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Effect of Winter Air Pollution on Lipid Peroxidation Product Levels of Patients with Chronic Obstructive Pulmonary Disease

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Air pollution and its effects on human health have been considered to the a serious problem in urban areas. The health hazards of outdoor air pollution are manifest across a wide spectrum of effects. Patients with respiratory disease and chronic obstructive pulmonary disease (COPD), given their abnormal responses to noxious gases and particles. The causes of oxidative stress in COPD are cigarette smoking, air pollution and increase of free radicals in respiratory epithelial cells by inflammation and infections. In the present study, we aim to investigate the effect on oxidative stress due to air pollution in patients with COPD in Diyarbakir (SE Anatolia) Turkey. Clinically stable COPD outpatients ($n = 52$) and healthy controls ($n = 42$) were studied. The lipid peroxidation product malonyldialdehyde (MDA) in serum samples was measured spectrophometrically by the Buege method. The serum MDA levels of patients with COPD (3.06 ± 0.7 nmol/ml) were significantly higher than those of control groups (0.62 ± 0.19 nmol/mL) ($p < 0,001$)

Key Words: Winter air pollution, Particulate matter, Pulmonary inflammation, Lung injury, Oxidative stress, Malonyldialdehyde.

INTRODUCTION

Air pollutions are most commonly associated with exacerbation of acute respiratory tract illnesses such as asthma or hospital admission for Chronic obstructive pulmonary disease (COPD)[1, 2]. Exacerbations of asthma and COPD feature increased the severity of the inflammatory response, which could be triggered by increased particle-derived oxidative stress and inflammation in the lungs[3].

Reactive oxygen species (ROS) are unstable compounds with unpaired electrons, capable of inititating oxidation. Several inflammatory cells that participate in the inflammatory response such as neutrophils and eosinofils and macrophages release ROS in amounts that exceed the capacity of tissue antioxidant defences[4]. At the same time ROS can cause lung injury through lipid peroxidation because superoxide ions and hydrogen peroxide released by activated immune and inflammatory cells can induce[5] the lipid peroxidation of polyunsaturated membrane fatty acids, impair membrane function and inactive membrane-bound receptors and enzymes, increase tissue permeability and therefore promote airflow limitation[6].

In the present study, we aim to investigat the effect on oxidative stress due to air pollution patients with COPD and healthy persons.

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EXPERIMENTAL

Study population and control population: 52 patients (male: 32, female: 20) with COPD hospitalized at Hospital of Dicle University between December 2003 to March 2004 were chosen for the study. The patients had no other disease except for COPD and used no other drugs. Patients were classified according to GOLD 2003 (GOLG 2003) Update.

The control population consisted of 42 healthy, non-smoker and smoker volunteers (Male: 18, Female: 26) with no history of lung disease.

Mean characteristics of the study population are shown in Table-1.

TABLE-1
THE CHARACTERISTICS AND SPIROMETRIC DATA ARE OF THE PATIENTS AND CONTROL GROUP

	COPD ($n = 52$)	Control ($n = 42$)
Age*	65.41 ± 10.33	53.19 ± 10.38
Male/Female	32/20	18/24
Smoker	31	22
Non-smoker	21	20
FEV_1 (%)*	38.70 ± 20.25	79.20 ± 13.82
FEV_1/FVC (%)*	64.86 ± 13.30	89.80 ± 13.95

*Results are mean ± SD.

Venous blood samples from study and control populations were collected and centrifuged at 3500 rpm for 10 min (Heraus Institute, GMBH Germany). Serums were separated and stored at –20°C until analysis.

The levels of serum malonyldialdehyde (MDA) (nmol/mL) were measured spectrophotometricaly by the Buege method[7]. Spectrophotometric measurements were done with Shimadzu UV-1208 spectrophotometer (Shimadzu Co., Japan).

The Mann-Whitney U-test was used to analyse the results of the study. The results were expressed in terms of arithmetic means and standard deviation.

RESULTS AND DISCUSSION

The levels of serum MDA in the study and control populations can be seen from Table-2. MDA levels of the study population having COPD were significantly higher than those of the control population ($p < 0.001$).

TABLE-2
MDA CONCENTRATIONS IN STUDY AND CONTROL POPULATIONS

	n	MDA (nmol/mL)
Control population	42	0.62 ± 0.19
Study population	52	3.06 ± 0.19

*Results are mean ± SD; $p < 0.001$.

Chronic obstructive pulmonary disease is a chronic and progressive airway obstruction and shows the characteristics of a chronic systemic inflammatory

disease. Hypoxia, systemic inflammation and oxidative stress have been described to contribute to the pathophysiology of COPD[8–11].

Free radical species affect all important cells lipids. Since lipids are oxidized by free radical attack, cell membranes are damaged. Lipid peroxides disintegrate quickly and form reactive carbon compounds among these, MDA is an important and commonly used indicator of lipid peroxidation[12].

There are many theoretical factors that can increase susceptibility to air pollution in COPD. These include reduced pulmonary reserve; airways characterized by a chronic inflammatory environment (*e.g.*, neutrofils that can respond with an oxidative response to particulates), increased comorbidity, *e.g.*, cardiovascular disease, perhaps caused by systemic inflammation, increasing airway particle dosing[13].

During the course of this study, the most significant observation was that no emission data on carbon monoxide, nitrogen oxide and hydrocarbons known as air pollution parameters were ever recorded. Only two parameters, *i.e.*, sulphur oxide and particulate matter concentrations in the atmosphere were determined in Diyarbakir (Fig. 1).

Pollution parameters, namely, the average, maximum values of sulphur dioxide and particulate matter concentrations were provided by the General Directorates of Basic Health Services of the Ministry of Health.

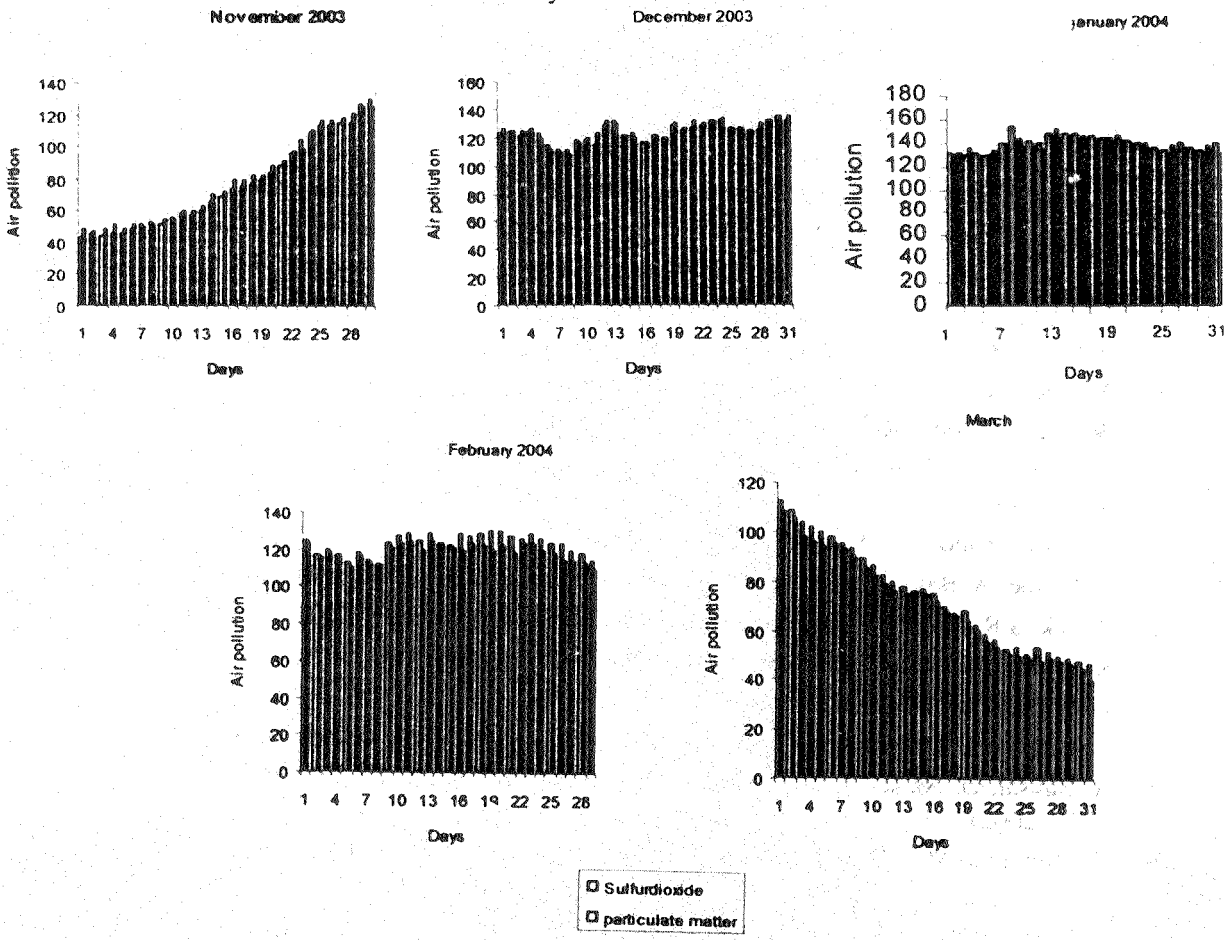

Fig. 1. Daily average sulfur dioxide and particulate matter concentrations studied for 3 months in Diyarbakir, Turkey (μg m^{-3})

In the winter seasons of year 2000–2004 in Diyarbakir (SE Anatolia of Turkey), particulate matter average concentrations were 110, 124, 104, 117 $\mu g/m^3$ respectively.

During winter seasons of year 2003–2004 the most polluted month was January with respect to particulate average concentrations, *i.e.*, 137 $\mu g/m^3$ and sulphur dioxide average, *i.e.*, 137 $\mu g/m^3$.

Air pollution was caused by sulphur dioxide and particulate discharged from low chimneys of the domestic heating, firing asphaltites. There are many asphaltite deposits around the Southeastern Anatolia of Turkey. They have been used for home heating, adding to local air pollution in the towns and cities of Southeast Anatolia region. However, these asphaltites tend to be of poor quality as fuels due to the relatively high ash, sulfur and moisture contents. The smoke was trapped in the surface inversion layer which was peculiar to the moderate weather condition[14–16].

This suggests that exposure to particles in moderate concentrations can induce oxidative stress and increase MDA concentrations in blood. Personal exposure appears more closely related to this biomarker potentially related to COPD disease than is ambient particulate matter background concentration. These effects, demonstrated in a small susceptible group of subjects with COPD, indicate that adverse outcomes can be measured in response to pollution levels that are within current guidelines.

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