Correlation between fine particles in the environmental air and causes of diseases among inhabitants.

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ABSTRACT: It has been reported that fine particular matters suspended in the environmental air may cause cardiopulmonary disease including lung cancer, and may increase the daily death rate among the inhabitants of the area. To clarify what kinds of diseases, not a group of diseases, can correlate to fine particle concentration in the air, the Vital Statistics and the air pollution estimates of 1881 points in Japan were used for the present study. As limited data of $PM_{2.5}$ (particulate matter less than 2.5 micron-meter in diameter) were available, the converted $PM_{2.5}$ from PM_{10} (0.7 x PM_{10}) values were used in accordance with the results of 25 investigations in Japan. Among various causes-of-death, a significant correlation was observed between the presumed $PM_{2.5}$ levels in the district and from the age-adjusted death rates from ischemic heart disease and hypertensive heart disease in both genders. Only females showed significant correlations with pneumonia, chronic obstructive lung disease, asthma, lung cancer and surprisingly, breast cancer, endometrial cancer and ovarian cancer. For evaluation of these data, confounding factors such as tobacco smoking, hormone-relating phenomena and population density were considered and examined by multivariable regression analysis. Urban life-associated factors could be contributed to some extent, however, $PM_{2.5}$ level remained to be significant in any analysis even in female cancers noted above. Esterogenic activities of suspended particulate matter in the air were considered as a possible reason for the data.

INTRODUCTION: Effects of long-time exposure of polluted air to the inhabitants have been studied in large- scale epidemiological studies carried out in U.S.A, indicating the increased risks of cardiopulmonary, lung cancer and total deaths(1,2) with shortening of life span of the inhabitants in the polluted areas(3). These hazardous effects are considered due to mainly fine particulate matters but partly sulfates, which are thought to originate largely from fossil fuel combustion, and diesel engine exhaust may account for a portion of these fine particles. Particles less than 2.5 micron meter in diameter ($PM_{2.5}$) are the consequential constituent, because of penetrability deep to the respiratory organ and of mostly man-made particles. And, ultrafine particles of less than 100 nm in diameter can be absorbed directly through the alveolar walls to the lymphatic or blood vessels (4), thus may give some unknown hazardous effects to the afferent organs than the lung.

To confirm the previously reported increase of cardiopulmonary and lung cancer deaths in the Japanese population, and to make it more precisely, not a group of diseases but at disease level, we conducted an analysis of the effects of fine particles in health, using the nation-wide, age-adjusted cause-of death statictics as well as air pollution data covering all Japan, and studied a correlation between mortality of each disease in ICD-10 to the air pollution level in each district of Japan. Similarly to the previous reports, cardiopulmonary diseases and lung cancer were revealed to have statistically significant correlation to the fine particle level, and unexpectedly, significant correlations were observed also with breast, endometrium and ovarian carcinomas.

MATERIALS AND METHODS:

Air pollutant estimates were obtained from the All Japan Air Pollution Data Book, 2000, Ministry of Environment, which is open to public. Annual mean concentration of suspended particulate matter (PM_{10}), nitrogen dioxide, sulfur dioxide and oxidant, estimated at 1553 residential/commercial areas and 320 road-side spots, were used. The annual mean of each pollutant in each of 47 prefectures and 13 large citied, in total, 60 districts of Japan, were calculated from the data book. Because the standard estimates of suspended particulate matter in Japan at present is PM_{10}, a conversion of PM_{10} to $PM_{2.5}$ value was necessary and reviewing and summarizing the results of 25 studies on the ratio of $PM_{2.5}$ to PM_{10} were made. Mean value of 0.7 was tentatively used for this conversion rate.

Statistics of age-adjusted causes-of-death (ICD-10) by 47 prefectures and 13 large cities (60 districts) were obtained from the Vital Statistics in Japan, 2000, Volume 3, and statistics of the age of marriage, first delivery and number of children delivered were obtained also from another volume of the same book. Smoking rates by age, gender and 60 districts were given from the data book, the Nation's Basic Life Survey, Ministry of Health, Labor and Welfare. Population density in the each districts were obtained from the Population Census, Japan. 2002.

Statistics; Poison's regression model was used for estimation of equation and slope of regression line, correlation coefficiency and p-value in uni- and multi-variable regression analysis. For calculation, statistics software (StatMate, ATMS Co Ltd, Tokyo and SPSS in Japanese edition) were used.

RESULTS:

1. Annual mean level of each air pollutant and correlation of each pollutant: Calculated $PM_{2.5}$ level in each district ranged from 11.6 mcg/m^3 to 32.1 mcg/m^3, and the annual mean value for the whole country showed 20.8 mcg/m^3 that is exceeding from the US Ambient Air Control Standards of 15 mcg/m^3. A map demonstrating the distribution of $PM_{2.5}$ is shown in Figure 1, demonstrating its markedly high level at the economical back born belt area of Japan, especially in and around a large city like Tokyo, Nagoya and Osaka, in parallel with population density. Correlation between PM2.5 and three gaseous constituents was shown in table 1, demonstrating a close correlation between PM2.5 and NO2, next SO2 but an inverse correlation with oxidant.

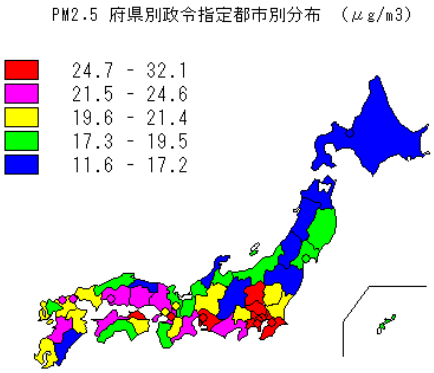
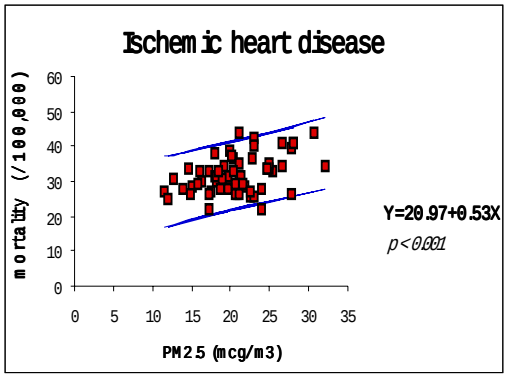


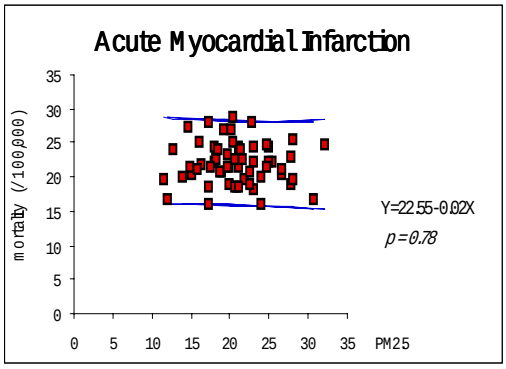
Table 1. Correlation coefficiency between the four pollutants

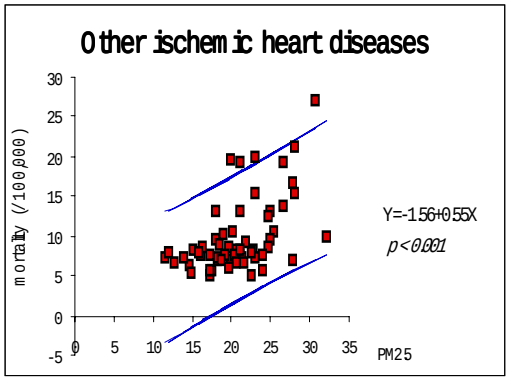
	$PM_{2.5}$	NO_2	SO_2	Oxidant
$PM_{2.5}$ mcg/m^3	1.0	0.755	0.606	-0.391
NO_2 ppm	0.755	1.0	0.571	-0.505
SO_2 ppm	0.606	0.571	1.0	-0.487
Oxidant ppm	-0.391	-0.505	–0.487	1.0

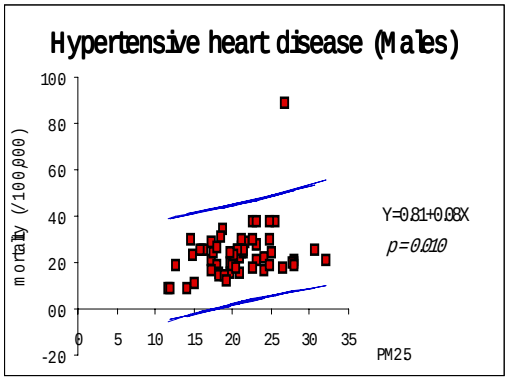
2. Correlation between $PM_{2.5}$ level in each district and the age-adjusted mortality of diseases.

Age-adjusted mortality in each district correlated to some of the cardiovascular diseases as seen in Figure 2 to 5. There was a positive correlation beween ischemic heart disease in each gender and PM2.5 levels in each district, with r=0.41 and r=0.459, p=0.0011 and p=0.0002 in males and females, respectively. However, when acute myocardial infarction (ICD-10: I-21) was separated from other ischemic heart diseases (ICD-10: I-20, I-22, I-24, I-25), then acute myocardial infarction and angina pectoris (ICD-10: I-20) did not show any correlation with level $PM_{2.5}$ but "other ichemic heart diseases", majority of which are chronic ischemic heart disease (ICD-10: I-25), showed a significant correlation to $PM_{2.5}$. And, hypertensive heart disease showed correlation to $PM_{2.5}$ level, emerging the possibility that atherosclerotic changees may be an underlying pathological condition of these cardiovascular illness. It is recently reported in an experimental study using hreditable hyperlipidemic rabbits that exposure of PM_{10} for 4 weeks have induced systemic inflammatory response with progression of the hisologically confirmed atherosclerotic changes in the coronary artery in relation with a number of particle laden alveolar macrophages (4). No further analytical study can not be done on the obtained data. No correlation was noted in arrythmia , intracranial bleeding or pulmonary infarction with PM2.5 levels.









Respiratory diseases in male did not show any correlation to PM2,5 levels, however, statistically significant correlation was note in females for pneumonia ($p<0.001$), chronic obstructive pulmonary diseases (COPD, $p<0.05$) and asthma ($p<0.01$). Lung cancer of females also showed a significant correlation with PM2.5 only in females($p<0.05$)

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