sets of $(\alpha,\ \gamma,\ \delta)$ parameters. These 1000 joint parameters were given by Monte Carlo method for various diseases and specific age groups.

In previous studies, theoretical minimum exposure concentration of risk was $5.8\mu g/m^3$ to $8.8\mu g/m^3$ [17]. Thus, this study adopted recommendation of Apte et al., (2015) [18] to set the $PM_{2.5}$ threshold as $5.8\mu g/m^3$.

3.2.2 Estimation of Age-Specific Exposure Risk

In previous studies, YPLL caused by $PM_{2.5}$ was estimated using the mean exposure response coefficient. Numerous studies have shown that children and the elderly are more susceptible to air pollution than adults [19]. Using the mean exposure response coefficient to calculate weakens the impact of $PM_{2.5}$ on human life. Many previous studies have shown that the exposure risk of $PM_{2.5}$ is closely related to age. In other words, it is closely related to the state of human body.

The aging and degeneration of the human body is an inevitable natural law. The experimental report [120] published by Research Institute of the University of Virginia in journal, Neurboiology of Aging ,showed that when a person is 22 years old, his energy reaches its peak, and the next 5 years is a period of maintenance. After 27 years of age, all aspects of human body function began to decline.

Therefore, we made a assumption that the exposure risk of $PM_{2.5}$ and age are inversely related and decrease with age before the age of 20, reaching a minimum when a person is 20 years old. From age of 20 to 30, human body maintains its peak state, so exposure risk is minimal at this time. After the age of 30, exposure risk and age show a positive relationship, increasing with age.

We divided 0 years old and 1 to 4 years old into one group respectively, and the rest are divided into groups every 5 years old, dividing age into 19 groups in total. Then we hypothesized several kinds of relationships between exposure risk $PM_{2.5}$ and age:

(i)The exposure response coefficient of each group is regarded as a discrete point. It is assumed that these discrete points have a decreasing relationship before the age of 20 and an increasing relationship after the age of 30, and their tolerances are the same. The formula is as follows:

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

$$\gamma_i = \begin{cases} \gamma_0 + (6-i)d, i = 1,2,3,4,5 \\ \gamma_0, i = 6,7 \\ \gamma_0 + (i-7)d, 7 \le i \le 19 \end{cases} \tag{3}$$

γ_0 is the minimum exposure response coefficient, d is the tolerance of the sequence of the difference. Under this assumption, give the above formula a restriction:

$$\sum_{i=1}^{n} \gamma_i P_i = \gamma_c P \tag{4}$$

Where, γ_c is mean exposure response coefficient, P is total exposed population, P_i is the exposed population of each group. n is the total number of groups, where n=19.

(ii)We assume that there is a linear relationship between exposure risk and age, and is symmetric about t=25. The formula is as follows:

$$\gamma_t = \begin{cases} kt + a, 0 \le t \le 20 \\ \gamma_0, 20 < t \le 30 \\ -kt + b, 30 < t \end{cases} \tag{5}$$

t is defined as age, γ_t is the exposure response coefficient at age t. k is the slope, a and b are the intercept terms of two linear functions respectively. γ_i is equal to the mean of γ_t in each group, and treat Eq. (4) as conditional restriction.

(iii)Because there is a extreme point, we can assume that the relationship between exposure risk and age can be modeled by a quadratic polynomial, which is symmetric about t=25. The formula is as follows:

$$\gamma_t = \begin{cases} l((x-25)^2 + h), 0 \le t \le 20, 30 \le t \\ \gamma_0, 20 < t < 30 \end{cases} \tag{6}$$

l is the leading coefficient. The mean value of each group can be solved by mean value theorems for definite integrals.

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

3.2.3 Life Table

In this study, life table published in "GBD 2017" was used to calculate YPLL in all age groups. Life expectancy not only comprehensively reflects death level of each age group, but also illustrates the health level of population, which is one of main indicators to evaluate social health condition in different countries or regions. In this study, $\hat{I}$ of each age group obtained in Eq.(2) is brought into life table to calculate YPLL.

4. Results

4.1 Estimation of Age-Specific Exposure Risk

It could be seen from Table.1 that the estimated exposure risk range was large when the relationship was equal. Taking maximum exposure risk as the initial value, the range could reach 66.74%. In the first hypothesis, the value range of the minimum exposure risk is (2.32, 2.96) and ratio of the range to the original interval is (0, 57.08). When the minimum risk value is 2.96, the tolerance would be 0. Therefore, this current assumption also satisfied the situation that the age-specific exposure risks are all the mean exposure response coefficient. Under the second hypothesis, the maximum exposure response coefficient range is (2.96, 4.36) and ratio of the range to the original interval is (0, 66.74). In the third hypothesis, we took the slope as independent variable. The value range that satisfied restriction condition was (-0.031,0) and ratio of the range to the original interval is (0, 52.01). In the fourth hypothesis, taking the first coefficient as the independent variable, the value range that satisfied restriction condition was (0, 0.00038) and ratio of the range to the original interval is (0, 44.63). Therefore, four assumptions all satisfied the situation that the age-specific exposure risks are all the mean exposure response coefficient. The relational formula in Table.1 is just an example, which could best reflect the original interval range under four assumptions. The interval proportion of the hypothetical functional relationship also changed with the independent variables change. However, it is obvious that the impact of its exposure risk is also between the mean exposure response coefficient and the functional relationship in Table.1.

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

Table 1 Functional Relationship between Age and Exposure Risk

Index	Functional relationship between age and exposure risk			
Function classification	Arithmetic progression		Linear relationship	Quadratic function relationship
	Calculate from the lowest risk	Calculate from the maximum risk		
Function formula	$\gamma_i = \begin{cases} 3.34 - 0.17i, \\ 1 \le i \le 5 \\ 2.32, \\ i = 6,7 \\ 1.13+0.24i, \\ 7 \le i \le 19 \end{cases}$	$\gamma_i = \begin{cases} 3.16 - 0.20i, \\ 1 \le i \le 5 \\ 1.97, \\ i = 6,7 \\ 0.58 + 0.20i, \\ 7 \le i \le 19 \end{cases}$	$\gamma_t = \begin{cases} -0.031 \\ 0 \le t \le \\ 2.49, \\ 20 < t \le \\ 0.031t \\ 30 < t \end{cases}$	$\gamma_t = \begin{cases} 0.00038((x - 25 \\ 0 \le t \le 20, 30 \le \\ 2.74, \\ 20 < t < 30 \end{cases}$
Range of exposure risk	(2.32, 4.36)	(1.97, 4.36)	(2.49, 4.36)	(2.74, 4.36)
Percentage (%)	57.08	66.74	52.01	44.63

4.2 Number of Premature Death

Table 2 Number of Premature Death by Age under Five Hypothetical Situations in 2018

Age groups	Death toll under various hypothetical relationships				
	Arithmetic progression(lowest)	Arithmetic progression(maximum)	Linear relationship	Quadratic function relationship	Mean exposure response coefficient
0	6	5	6	5	5
1-4	2	2	2	2	2
5-9	1	1	1	1	1
10-14	1	1	1	1	1
15-19	1	1	1	1	1
20-24	2	2	2	2	3
25-29	7	6	7	8	9
30-34	12	10	12	13	14
35-39	12	11	12	13	14
40-44	17	15	17	17	17
45-49	33	31	34	32	33
50-54	86	81	86	82	81
55-59	74	71	74	70	66

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

60-64	153	147	152	142	130
65-69	168	163	166	156	136
70-74	160	156	157	148	124
75-79	188	185	185	176	139
80-84	347	345	340	329	247
85+	692	692	690	688	473
Total	1963	1926	1946	1886	1496

It could be seen from Table.2 that the results under four hypotheses were not much different, all around 1900. Compared with taking the mean exposure response coefficient, the gap had widened significantly. After the age of 60, the gap between mean exposure response coefficient and the other four hypotheses had gradually widened. Over 80 years of age, this gap was pulled to 220, final results were also about 400 different. Before the age of 60, the five results were almost the same. The number of premature deaths caused by $PM_{2.5}$ under the age of 20 accounted for about 1% of the total. Under four hypotheses, the number of people who died prematurely over 60 accounted for 87%, and the mean exposure response coefficient was about 83%. Maybe we couldn't tell which of these four hypotheses was more suitable to fit exposure risk of each age group, but we could definitely see the gap between estimated exposure response coefficient by age and directly used the mean exposure response coefficient. The results had shown that taking the mean exposure response coefficient could weaken the impact of $PM_{2.5}$ on the elderly and underestimates impact of $PM_{2.5}$ on health. In this way, when relevant researches on $PM_{2.5}$ was based on the population of all ages, the results obtained by applying the mean exposure response coefficient could be used as the lower limit of the desired interval.

As shown in Table.3, with the decrease of $PM_{2.5}$ concentration, the number of premature death showed a downward trend. Although the difference between four assumptions and the mean exposure response coefficient was decreasing year by year, the gap in the rate of decline had been increasing year by year. Between $80 \sim 90 \mu g/m^3$, for every drop of concentration, 18 deaths would be reduced under the four assumptions, and 16 deaths would be reduced under the mean exposure response coefficient. When the concentration was $40 \sim 80 \mu g/m^3$, 40~50 deaths would be reduced under the four assumptions, and 30~40 deaths would be reduced under the mean exposure response coefficient, the difference was about 10.

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

Table 3 the Number of Premature Deaths Caused by $Pm_{2.5}$ in Beijing from 2014 to 2018

Year	$PM_{2.5}$ concentration	Number of premature death under various hypothetical relationships				
		Arithmetic progression (lowest)	Arithmetic progression(maximum)	Linear relationship	Quadratic function relationship	Mean exposure response coefficient
2014	91.54	4171	4081	4132	3994	3254
2015	82.31	4002	3920	3965	3835	3098
2016	73.92	3561	3489	3529	3415	2749
2017	60.82	2971	2914	2945	2852	2279
2018	41.92	1963	1926	1946	1886	1496

4.3 Estimation of Ypll

As shown in Table.4, YPLL decreased with the decrease of $PM_{2.5}$ concentration. The results under four hypotheses were not much different. The difference was about 0.1 y between four assumptions and the mean exposure response coefficient. Between $80 \sim 90 \mu g/m^3$, for every drop of concentration, YPLL would be reduced by 0.05 y under the four assumptions, and 0.04 y would be reduced under the mean exposure response coefficient. When the concentration was $40 \sim 80 \mu g/m^3$, 0.0036~0.0038 y would be reduced under four assumptions, while taking the mean exposure response coefficient would reduce 0.0025 y, the difference was about 0.0012 y. Under the four assumptions, every 100,000 population would lose at least 27.18 thousand years for Beijing in 2014, which was a huge loss to society. Fortunately, the concentration of $PM_{2.5}$ in Beijing would be further reduced in 2019 with the increase in haze control, and YPLL would also drop below 0.1 y.

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

Table 4 Ypll Caused by $Pm_{2.5}$ in Beijing from 2014 to 2018

Year	Under actual conditions	Life loss under various hypothetical relationships				
		Arithmetic progression(lowest)	Arithmetic progression(maximum)	Linear relationship	Quadratic function relationship	Mean exposure response coefficient
2014	81.81	0.3415	0.3324	0.3388	0.3276	0.2718
2015	81.95	0.2943	0.2864	0.2919	0.2820	0.2340
2016	82.03	0.2623	0.2554	0.2602	0.2514	0.2080
2017	82.15	0.2110	0.2055	0.2093	0.2022	0.1667
2018	82.30	0.1343	0.1309	0.1333	0.1287	0.1059

5. Discussion

The related issues of air pollution have been a hot research topic in recent years. However, many problems choose to use the mean exposure response coefficient when it involves population. The risk of human exposure to pollutants varies with the human body. The weaker the body, the higher the risk of exposure. This study gave several ways to reduce the error range. Many studies have shown that children under the age of 14 and the elderly over 65 are the most vulnerable to $PM_{2.5}$ [21, 22], and their exposure risk is relatively high. However, people's physical condition changes gradually with age. Therefore, it is necessary to study the exposure response coefficients of age groups.

However, it is impractical to estimate the exposure response coefficient of various age groups. At first, it is difficult to find suitable research data. The study of this data requires a large number of experimental subjects, covering all age groups, and needs to track their physical conditions in air pollution, which is unethical. Secondly, the diversity of pollutants. Air pollution is the fusion of various substances harmful to the human body, and there are also complex chemical reactions such as reversible reactions [23]. Thirdly, the uncontrollability of the human body state. The physical state of a person is closely related to personal living habits, which varies from person to person. Therefore, there is no model has been developed that can simulate the change of a person's physical condition with age.

The experimental report [19] provided important theoretical basis for several hypotheses of this paper. And we gave a certain restriction to simulate the relationship between exposure risk and age. This study made four hypotheses and compared them with the mean exposure response coefficient. The fitting relationship under four hypotheses could also satisfy the situation that the exposure risk coefficients of age groups are all the mean exposure response coefficient. Under the assumptions, the exposure risk of 0-year-old children is similar to that of the 50~54 age group. Strosnider et al., (2018) [12] pointed out that the exposure risk of children under 14 was lower than that of people over 65,

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

and is higher than the average level of 14~65. Therefore, this assumption is also reasonable.

If the relationship between $PM_{2.5}$ exposure risk and age is similar to the functional relationship under the four hypotheses, then the quantitative results are in the interval with the functional relationship in Table.1 as the upper limit and the mean exposure response coefficient as the lower limit. Even though the function relationship between the exposure risk of $PM_{2.5}$ and age is far more complicated than this study, results, using the mean exposure response coefficient, should also be the lower limit.

In the empirical analysis part, the research results shown that the number of premature deaths caused by $PM_{2.5}$ in Beijing has decreased by about 2,000 during 2014 to 2018 under hypothetical conditions, and 1,700 under the mean exposure response coefficient. YPLL is reduced by about 0.2 y under hypothetical conditions, and 0.16 y under the mean exposure response coefficient. Chen et al., (2014) [24] studied YPLL of 74 cities in China caused by $PM_{2.5}$ in 2013. The mean exposure response coefficient were used for the study, showing that the lifespan of urban residents increased by 0.0024 y for every decrease in $PM_{2.5}$ concentration. The study of Ma et al., (2019) [25] showed that YPLL of Chinese urban residents in 2017 decreased by 0.16 y compared with 2013. These were consistent with the conclusion of this study.

The ideas of this study can also be extended to other studies on air pollutants similar to $PM_{2.5}$ and their health effects. However, there are also many shortcomings of this article. For example, the selected fitting function relationships are too simple, and the restriction function has a lot of room for improvement. In the next step, the complexity of the fitting function can be increased, and the research can be conducted in combination with real data.

6. Conclusions

In this paper, we set up four assumptions. Taking Beijing from 2014 to 2018 as an example, we compared health effects of $PM_{2.5}$ under four assumptions and the mean exposure response coefficient. Results had shown that when doing researches on air pollution, it is necessary to study exposure risks of all age groups. When the question involves the population of all ages, the result of mean exposure response coefficient could be used as the lower limit of the research results. $PM_{2.5}$ does affect people's health. We hope that people will pay attention to it and protect themselves.

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ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

References

[1] Mu, Q., Zhang, S (2013). An evaluation of the human capital loss due to the heavy haze during January 2013 in China. J. Environ. Sci-C, no.33, pp.2087-2094.

[2] Katanoda, K., Sobue, T., Satoh, H., et al (2011). An Association between Long-Term Exposure to Ambient Air Pollution and Mortality from Lung Cancer and Respiratory Diseases in Japan. J. Epidemiol, no.21, pp.132-143.

[3] Stafoggia, M., Cesaroni, G., Peters, A., et al (2014). Long-Term Exposure to Ambient Air Pollution and Incidence of Cerebrovascular Events: Results from 11 European Cohorts within the ESCAPE Project. Environ. Health. Persp, no.125, pp.919.

[4] Brook, R.D., Rajagopalan, S., Pope, C.A., et al (2010). Particulate Matter Air Pollution and Cardiovascular Disease: An Update to the Scientific Statement from the American Heart Association. Circulation, no.121, pp.2331-2378.

[5] Ashley-Martin, J., Lavigne, E., Arbuckle, T., et al (2016). Air pollution during pregnancy and cord blood immune system biomarkers. J. Occup. Environ. Men, no.58, pp.979-986.

[6] Maji, K., Ye, W., Arora, M., et al (2018). $PM_{2.5}$-related health and economic loss assessment for 338 Chinese cities. J. Environ, no.121, pp.392-403.

[7] Liu, S., Song, G (2016). Evaluation of $PM_{2.5}$'s adverse human health effect in cities. J. Environ. Sci-C, no.36, pp.343-351.

[8] Liu, S., Jia, Z., Song, G (2016). Application of Human Capital Law in Assessment of the Loss Value of Life and Health in Air Pollution. Environ. Prot. Sci, no.42, pp.48-52.

[9] Strosnider, H.M., Chang H.H., Darrow L.A (2018). Age-Specific Associations of Ozone and $PM_{2.5}$ with Respiratory Emergency Department Visits in the US. AM. J. RESP. CRIT. CARE, pp.199.

[10] Qiao, L., Cai, J., Wang, H., et al. 2014. $PM_{2.5}$ Constituents and Hospital Emergency-Room Visits in Shanghai, China. Environ. Sci. Technol, vol. 48, no.17, pp.10406-10414.

[11] Burnett, R.T., Pope, C.A., Ezzati, M., et al. 2014. An Integrated Risk Function for Estimating the Global Burden of Disease Attributable to Ambient Fine Particulate Matter Exposure. Environ. Health. Persp, no.122, pp.397-403.

[12] Lim, SS., Vos, T., Flaxman, A.D., et al. 2012. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet Gastroenterol Hepatol, no.380, pp.2224-2260.

[13] Apte, J.S., Marshall, J.D., Cohen, A.J., et al. 2015. Addressing Global Mortality from Ambient $PM_{2.5}$. Eviron. Sci. Techonl, no.49, pp.8057-8066.

[14] Hwang, J.S., Hu, T.H. 2020. Later-Life Exposure to Moderate $PM_{2.5}$ Air Pollution and Life Loss of Older Adults in Taiwan. Int. J. Environ. Res. Public Health, vol.17, no.6, pp.1873.

[15] Salthouse, T.A (2009). When does age-related cognitive decline begin? Neurobiology of Aging, vol.30, no.4, pp.507-514.

ISSN 2616-7433 Vol. 2, Issue 17: 23-36, DOI: 10.25236/FSST.2020.021704

[16] Cai, J., Peng, C., Yu, S., et al (2019). Association between $PM_{2.5}$ Exposure and All-Cause, Non-Accidental, Accidental, Different Respiratory Diseases, Sex and Age Mortality in Shenzhen, China. Int. J. Environ. Res. Public Health, vol.16, no.3, pp.401.
[17] Shimohara, T., Oishi, O., Utsunomiya, A., et al (2001). Characterization of atmospheric air pollutants at two sites in northern Kyushu, Japan-chemical form, and chemical reaction. Atmospheric Environ, vol.35, no.4, pp.667-681.