CM Wong 黃浙明
CQ Ou 歐春泉
TQ Thach 石國順
KP Chan 陳敬斌
YK Chau 周婉君
RY Chung 鍾一諾
TH Lam 林大慶
AJ Hedley 賀達理

Lifestyle-modified mortality associated with air pollution: a time-series study

Key Messages

1. Smoking increases the hazards of air pollution while moderate exercise protects against the latter's adverse effects. Comprehensive and coherent public health policies aimed at the reduction of avoidable air pollution–associated mortality should target both environmental air quality and tobacco use.
2. Frequent consumption of fresh vegetables and fruits should be encouraged in the elderly.
3. Healthy lifestyle is an important consideration for public health policies on environmental health.
4. Further research on the roles of effect modification to the hazards of air pollution is necessary to clarify the hypotheses for the mechanism by which air pollution impacts on health.

Hong Kong Med J 2008;14(Suppl 3):S13-6

Department of Community Medicine, 5/F, William MW Mong Block, Faculty of Medicine Building, The University of Hong Kong, 21 Sassoon Road, Hong Kong SAR, China
CM Wong, CQ Ou, TQ Thach, KP Chan, YK Chau, RY Chung, TH Lam, AJ Hedley

HCPF project number: 215032

Principal applicant and corresponding author:
Dr CM Wong
School of Public Health and Department of Community Medicine, 5/F, William MW Mong Block, Faculty of Medicine Building, The University of Hong Kong, 21 Sassoon Road, Hong Kong SAR, China
Tel: (852) 2819 9109
Fax: (852) 2855 9528
E-mail: hrmrwcm@hkucc.hku.hk

Introduction

Air pollution is a major public health problem that commonly accompanies economic development. In setting the relevant policy, health effects are usually based on estimates from daily time-series studies. In estimating short-term effects of air pollution, we have (1) utilised histories of individual dead person's personal factors in assessing whether there is any interaction between effects of lifestyle and air pollution; and (2) identified lifestyle subgroups which may be more vulnerable to air pollution than the others. Thus, most limitations of ecological studies have been taken into account.

Methods

This study was conducted from February 2003 to July 2005.

Data

This study included 24 656 ethnic Chinese subjects, with male-to-female ratio of 1.25:1, who died in 1998 at the age of 30 years or more. Lifestyle factors (smoking status, physical exercise, alcohol drinking, and diet 10 years before death) and demographic factors for each decedent were obtained by interviewing the informants (ie relatives) in all four death registries in Hong Kong. Daily concentrations of air pollutants, including nitrogen dioxide (NO_2), ozone (O_3), suspended particulate matter (PM_{10}), and sulfur dioxide (SO_2), were provided by the Environmental Protection Department. Daily mean temperature and relative humidity were obtained from the Hong Kong Observatory.

Statistical methods

The data were stratified into subgroups for each lifestyle variable (ie smoking, physical exercise, and alcohol consumption). In order to develop the core model, separate Poisson regression models for daily counts of death were fitted for each stratum to estimate the effects of air pollution in each subgroup of lifestyle factors after adjustments for seasonality, meteorological variables, day of the week, holidays, and influenza epidemics. The effect of air pollutant was measured by excess risk (ER) of mortality per 10 $\mu g/m^3$ increase of a pollutant. Then, in order to estimate the difference in ER between two lifestyle subgroups, two series for each lifestyle factor were fitted simultaneously, using multi-series Poisson regression with conclusion of the interaction between air pollution and the indicated lifestyle factor.

Factor analysis with principal component method was applied to derive principal dietary factors on the basis of consumption frequency of six food groups. Factor scores for each individual were computed using regression methods. Conditional logistic regression with case-crossover design was performed to evaluate the interaction between the factor score and air pollution on mortality.

Results

In all ages, among the 24 656 subjects who died from all natural causes (International Classification of Diseases version 9 [ICD9] codes 1-799), 11 627 (47%) did so from cardiorespiratory diseases (ICD9 codes 390-519). In the age-group of 65+, of the 19 392 who died from all natural causes, 10 242 (53%) had a cardiorespiratory cause.

Table 1. Descriptive statistics of deaths by lifestyle factors

Description	No. (%)	
	Female (n=13 631)	Male (n=11 025)
Smoking		
Daily smoking	1438 (13)	6901 (51)
Occasionally smoking	102 (1)	175 (1)
Ex-smoking	804 (7)	2340 (17)
Never smoking	8665 (79)	4182 (31)
Exercise		
None	6596 (61)	8462 (64)
<1/month	577 (5)	779 (6)
1-3/month	575 (5)	729 (6)
1-3/week	711 (7)	837 (6)
≥4/week	2334 (22)	2453 (18)
Drinking		
Ex-drinking	291 (3)	1455 (11)
Never drinking	9681 (88)	6561 (48)
Occasionally drinking (<1/week)	578 (5)	1586 (12)
Weekly drinking (≥1/week)	431 (4)	3899 (29)

Among all the subjects, there were more daily smokers and drinkers among males (51% and 29% respectively) than females (13% and 4% respectively), while the number of people exercising was similar for both sexes. Males tended to consume meat more often while females consumed fruits, vegetables, dairy, and bean food items more often (data not shown). Since the vast majority of the smokers and drinkers were males (>80%), only males were included in the analysis of smoking and drinking (Table 1).

Smoking

Compared with smokers, never-smokers were more likely to be older, locally born, better educated, and living in self-owned housing (P<0.001) [data not shown]. The ER associated with PM_{10} exposure in smokers was significant (P<0.05) for all natural causes and cardiorespiratory diseases in the range of 1.8 to 3.2% per 10 μg/m^3 increase in pollutant concentration (Table 2), but we did not observe significant effects of PM_{10} in never-smokers. The differences between smokers and never-smokers in ER from all causes were significant (P<0.05), but the differences from cardiorespiratory diseases did not attain significance (0.05<P<0.1).

Exercise

The subjects who exercised were more likely to be female, more highly educated, living longer, or having a better health status before death (P<0.001) [data not shown]. In the exercising subjects, in general air pollution was not significantly associated with mortality (P>0.05), except for SO_2 in the age-group of ≥30 years. In those who never exercised, prevailing air concentrations of NO_2, PM_{10}, and O_3 were significantly associated with mortality from all natural causes (P<0.05) [Table 3]. There were significant differences (P<0.05) between those who exercised and never exercised, in terms of ER of mortality associated with NO_2 and PM_{10} regardless of age-group and cause of death.

Drinking

Never-drinkers in this sample had died at older age and were more likely to be better educated, living in better housing, retired, and less likely to be daily smokers than weekly drinkers (P<0.001) [data not shown]. In drinkers, the air pollution–associated ERs of mortality were consistently higher from cardiorespiratory diseases than from all natural causes irrespective of age and all pollutants under study (Table 4). Sulphur dioxide showed the highest association with mortality with ERs in the range of 3.6 to 9.9% (P<0.05). The differences in ERs were higher in drinkers than in never-drinkers, but statistically significant only for cardiorespiratory diseases associated with SO_2.

Diet

Three dietary factors were derived with each one loading dominantly on two food groups. Cumulatively, they accounted for 70% of total variance in food consumption frequency. The three factors were: meat/fish, vegetable/fruit, and bean/dairy (Table 5).

There were no significant interactions between the meat/fish factor and air pollution. For the vegetables/fruit factor, all the interactions were negative, and were significant

Table 2. Excess risk of mortality per 10 μg/m^3 increase in pollutants at best lag days for male smokers and never-smokers*

	Excess risk % (95% confidence interval)			
	≥30 years old		≥65 years old	
	Never-smokers	Smokers	Never-smokers	Smokers
All natural causes				
NO_2	1.6 (-0.7 to 4.0)	0.6 (-1.4 to 2.5)	2.4 (-0.3 to 5.2)	1.5 (-1.0 to 4.1)
SO_2	2.0 (-1.8 to 6.0)	-2.3 (-5.6 to 1.1)	3.0 (-1.4 to 7.6)	2.4 (-1.5 to 6.5)
PM_{10}	-0.7 (-2.5 to 1.1)	1.8† (0.4 to 3.3)	-1.1 (-3.1 to 1.0)	3.2† (1.4 to 5.1)
O_3	1.3 (-0.8 to 3.4)	1.4 (-0.4 to 3.1)	0.5 (-1.8 to 2.9)	1.9 (-0.3 to 4.2)
Cardiorespiratory causes				
NO_2	2.4 (-1.1 to 5.9)	-0.8 (-3.7 to 2.2)	3.0 (-0.6 to 6.7)	1.2 (-2.2 to 4.8)
SO_2	4.0 (-2.2 to 10.6)	1.8 (-3.0 to 6.8)	6.8† (0.3 to 13.8)	3.6 (-1.7 to 9.2)
PM_{10}	-0.8 (-3.3 to 1.8)	2.3† (0.2 to 4.5)	-0.8 (-3.4 to 1.9)	3.0† (0.5 to 5.6)
O_3	-0.8 (-3.7 to 2.3)	0.7 (-2.0 to 3.6)	-1.1 (-4.1 to 2.1)	1.6 (-1.5 to 4.8)

* NO_2 denotes nitrogen dioxide, SO_2 sulfur dioxide, PM_{10} suspended particulate matter, and O_3 ozone; best lag days = the lag day when the P value associated with the effect estimate of air pollution was the smallest

† P<0.05

Table 3. Excess risk of mortality per 10 μg/m³ increase in pollutants at best lag days for exercise and never-exercise groups*

	Excess risk % (95% confidence interval)			
	≥30 years old		≥65 years old	
	Never exercise	Exercise	Never exercise	Exercise
All natural causes				
NO_2	1.2 (-0.1 to 2.4)	0.3 (-1.5 to 2.1)	1.5† (0.1 to 2.8)	0.5 (-1.4 to 2.5)
SO_2	0.9 (-1.3 to 3.1)	3.0† (0.1 to 5.9)	1.7 (-0.8 to 4.2)	2.7 (-0.5 to 5.9)
PM_{10}	1.0† (0.1 to 2.0)	0.1 (-1.2 to 1.4)	1.3† (0.3 to 2.3)	0.2 (-1.2 to 1.7)
O_3	1.6† (0.5 to 2.6)	-0.7 (-2.0 to 0.6)	1.4† (0.2 to 2.6)	-0.2 (-1.6 to 1.2)
Cardiorespiratory causes				
NO_2	0.9 (-1.0 to 2.9)	0.6 (-1.9 to 3.2)	1.4 (-0.7 to 3.5)	0.4 (-2.2 to 3.1)
SO_2	1.6 (-1.7 to 4.9)	0.7 (-3.5 to 5.1)	2.9 (-0.6 to 6.6)	0.6 (-3.7 to 5.1)
PM_{10}	1.0 (-0.4 to 2.3)	0.5 (-1.4 to 2.4)	1.0 (-0.5 to 2.4)	0.3 (-1.7 to 2.3)
O_3	1.4 (-0.1 to 2.9)	-0.8 (-2.7 to 1.1)	1.6† (0.1 to 3.2)	-0.8 (-2.8 to 1.2)

* NO_2 denotes nitrogen dioxide, SO_2 sulfur dioxide, PM_{10} suspended particulate matter, and O_3 ozone; best lag days = the lag day when the P value associated with the effect estimate of air pollution was the smallest

† P<0.05

Table 4. Excess risk of mortality per 10 μg/m³ increase in pollutants at best lag days for weekly drinkers and never-drinkers*

	Excess risk % (95% confidence interval)			
	≥30 years old		≥65 years old	
	Never-drinkers	Drinkers	Never-drinkers	Drinkers
All natural causes				
NO_2	0.8 (-1.1 to 2.8)	-0.5 (-2.8 to 1.9)	1.5 (-0.6 to 3.7)	1.1 (-2.1 to 4.4)
SO_2	-0.9 (-4.1 to 2.5)	3.6 (-0.4 to 7.8)	1.1 (-2.6 to 5.0)	5.8† (0.6 to 11.3)
PM_{10}	1.2 (-0.3 to 2.6)	-1.2 (-3.0 to 0.6)	1.8† (0.1 to 3.5)	-1.9 (-4.2 to 0.4)
O_3	1.9† (0.3 to 3.5)	1.2 (-0.5 to 2.9)	2.5† (0.6 to 4.4)	1.9 (-0.4 to 4.2)
Cardiorespiratory causes				
NO_2	1.5 (-1.1 to 4.2)	1.3 (-2.5 to 5.4)	2.3 (-0.6 to 5.3)	3.3 (-1.3 to 8.2)
SO_2	-2.8 (-6.9 to 1.5)	7.4† (0.8 to 14.5)	-3.3 (-7.8 to 1.5)	9.9† (2.5 to 17.9)
PM_{10}	-1.2 (-3.1 to 0.7)	2.7 (-0.2 to 5.5)	1.6 (-0.5 to 3.8)	2.4 (-1.0 to 6.0)
O_3	1.4 (-1.0 to 3.7)	3.2† (0.3 to 6.1)	1.7 (-0.9 to 4.2)	2.8 (-0.4 to 6.2)

* NO_2 denotes nitrogen dioxide, SO_2 sulfur dioxide, PM_{10} suspended particulate matter, and O_3 ozone; best lag days = the lag day when the P value associated with the effect estimate of air pollution was the smallest

† P<0.05

(P<0.05) for all natural causes of death in the 30+ age-group for all four air pollutants, as well as on both health outcomes for PM_{10} (Table 6). For the bean/dairy factor, significant negative (SO_2) and positive (PM_{10}) interactions were noted.

Table 5. Factor loadings and variance explained by each constituent of the three principal dietary factors

Diet	Factor		
	1	2	3
Meat	**0.875**	0.063	-0.007
Fish	**0.862**	0.129	0.026
Vegetables	0.164	**0.820**	0.018
Fruit	0.033	**0.789**	0.175
Dairy	0.020	-0.041	**0.855**
Bean	0.003	0.253	**0.684**
Variance explained	32%	22%	16%

Discussion

Increased effects of air pollution in smokers

We found significant effects of PM_{10} in smokers but not in never-smokers. The biological mechanism was most likely related to the damage induced by smoking to the mucociliary system, as well as endothelial and cardiovascular functions. As a result, the uptake of particulate pollutants into arterial walls of smokers is increased, and atherogenesis accelerates.[1] Body defence in terms of phagocytosis may also be inhibited by the damage, leading to stronger effects of air pollution in smokers than never-smokers.[2]

Protective effects of exercising against hazards of air pollution

We found significant effect modification of air pollution by exercise levels; the effect being higher in those who never exercised than in those who did. This provided the first evidence that moderate exercise is protective against the adverse effect of air pollution, particularly in the population aged older than 65 years. The biological mechanism may be related to the individuals' susceptibility to hazards of air pollution and capability in clearing pollutants from the body. Those who never exercised appeared to have relatively worse health status and lower reserve capacity. Conversely, those who exercised appeared to have improved pulmonary and immune function, resulting in more active removal of inhaled air pollutants.[3]

Table 6. Interaction between air pollution and dietary factor scores at best lag days*

	Excess risk % (95% confidence interval)			
	All natural causes		Cardiorespiratory causes	
	≥30 years old	≥65 years old	≥30 years old	≥65 years old
Meat/fish				
NO_2	0.9 (-0.1 to 2.0)	1.1 (0.0 to 2.2)	0.6 (-0.8 to 2.1)	0.6 (-0.8 to 2.1)
SO_2	-1.5 (-3.2 to 0.3)	1.1 (-0.9 to 3.2)	-1.9 (-4.4 to 0.6)	-1.9 (-4.4 to 0.8)
PM_{10}	-0.7 (-1.4 to 0.1)	-0.1 (-0.9 to 0.7)	-0.6 (-1.6 to 0.4)	-0.5 (-1.6 to 0.5)
O_3	0.6 (-0.2 to 1.5)	0.7 (-0.2 to 1.7)	0.8 (-0.4 to 2.1)	1.0 (-0.3 to 2.3)
Vegetables/fruit				
NO_2	-1.0† (-2.0 to 0.0)	-1.1 (-2.3 to 0.0)	-0.8 (-2.3 to 0.7)	-1.1 (-2.6 to 0.5)
SO_2	-1.9† (-3.6 to -0.2)	-2.4† (-4.3 to -0.4)	-1.2 (-3.7 to 1.3)	-2.0 (-4.7 to 0.7)
PM_{10}	-1.0† (-1.7 to -0.3)	-1.0† (-1.8 to -0.1)	-1.4† (-2.5 to -0.3)	-1.5† (-2.7 to -0.4)
O_3	-1.0† (-1.8 to -0.1)	-0.6 (-1.5 to 0.3)	-0.9 (-2.1 to 0.3)	-0.5 (-1.9 to 0.8)
Bean/dairy product				
NO_2	0.8 (-0.2 to 1.9)	0.5 (-0.6 to 1.7)	1.8 (0.3 to 3.3)	1.5 (-0.1 to 3.1)
SO_2	-2.0† (-3.7 to -0.3)	-2.2† (-4.2 to -0.3)	-2.4 (-4.9 to 0.1)	-3.0† (-5.6 to -0.3)
PM_{10}	0.8† (0.0 to 1.5)	0.8† (0.0 to 1.6)	0.8 (-0.2 to 1.9)	1.0 (-0.1 to 2.1)
O_3	0.7 (-0.2 to 1.5)	0.8 (-0.1 to 1.8)	1.0 (-0.2 to 2.2)	0.8 (-0.4 to 2.1)

* NO_2 denotes nitrogen dioxide, SO_2 sulfur dioxide, PM_{10} suspended particulate matter, and O_3 ozone; best lag days = the lag day when the P value associated with the effect estimate of air pollution was the smallest

† P<0.05

Apparent effects of alcohol drinking on hazards of air pollution

There is no strong evidence from the literature as to whether drinking exacerbates or attenuates air pollution effects. The apparent effect modification by drinking observed in our study needs further elaboration.

Protective effects of vegetables and fruit against hazards of air pollution

Increasing consumption frequency of vegetables and fruit was associated with decreased mortality risk from air pollution. Intake of vegetables and fruit, being antioxidants, may improve defence response (eg antioxidant defence) of individuals against the hazards of air pollution.[4]

Acknowledgements

This study was supported by the Health Care and Promotion Fund (#215032). The authors thank the Census and Statistics Department of the Government of the Hong Kong Special Administrative Region for providing the data and for their kind assistance. We also thank Sai-Yin Ho for sharing the original data collected for the Lifestyle and Mortality (LIMOR) study and Marie Chi for her secretarial support. Furthermore, we thank Deacon Lee, a medical student from Aberdeen University Medical School, for his contribution to the literature review and assistance in clerical work during his summer internship at the School of Public Health and Department of Community Medicine of The University of Hong Kong.

References

1. Verra F, Escudier E, Lebargy F, Bernaudin JF, De Crémoux H, Bignon J. Ciliary abnormalities in bronchial epithelium of smokers, ex-smokers, and non-smokers. Am J Respir Crit Care Med 1995;151:630-4.
2. Kim JY, Chen JC, Boyce PD, Christiani DC. Exposure to welding fumes is associated with acute systemic inflammatory responses. Occup Environ Med 2005;62:157-63.
3. Murphy EA, Davis JM, Brown AS, et al. Role of lung macrophages on susceptibility to respiratory infection following short-term moderate exercise training. Am J Physiol Regul Integr Comp Physiol 2004;287:R1354-8.
4. Moller P, Wallin H, Knudsen LE. Oxidative stress associated with exercise, psychological stress and life-style factors. Chem Biol Interact 1996;102:17-36.