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## Effects of the Use of Hypochlorite as a Cleaning Substance on Pulmonary Functions

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**Abstract:** This study was carried out to investigate the effects of a low hypochlorite concentration as a cleaning substance on pulmonary functions. For this purpose, 23 cleaning workers from the Faculty of Medicine of Suleyman Demirel University were selected as the study group and 14 people from technical personnel as the control group. The study group consisted of 17 females and 6 males with a mean age of  $26.4 \pm 5.6$  years and the mean duration of exposure to the substance in question was  $10.3 \pm 10.7$  months without any atopy history. The control group consisted of 12 females and 2 males with a mean age of  $24.4 \pm 5.4$  years. There was no atopy history. The total IgE level and the total blood eosinophil count of the study group and those of the control group were similar. Pulmonary function tests were applied three times to all the subjects on Monday morning, and Monday and Friday afternoon. In the study group, 16 subjects were symptomatic under the working circumstances and the results of the physical examination were positive in 5 out of the symptomatic subjects. The baseline values of FVC (%), FEV1 (%), FEV1/FVC (%), FEF25-75 (%), PEFR (L/s), and PEFR (%) were higher than those of the control group.

It was found that the decreases in the FEV1 (L), FEV1 (%), FEV1/FVC (%), PEFR (L/s), and PEFR (%) values of pulmonary function tests measured on Monday afternoon and the decreases in all the test values except the FVC value measured on Friday afternoon were statistically significant. There were no differences between the baseline values of pulmonary function tests except the FVC (L) value of the symptomatic and the nonsymptomatic subjects. There were significant decreases in the FEV1 (%), FEV1/FVC (%), and PEFR (%) weekend values of the nonsymptomatic group. There were statistically significant increases in the FEV1 (L) and FEV1/FVC (%) weekend values of the control group compared to the baseline values. There were statistically significant decreases in the FEV1 (L), FEV1 (%), FEV1/FVC (%), and PEFR (%) weekend values of the study group compared to those of the control group. These results suggest that even a low concentration of hypochlorite can affect the pulmonary functions by causing irritation in the airways.

**Key Words:** Hypochlorite, pulmonary functions

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### Introduction

Hypochlorite is widely used as a cleaning substance in homes and work places in our society. During the use of this substance, chlorine gas having an intermediate water solubility is released. When inhaled in low concentrations, chlorine gas can cause irritation, inflammation of the mucosal cells in the upper respiratory tract and proximal airways and exaggerated physiological responses. Inhaled in high concentrations, it can cause interstitial pneumonia, pulmonary edema, progressive respiratory failure, and death by affecting the gas exchanging parts of the lungs (1,2).

This study was conducted to investigate the effects of a low hypochlorite concentration as a cleaning substance on pulmonary functions.

### Materials and Methods

This study was carried out with 23 cleaning workers (17 females, 6 males) from the Faculty of Medicine of Suleyman Demirel University as the study group, and 14 people from the technical personnel (12 females, 2 males) of the Faculty as the control group in July 1998.

All the subjects were asked to complete a questionnaire covering the information on upper respiratory symptoms, respiratory symptoms, ocular symptoms, smoking history, and family history of atopy. In addition all of them underwent a thorough physical examination.

Blood samples were taken for the total IgE level and the total blood eosinophil count. The total IgE level was estimated by enzyme-linked immunosorbent assay

(ELISA) (Abbott Diagnostic, USA), and the total blood eosinophil count was measured by using a model STKS Coulter Counter (Coulter Electronics, USA).

Pulmonary function tests were applied three times to all the subjects on Monday morning, Monday afternoon (4 p.m.) and Friday afternoon (4 p.m.) in the seated position using a dry spirometer (Spirometrics, Inc SMI III, USA). The absolute and the predicted values of forced vital capacity (FVC), forced expiratory volume in one second (FEV1), FEV1/forced vital capacity ratio, forced expiratory flow from 25 to 75 percent of vital capacity (FEF25-75), and peak expiratory flow rate (PEFR) parameters were determined. By using the American Thoracic Society standards (3), at least three pulmonary function tests were recorded for each subject, and the best value was used as the result of the test.

The study group was divided into subgroups according to symptoms and smoking habits. Ten subjects in the study group had 5.9±5.2 pack-year and 5 subjects of the control group had 3.5±3.1 pack-year smoking status. Nine subjects in the symptomatic group had 5.6±5.5 pack-year and 1 subject of the nonsymptomatic group had 7.5±0.0 pack-year smoking status.

In this study, the concentrations of hypochlorite and chlorine were not measured because of the unavailability of the laboratory. However, hypochlorite was used diluted with water.

The results were expressed as mean values±standard error of mean (S.E.M.). The demographic and clinical characteristics between the groups were assessed by the independent samples t test. For statistical analysis of the mean values of the pulmonary function tests (baseline, Monday afternoon and Friday afternoon) in the study group or the control group, the paired t test was used. Comparisons of the mean values of the pulmonary function tests (baseline, Monday afternoon and Friday afternoon) between the study group and the control group or the symptomatic subjects and the nonsymptomatic subjects or the smoking subjects and the nonsmoking subjects were made by the Mann-Whitney U test. The differences in the mean values of pulmonary function tests (baseline, Monday afternoon and Friday afternoon) in the symptomatic subjects or the nonsymptomatic subjects and the smoking subjects or the nonsmoking subjects were tested by the Wilcoxon test. Statistical analysis were conducted by the SPSS statistical package (SPSS 9.0 for

Windows, Chicago, IL) and a p value less than 0.05 was assumed to be significant (4).

**Results**

In the study group, the mean age was 26.4± 5.6 years, the mean duration of exposure to hypochlorite 10.3±10.7 months, the total serum IgE level 93.0±99.0 IU/ml, and the total blood eosinophil count 0.2±0.1 %. In the control group, the mean age was 24.4±5.4 years, the total serum IgE level 53.7±50.8 IU/ml, and the blood eosinophil count 0.2±0.2 %.

There were no statistically significant differences between the study group and the control group for the total serum IgE level, the total blood eosinophil count, and the smoking status.

Both the study group and the control group had no history of atopy such as bronchial asthma, allergic rhinitis, or atopic dermatitis.

While the study group revealed some symptoms developed in the working circumstances during the study, the control group did not. As shown in Table 1, of the 23 cleaning workers interviewed, 16 (82.0 %) had the working symptoms. Sixteen (100.0%) out of the symptomatic subjects had respiratory symptoms, 11 (68.8%) had symptoms related to ear, nose and throat, 8(50.0%) ocular symptoms, and 7 (43.8%) had headaches.

Table 1. The distribution of the working symptoms among the study group.

Symptoms	Subject number	(%)	Symptoms	Subject number	(%)
Cough	5	31.3	Throat tickling	4	25.0
Dyspnea	5	31.3	Rhinorrhea	2	12.5
Wheeze	3	18.8	Sore throat	4	25.0
Chest tightness	3	18.8	Sneezing	1	6.3
Ocular symptoms	8	50.0	Headache	7	43.8

The baseline (Monday morning) values of pulmonary function tests of the study group and the control group are listed in Table 2. The results suggest that there were statistically significant increases in the FVC (%), FEV1 (%), FEV1/FVC (%), FEF25-75 (%), PEFR (L/s), and PEFR (%) baseline values of the study group (p<0.05).

Table 2. The baseline (Monday morning) values of pulmonary function tests of the study group and the control group.

	Study group	Control group	p
FVC (L)	3.77±0.7	3.67±0.8	p>0.05
FVC (%)	106.2±13.2	96.2±11.4	p<0.05
FEV1 (L)	2.90±0.7	2.53±0.9	p>0.05
FEV1 (%)	94.2±17.4	75.6±16.3	p<0.05
FEV1/FVC (%)	88.3±14.1	78.5±16.9	p<0.05
FEF25-75 (L/s)	2.84±1.0	2.50±1.4	p>0.05
FEF25-75 (%)	76.7±23.4	62.1±25.5	p<0.05
PEFR (L/s)	4.23±1.7	3.67±2.8	p<0.05
PEFR (%)	57.3±18.0	47.0±26.9	p<0.05

The values of pulmonary function tests of the study group measured on Monday morning and Monday afternoon show statistically significant decreases in the FEV1 (L), FEV1 (%), FEV1/FVC (%), PEFR (L/s), and PEFR (%) values on Monday afternoon compared to those in the baseline values (p<0.05) (Table 3).

Table 3. The values of pulmonary function tests of the study group on Monday morning and Monday afternoon.

	Study group Monday morning	Study group Monday afternoon	p
FVC (L)	3.77±0.7	3.45±0.6	p>0.05
FVC (%)	106.2±13.2	100.9±14.9	p>0.05
FEV1 (L)	2.90±0.7	2.41±0.7	p<0.05
FEV1 (%)	94.2±17.4	80.7±20.6	p<0.05
FEV1/FVC (%)	88.3±14.1	79.7±18.2	p<0.05
FEF25-75 (L/s)	2.84±1.0	2.36±1.1	p>0.05
FEF25-75 (%)	76.7±23.4	64.9±27.6	p>0.05
PEFR (L/s)	4.23±1.7	3.47±1.8	p<0.05
PEFR (%)	57.3±18.0	49.9±19.9	p<0.05

The values of pulmonary function tests of the study group measured on Monday morning and Friday afternoon reveal statistically significant decreases in all the test values except the FVC value on Friday afternoon (p<0.05, p<0.01, p<0.001) (Table 4).

Table 4. The values of pulmonary function tests of the study group on Monday morning and Friday afternoon.

	Study group Monday morning	Study group Friday afternoon	p
FVC (L)	3.77±0.7	3.64±0.6	p>0.05
FVC (%)	106.2±13.2	102.8±11.5	p>0.05
FEV1 (L)	2.90±0.7	2.33±0.8	p<0.01
FEV1 (%)	94.2±17.4	72.0±21.4	p<0.001
FEV1/FVC (%)	88.3±14.1	70.8±20.3	p<0.001
FEF25-75 (L/s)	2.84±1.0	2.19±1.1	p<0.05
FEF25-75 (%)	76.7±23.4	59.9±26.8	p<0.01
PEFR (L/s)	4.23±1.7	3.23±1.7	p<0.05
PEFR (%)	57.3±18.0	44.4±22.6	p<0.05

The values of pulmonary function tests of the study group and the control group measured on Monday afternoon are listed in Table 5. It is evident that there was a statistically significant decrease in the FEV1 (L) value of the study group compared to that of the control group (p<0.05).

Table 5. The values of pulmonary function tests of the study group and the control group on Monday afternoon.

	Study group Monday afternoon	Control group Monday afternoon	p
FVC (L)	3.45±0.6	3.77±0.7	p>0.05
FVC (%)	100.9±14.9	96.4±9.5	p>0.05
FEV1 (L)	2.41±0.7	2.88±0.7	p<0.05
FEV1 (%)	80.7±20.6	84.2±12.2	p>0.05
FEV1/FVC (%)	79.7±18.2	88.2±15.1	p>0.05
FEF25-75 (L/s)	2.36±1.1	2.88±1.2	p>0.05
FEF25-75 (%)	64.9±27.6	70.4±19.5	p>0.05
PEFR (L/s)	3.47±1.8	3.77±1.6	p>0.05
PEFR (%)	49.9±19.9	48.2±14.6	p>0.05

The values of pulmonary function tests of the study group and the control group measured on Friday afternoon are listed in Table 6. We can see that there were statistically significant decreases in the FEV1 (L), FEV1 (%), FEV1/FVC (%), and PEFR (%) weekend values of the study group compared to those of the control group (p<0.05). Although the FVC (%) value proved to be lower in the control group, it was within normal limits.

Table 6. The values of pulmonary function tests of the study group and the control group on Friday afternoon.

	Study group Friday afternoon	Control group Friday afternoon	p
FVC (L)	3.64±0.6	3.67±0.8	p>0.05
FVC (%)	102.8±11.5	95.0±8.2	p<0.05
FEV1 (L)	2.33±0.8	2.86±0.7	p<0.05
FEV1 (%)	72.0±21.4	84.3±11.9	p<0.05
FEV1/FVC (%)	70.8±20.3	89.0±16.1	p<0.05
FEF25-75 (L/s)	2.19±1.1	2.88±1.2	p>0.05
FEF25-75 (%)	59.9±26.8	72.4±22.4	p>0.05
PEFR (L/s)	3.23±1.7	4.50±20.9	p>0.05
PEFR (%)	44.4±22.6	56.8±16.2	p<0.05

The values of pulmonary function tests of the control group measured on Monday morning and Friday afternoon confirm that there were statistically significant increases in the FEV1 (L), FEV1/FVC (%) weekend values compared to those in the baseline values (p<0.05) (Table 7).

Table 7. The values of pulmonary function tests of the control group on Monday morning and Friday afternoon.

	Control group Monday morning	Control group Friday afternoon	p
FVC (L)	3.67±0.8	3.67±0.8	p>0.05
FVC (%)	96.2±11.4	95.0±8.2	p>0.05
FEV1 (L)	2.53±0.9	2.86±0.7	p<0.05
FEV1 (%)	75.6±16.3	84.3±11.9	p<0.05
FEV1/FVC (%)	78.5±16.9	89.0±16.1	p<0.05
FEF25-75 (L/s)	2.50±1.4	2.88±1.2	p>0.05
FEF25-75 (%)	62.1±25.5	72.4±22.4	p>0.05
PEFR (L/s)	3.67±2.8	4.50±20.9	p>0.05
PEFR (%)	47.0±26.9	56.8±16.2	p>0.05

In the symptomatic subjects of the study group the total IgE level was 100.8±10.5 IU/ml and the total blood eosinophil count was 0.2±0.2%. In the nonsymptomatic subjects of the study group the total serum IgE level was 74.9± 89.2 IU/ml and the total blood eosinophil count was 0.2±0.1%. There were no statistically significant differences between the symptomatic subjects and the nonsymptomatic subjects for the total IgE level, the total blood eosinophil count, and smoking status.

The baseline values of pulmonary function tests of the symptomatic and the nonsymptomatic subjects in the study group are listed in Table 8. There were nonsignifi-

cant differences between the baseline values except the FVC (L) value of the symptomatic and the nonsymptomatic subjects. Although it was found that the FVC (L) value was higher in the nonsymptomatic subjects, it was within normal limits.

Table 8. The baseline values of pulmonary function tests of the symptomatic and the nonsymptomatic subjects in the study group.

	Study group (symptomatic) Monday morning	Study group (nonsymptomatic) Monday morning	p
FVC (L)	3.58±0.5	4.21±0.7	p<0.05
FVC (%)	104.1±12.4	111.0±14.7	p>0.05
FEV1 (L)	2.88±0.6	2.95±0.8	p>0.05
FEV1 (%)	95.5±16.3	89.7±20.7	p>0.05
FEV1/FVC (%)	91.6±11.5	80.7±17.5	p>0.05
FEF25-75 (L/s)	2.93±1.0	2.63±0.9	p>0.05
FEF25-75 (%)	80.8±24.9	67.3±17.8	p>0.05
PEFR (L/s)	4.32±1.7	4.03±1.9	p>0.05
PEFR (%)	60.9±17.6	49.9±18.0	p>0.05

There were no statistically significant differences between the values of pulmonary function tests of the symptomatic subjects in the study group measured on Monday morning and Monday afternoon.

The values of pulmonary function tests of the symptomatic subjects in the study group measured on Monday morning and Friday afternoon illustrate statistically significant decreases in the FEV1 (L), FEV1 (%), and FEV1/FVC (%) weekend values compared to those in the baseline values (p<0.05) (Table 9).

Table 9. The values of pulmonary function tests of the symptomatic subjects on Monday morning and Friday afternoon.

	Study group (symptomatic) Monday morning	Study group (symptomatic) Friday afternoon	p
FVC (L)	3.58±0.5	3.48±0.5	p>0.05
FVC (%)	104.1±12.4	101.9±12.4	p>0.05
FEV1 (L)	2.88±0.6	2.40±0.8	p<0.05
FEV1 (%)	95.5±16.3	78.4±21.4	p<0.05
FEV1/FVC (%)	91.6±11.5	77.4±18.8	p<0.05
FEF25-75 (L/s)	2.93±1.0	2.43±1.2	p>0.05
FEF25-75 (%)	80.8±24.9	67.4±28.9	p>0.05
PEFR (L/s)	4.32±1.7	3.75±1.9	p>0.05
PEFR (%)	60.9±17.6	53.7±22.1	p>0.05

The values of pulmonary function tests of the symptomatic and the nonsymptomatic subjects measured on Monday afternoon are listed in Table 10. The decreases in the FEV1 (L), FEV1 (%), FEV1/FVC (%), FEF25-75 (L/s), FEF25-75 (%), PEFR (L/s), and PEFR (%) values of the nonsymptomatic subjects were statistically significant ( $p<0.05$ ).

Table 10. The values of pulmonary function tests of the symptomatic and the nonsymptomatic subjects on Monday afternoon.

	Study group (symptomatic) Monday afternoon	Study group (nonsymptomatic) Monday afternoon	p
FVC (L)	3.38±0.6	3.67±0.5	$p>0.05$
FVC (%)	99.4±16.3	105.4±10.9	$p>0.05$
FEV1 (L)	2.59±0.7	1.90±0.5	$p<0.05$
FEV1 (%)	88.3±19.2	62.3±15.2	$p<0.05$
FEV1/FVC (%)	86.5±13.9	59.1±16.2	$p<0.05$
FEF25-75 (L/s)	2.63±1.2	1.54±0.5	$p<0.05$
FEF25-75 (%)	72.6±28.6	41.9±12.3	$p<0.05$
PEFR (L/s)	3.95±1.9	2.00±0.7	$p<0.05$
PEFR (%)	58.2±18.3	24.7±7.5	$p<0.05$

The values of pulmonary function tests of the symptomatic and the nonsymptomatic subjects measured on Friday afternoon reveal statistically significant decreases in the FEV1 (%), FEV1/FVC (%), and PEFR (%) weekend values of the nonsymptomatic subjects ( $p<0.05$ ) (Table 11).

Table 11. The values of pulmonary function tests of the symptomatic and the nonsymptomatic subjects on Friday afternoon.

	Study group (symptomatic) Friday afternoon	Study group (nonsymptomatic) Friday afternoon	p
FVC (L)	3.48±0.5	4.02±0.8	$p>0.05$
FVC (%)	101.9±12.4	104.5±10.1	$p>0.05$
FEV1 (L)	2.40±0.8	1.89±0.5	$p>0.05$
FEV1 (%)	78.4±21.4	58.1±15.5	$p<0.05$
FEV1/FVC (%)	77.4±18.8	56.3±19.0	$p<0.05$
FEF25-75 (L/s)	2.43±1.2	1.64±0.5	$p>0.05$
FEF25-75 (%)	67.4±28.9	43.3±11.8	$p>0.05$
PEFR (L/s)	3.75±1.9	2.02±0.5	$p>0.05$
PEFR (%)	53.7±22.1	26.1±8.7	$p<0.05$

The results of the physical examination made on Friday afternoon were positive (rhonchus) in 5 of the symptomatic subjects.

There were nonsignificant differences between the smokers and the nonsmokers in the study group for the total serum IgE level and the total blood eosinophil count. The FEV1 (L), FEV1/FVC (%), FEF25-75 (L/s), FEF25-75 (%), PEFR (L/s), and PEFR (%) baseline values of pulmonary function tests were lower in the nonsmokers. There were statistically significant decreases in the FEV1 (L), FEF25-75 (L/s), and PEFR (L/s) weekend values of pulmonary function tests of the nonsmokers compared to those of the smokers. No statistically significant differences were found between the values of pulmonary function tests of the smokers measured on Monday morning and Friday afternoon.

## Discussion

Chlorine gas, which is released during the use of hypochlorite, leads to the formation of hydrochlorite acid and free oxygen radicals by reacting with water in the mucous membranes. Chlorine gas, the hydrochlorite acid and the free oxygen radicals can cause mucous membrane irritation, bronchospasm, pneumonia, and pulmonary edema (5,6).

Hypochlorite (diluted 1:10) includes 5.0 ppm free chlorine (7). The pungent odor of chlorine is even detected in concentrations as low as 0.5 ppm, but chlorine causes respiratory damage only at levels above 20.0 ppm (8). According to the guide of the National Institute of Occupational Safety and Health, the recommended 8-hour occupational permissible exposure level for chlorine is 0.5 ppm and the recommended occupational 15-minute ceiling short-term exposure limit for chlorine is 1.0 ppm (9).

The acute inhalation in toxic concentrations of the respiratory irritants such as hypochlorite and chlorine gas results in a clinical entity described as reactive airways dysfunction syndrome (RADS) (10,11). It is generally believed that exposure to nontoxic levels of irritant gases does not lead to impaired pulmonary function, although little is known of the long-term consequences of an acute high-level or chronic exposure to these substances (10,12). Recently, it has been thought that repeated exposure to the irritant inhalation in tolerable concentrations can also cause RADS (10,11).

Reactive airways dysfunction syndrome is a clinical pathologic entity characterized by exposure to a toxic or irritant chemical, a negative history of obstructive symptoms prior to exposure, persistence of obstructive symptoms after exposure, objective evidence of obstructive airways disease and/or nonspecific bronchial hyperresponsiveness, and abnormal bronchial biopsy results (10,11). It differs from hypersensitivity-induced occupational asthma because of the absence of a preceding latent period and the onset of the illness after a single exposure (1,11).

In this study, 82.0% of the study group developed different symptoms related to the lungs, ear-nose-throat, and eyes which diminished or disappeared outside work. These symptoms are thought to occur as a result of the direct irritant effect of hypochlorite. Irritants deposited or dissolved in the upper respiratory tract and proximal airways may cause injury to airway mucosal cells, exaggerated physiological responses, an acute inflammatory reaction, or all three of these. Exaggerated physiological responses include coughing as a result of stimulation of afferent nerve endings in the airway mucosa, mucus secretion of submucosal and goblet cells, and acute airway narrowing (1,13).

The following findings suggest that there were obstructive signs in the hypochlorite exposed group compared to the control group: a) the baseline values of pulmonary function tests of the study group and the control group were in normal limits (Table 2); b) there were significant decreases in the weekend values of pulmonary function tests especially in the FEV1 (L), FEV1 (%), and FEV1/FVC (%) values of the study group compared to those of the baseline values (Table 4); c) there were significant decreases in the FEV1 (L), FEV1 (%), FEV1/FVC (%), and PEFR (%) weekend values of pulmonary function tests of the study group compared to those of the control group (Table 6); d) there were significant increases in the weekend values of pulmonary function tests of the control group compared to the baseline values (Table 7). In addition, it was found that there were significant decreases in the values of pulmonary function tests measured on Friday afternoon of the symptomatic and non-symptomatic subjects in the study group compared to the baseline values and the obstructive influence was higher in the nonsymptomatic subjects than that in the symptomatic subjects. Controlled human exposure data suggest that respiratory symptoms following irritant exposure are

associated with smoking and asthma and typically resolve quickly, and continuing symptoms are associated with persistent increased airway responsiveness without other pulmonary function abnormalities (14,15).

There were significant decreases in the baseline and the weekend values of pulmonary function tests of the nonsmokers compared to those of the smokers, and also nonsignificant differences in the weekend values of pulmonary function tests of the smokers compared to the baseline values show that smoking is not a risk factor. As a matter of fact, it is reported that smoking is a predisposing factor to the development of IgE-mediated occupational asthma, and it increases sensitiveness against the exposed agent and is not effectual in the irritant-induced asthma (16).

There is only limited controlled human chlorine exposure data. In a relevant controlled human chlorine exposure study in which eight healthy volunteers were exposed to chlorine in concentrations of 0.5 ppm or 1.0 ppm, there were unimportant changes observed with the 0.5 ppm exposure, and with the 1.0 ppm exposure, there was a decrease in airflow and an increase in airway resistance. In addition, there were significant changes after only 4-hour-exposure and most of the test results had returned to normal by the next day (17). In another study in which airway responses to chlorine inhalation (0.4 or 1.0 ppm concentrations) among subjects with nonspecific airway hyperreactivity were directly examined, there was an acute, short-term fall in airflow and an increase in airway resistance in both normal subjects and subjects with increased nonspecific airway reactivity after 1.0 ppm chlorine inhalation and a significantly greater response among the hyperresponsiveness group as compared with normal subjects. At 24-hour follow-up there were nonsignificant chlorine-related pulmonary function deficits and, in addition, after 0.4 ppm chlorine inhalation, there was also a nonsignificant pulmonary function effect (9).

Persistent asthma is reported to occur after the inhalation of the irritant in high concentrations (5). It is thought that the symptoms continuing for years are associated with persistent bronchial hyperreactivity without other pulmonary function abnormalities (15,18).

In conclusion, hypochlorite, even used in low concentrations, can lead to clinical and functional defects by causing irritation in the airways.

## References

1. Taylor AJN. Respiratory irritants encountered at work. *Thorax* 51:541-545,1996.
2. Schwartz DA, Smith DD, Lakshminarayan S. The pulmonary sequelae associated with accidental inhalation of chlorine gas. *Chest* 97:820-825, 1990.
3. American Thoracic Society statement: Standardization of spirometry-1994 update. *Am J Respir Crit Care Med* 152:1107-1136, 1995.
4. Sümbüloğlu K, Sümbüloğlu V. Biyoistatistik. 7. Baskı. Hatipoğlu Yayınevi. Ankara, 1997.
5. Deschamps D, Soler P, Rosenberg N, Baud F, Gervais P. Persistent asthma after inhalation of a mixture of sodium hypochlorite and hydrochloric acid. *Chest* 105:1895-1896, 1994.
6. Can H, Kaşıkır N, Özdemir M, Atabey F, Öztürk S, Dodurgalı R, Poluman A. Hidroklorik asit ve hipokloridin solunum yollarına etkisi. *Solunum* 20: 829-832, 1996.
7. Berry MA, Bishop J. Suggested guidelines for remediation of damage from sewage backflow into buildings. *J Environ Health* 57:9-16, 1994.
8. Moore BB, Sherman M. Chronic reactive airway disease following acute chlorine gas exposure in an asymptomatic atopic patient. *Chest* 100:855-856,1991.
9. D'Alessandro A, Kushner W, Wong H, Boushey HA, Blanc PD. Exaggerated responses to chlorine inhalation among persons with nonspecific airway hyper-reactivity. *Chest* 109:331-337,1996.
10. Alberta WM, do Pico GA. Reactive airways dysfunction syndrome. *Chest* 109:1618-1626, 1996.
11. Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome (RADS): persistent asthma syndrome after high level irritant exposure. *Chest* 88: 376-384, 1985.
12. Boulet LP. Increases in airway responsiveness following acute exposure to respiratory irritants. *Chest* 94:476-481, 1988.
13. Wang AL, Blacford TL, Lee LY. Vagal bronchopulmonary C-fibers and acute ventilatory response to inhaled irritants. *Respir Physiol* 104:231-239, 1996.
14. Das R, Blanc PD. Chlorine gas exposure and the lung: a review. *Toxicol Ind Health* 9:439-455,1993.
15. Blanc PD, Galbo M, Hiatt P, Olson KR, Balmes JR. Symptoms, lung function and airway responsiveness following irritant inhalation. *Chest* 103:1699-1705, 1993.
16. Çağatay T. Mesleksele bronşiyal astma. *Allerjik Hastalıklar ve Bronşiyal Astma* (Ed. Aydılek R). Cilt II. Özlem Grafik Matbaacılık. İstanbul 1998. 470-477.
17. Rotmann HH, Fliegelman MJ, Moore T, Smith RG, Anglen DM, Kowalski CG, Weg JG. Effects of low concentrations of chlorine on pulmonary function in humans. *J Appl Physiol* 54:1120-1124, 1983.
18. Tarlo SM, Broder I. Irritant-induced occupational asthma. *Chest* 96:297-300, 1989.