PULMONARY TOXICITY AMONG CAR SPRAY PAINTERS

BY

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ABSTRACT

Associations between isocyanate exposure and respiratory health effects have received little attention despite the extensive use of isocyanate compounds. Spray painters comprise a large population at risk, with potentially high isocyanate exposure because most lacquers contain hexamethylene diisocyanate. Repeated pulmonary function testing is not sufficient to diagnose and evaluate pulmonary toxicity induced by car paint sprays. Therefore, Clara cell secretory protein (CC16) was selected as a sensitive marker of bronchial tree injury. The present study involved 50 workers exposed to isocyanates during car spray-painting; in addition to 30 control subjects, (20 smokers and 10 non-smokers) who were never exposed to isocyanates. All participants were subjected to pulmonary function testing (FVC, FEV1, FEV1%, and PEFR), bronchoscopy and lavage, and serum CC16 estimation. Different pulmonary function indices as well as CC16 serum levels were found to be significantly decreased in the exposed group. Smokers showed significant reduction in CC16 serum levels. Clara cell were markedly damaged in the exposed subjects, whereas smokers exhibited excessive epithelial cell desquamation. In conclusion, clear associations between lung function parameters, CC16 serum levels, and exposure to isocyanate-containing paint-sprays were demonstrated. This stresses the importance of regulation and control of such exposure. Also, assay of serum CC16 could be used to detect pulmonary toxic effects in subjects exposed to isocyanate compounds.

Keywords: Isocyanate; Pulmonary Toxicity; Spray Painter; Clara Cell Secretory Protein; Pulmonary Function Test.

INTRODUCTION

Car spray-painters are exposed to isocyanates (Alexandersson et al., 1987), which are a group of low molecular weight aromatic and aliphatic compounds containing the highly reactive isocyanate group (–NCO) (Schauerte et al., 1985). The most commonly used isocyanates include toluene diisocyanate (TDI), diphenylmethane diisocyanate (MDI), hexamethylene diisocyanate (HDI), and biuret modified HDI (HDI-BT) (Vandenplas et al., 1993).

Most of the isocyanate compounds are colorless, yellow, or brown liquids with sharp pungent odors. Inhalation and dermal exposure can occur during the manufacture and use of these compounds. Isocyanates are used as cross-linking agents in polyurethane products, such as foams, varnishes, and paints. Therefore, workers and individuals in close proximity to spray applications of polyurethane paints are very likely to be exposed (Pronk et al., 2007).

In acute toxicity, all workers will develop eye, nose, and throat irritation with coughing and labored breathing. More severe exposure may result in hypersensitivity pneumonitis and pulmonary edema. Dermal contact will result in dermatitis and eczema (Gad, 2005).

Chronic inhalation can cause immune disorders as well as nasal and lung lesions. No human information is available for the reproductive or developmental toxicity of isocyanates; however, some effects (decreased placental and fetal weights) were noted in experimental animal studies (Kapp, 2005). There is inadequate evidence for the carcinogenicity of isocyanates in humans; however, there is sufficient evidence for the carcinogenicity of toluenediisocyanates in experimental animals (Bilban, 2004).

Associations between isocyanate exposure, sensitization, and respiratory disease have received little attention (Pronk et al., 2007), despite their extensive use during spray painting. Chronic inhalation exposure to isocyanates in plant workers has been linked to pulmonary effects that are characterized by dyspnea, wheezing, and bronchial constriction (Mapp et al., 1985; Moscato et al., 1991).

Occupational asthma (OA) is a disease characterized by variable airflow limitation and/or airway hyperresponsiveness due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace (Bernstein et al., 1999). It is a potentially fatal condition, and death from isocyanates-induced asthma has been reported (Lee and Koh, 2008). Nevertheless, it may present with varying degrees of respiratory compromise accompanied by rhinoconjunctivitis with ocular and nasal discharge, pruritis, and sneezing. Mild cases present with episodic dry cough, chest tightness, and increased breathing effort, whereas severely affected patients suffer from wheezing, cough, and dyspnea on exertion (Malo et al., 1997).

Detailed medical and occupational history should be obtained to investigate the relationship between asthmatic symptoms and workplace exposures (Balmes and

Scannell, 1997). In addition, different types of pulmonary function and inhalation challenge tests are used to confirm diagnosis of OA. However, since repeated pulmonary function testing is not sufficient to diagnose and evaluate pulmonary damage induced by car paint sprays (Vandenplas et al., 1993), low-molecular weight proteins were introduced as interesting peripheral biomarkers. This choice was based on the fact that they are exchangeable and have short half-lives in plasma and may thus respond to both acute and chronic toxic effects. Clara cell secretory protein, a new low-molecular weight protein, has been selected as a marker of bronchial tree injury (Kropski et al., 2009).

Clara cells are dome-shaped cells with short microvilli found in the small airways of the lungs (Atkinson et al., 2008). Their main function is to protect the bronchiolar epithelium by secreting Clara cell secretory protein (CCSP), which has anti-inflammatory activity and, therefore, plays a role in controlling airway inflammation (Bernard et al., 2005). These epithelial cells are altered in several pathological processes induced by various pulmonary toxicants (Petrek et al., 2002). Altered Clara cell functions can be demonstrated through CCSP measurements (Broeckaert and Bernard, 2000).

Clara cell secretory protein (also called CC16) is 16 kDa homodimeric protein secreted by Clara cells into the fluid lining the bronchioles. As a result of a passive transudation, CC16 occurs in small concentrations in serum where it appears to mirror the amount secreted in the respiratory tract (Halatek et al., 1998). From the serum, it follows the same glomerular filtration-tubular reabsorption route as other low-molecular weight serum proteins (Bernard et al., 1992 a).

SUBJECTS AND METHODS

Study population :

Exposed smoker subjects: 50 male car paint workers aged from 19 to 40 years with employment duration of 5 to 12 years. They were current smokers with a mean smoking index of 17.66 ± 1.58 pack/year (range : 15-20 pack/year) (Coultas et al., 1993). Inclusion criteria in the study was based on absence of pre-occupational respiratory symptoms suggestive of asthma and chronic bronchitis; negative history of diabetes, hypertension, renal, and hepatic insufficiency. In addition to normal physical examination.

Control subjects:

- **Control smokers :** 20 healthy male smokers, who were matched for age and smoking habits.
- **Control non-smokers** : 10 healthy non-smokers, who were matched for age.

Both exposed and control groups were subjected to the following :

1- Pulmonary function tests:

Forced vital capacity (FVC), forced expiratory volume in one second (FEV1), FEV1/FVC (FEV1%) and peak expiratory flow rate (PEFR) were assessed for all subjects with a spirometer (Medisoft, Dinant, Belgium) according to the standards of the American Thoracic Society (1995).

2- Bronchoscopy and lavage procedure:

According to Vandenplas et al. (1999), each participant was premedicated with 0.5 mg atropine. After topical anesthesia of the upper respiratory tract with 4% lidocaine, the fibreoptic bronchoscope was wedged in a subsegment of the right middle lobe or lingula. Four 50-mL aliquots of sterile 0.9% saline solution at 37.8°C were instilled through the bronchoscope channel. After each instillation, the fluid was gently aspirated with a syringe, collected in siliconized glass flasks and kept on ice throughout the procedure. The first recovered aliquot was considered a bronchial lavage sample and was separated from the pool of the following three aliquots. Immediately after the end of the lavage procedure, the recovered fluid was centrifuged at 2000 rpm and cytological smears were prepared for H&E staining according to Bancroft and Stevens (1975).

3- Serum CC16 estimation procedure:

Serum samples were collected and stored at -20°C. Thawed samples were mixed thoroughly and diluted 25x with Dilution Buffer just prior to the assay. CC16 was measured by a sandwich enzyme-linked immunosorbent assay (ELISA) according to the manufacturer instructions (Biovendor - Laboratorní medicína a.s.).

4- Statistical analysis:

The results of the study were recorded as mean and standard deviation for each group and a statistical analysis using Student's t-test was done. Results were considered significant when $P<0.05$. The SPSS for Windows (version 16) software package was used (SPSS Inc, Chicago, Illinois, USA).

RESULTS

Table (1) showed the distribution of subjects with respect to number, age, employment duration, and smoking index. The three groups were matched for age; all the smokers were matched for smoking index.

1- Pulmonary function tests:

All pulmonary function test indices revealed significant decrease in the exposed group compared to other groups (Table 2). At the same time, no significant difference could be detected between smokers and non smokers.

2- Cytological results:

Examination of cytological smears prepared from the broncho-alveolar lavage fluid (BALF) of the exposed subjects revealed excessive bronchial epithelial cell desquamation (figure 1). Moreover, degenerated Clara cells with foamy cytoplasm and fragmented nuclei were detected in 42 samples out of 50 exposed subjects (figure 2). Except for excessive cellular desquamation, no other abnormal cytological data could be detected in the remaining exposed samples and in the smoker controls. Neither Clara cell damage, nor epithelial cell desquamation was detected in the non-smoker controls.

3- Serum Clara cell protein (CC16):

Compared to control groups, the exposed subjects showed significant lowering in serum Clara cell protein levels. Similarly, a statistically significant difference between CC16 serum levels in smoker and non-smoker controls was detected (Table 3).

DISCUSSION

The results of this study provide evidence for exposure–response relationship between mixtures of isocyanates in car paints and work-related respiratory symptoms. A statistically significant reduction in pulmonary function tests (FEV1, FVC, FEV1%, and PEFR) was demonstrated in the exposed group. Such reduction is clinically diagnostic of obstructive pulmonary diseases like asthma and bronchitis.

The aforementioned results can be explained in light of the findings of Taylor (2000) and Wisnewski et al. (2006). They revealed that high exposure to polyisocyanates can induce extrinsic allergic alveolitis and reactive airway dysfunction syndrome (RADS), while sensitization-induced asthma can be provoked with very low exposure. Likewise, Glindmeyer et al. (2004) demonstrated statistically significant reduction in pulmonary functions in 240 painters spraying polyurethane enamels at four aircraft maintenance plants. Furthermore, they found that exposure-related adverse effects on lung functions were reduced by frequent use of respiratory protective equipment.

In addition, epidemiological survey by Vandenplas et al. (1999) showed asymptomatic decreases in FEV1 and expiratory flow rates during the course of a work-shift among workers exposed to toluene diisocyanate.

Decreased lung function parameters (Glindmeyer et al., 2004) and high prevalence of asthma symptoms have been reported in spray-painting industry (Eifan et al., 2005). A study conducted by Tornling et al. (1990) has incorporated exposure assessment and demonstrated a relation between peak exposure and reduced lung

function in car painters who smoke. Another study, that assessed complex exposure patterns of isocyanates, revealed that positive associations with exposure were found for asthma-like and COPD-like symptoms, work-related chest tightness, and work-related conjunctivitis (Pronk et al., 2007).

Cytological examination of Clara cells obtained from BALF of the exposed subjects revealed degenerated Clara cells with foamy cytoplasm and fragmented nuclei. This can be explained by their lysosomal enzymes content, which engulf airborne toxins and break them down via cytochrome P-450 enzymes present in the smooth endoplasmic reticulum (Bernard et al., 2005). In addition, excessive bronchial epithelial cell desquamation appeared in both exposed and non-exposed control smokers.

Clara cells are considered privileged targets of organic chemicals reaching the lungs. Their cytochrome P-450 content activates many cytotoxic chemicals with subsequent Clara cells destruction (Bernard and Lauwerys, 1995). In humans, antibodies against the CC16 specifically and exclusively stain Clara cells (Singh et al., 1988), which enlighten significant reduction in serum CC16 in exposed subjects.

Lung injury by smoking has been associated with excessive epithelial desquamation that goes hand in hand with the significant reduction of serum CC16. Likewise, Petrek et al. (2002) recorded significant lowering in serum CC16 levels in smokers. Earlier studies by Bernard et al. (1992b) and Bernard et al. (1994) recorded linear dose-response relationship between smoking history and serum CC16, the latter has decreased on average by about 15% for each 10 pack-year smoking history. Bernard and Lauwerys (1995) attributed the smoking-induced decrease of serum CC16 to the reduction of Clara cell number in smokers. Such reduction was due to Clara cells destruction by both toxic metabolites (generated by tobacco-induced cytochrome P-450 activities) and irritating substances (present in smoke).

Lomas et al. (2008) concluded that serum CC16 levels were reduced in individuals with COPD, and there was a weak correlation with disease severity in former smokers. Other studies have also considered the issue of serum CC16 and its relation to pulmonary status (Gioldassi et al., 2004; Kropski et al., 2009). Altered serum CC16 levels in such studies provide further validation of CC16 as a peripheral biomarker in numerous pulmonary pathologic and toxicologic states.

CONCLUSION

Our finding of exposure-related adverse effects on pulmonary functions sug-

gested that the current methods for spray painting may not be adequately safe. Use of PFTs for following up the exposed subjects requires both baseline and serial pulmonary function testing at different times (before, during, and after work shifts), which is difficult to apply. Hence, introduction of serum CC16 assay could detect pulmonary toxic effects in mildly affected or asymptomatic subjects. However, larger scale validation of these findings is warranted to better characterize the diagnostic and prognostic role of CC16 and its relation to isocyanate exposure levels in the work environment.

Table (1): Demographic characteristics of control and exposed subjects.

	Exposed smokers	Control smokers	Control non-smokers
Number	50	20	10
Age (years) Range Mean ± SD	 19 – 40 26.5 ± 6.04	 19 – 40 29.4 ± 7.3	 20 – 37 29.7 ± 5.53
Duration of employment (years) Range Mean ± SD	 5 - 12 8.500 ± 2.149	 -	 -
Smoking index (pack/year) Range Mean ± SD	 15 – 20 17.66 ± 1.58	 15 – 23 19.7 ± 2.34	 -

Table (2): Pulmonary function tests in exposed and control subjects.

	Exposed smokers	Control smokers	Control non-smokers	P1	P2	P3
FVC (L/min.) Range Mean ± SD	 59 – 79 70.30 ± 4.34	 85-93 87.1 ± 2.14	 85-92 88.6 ± 1.83	t = 15.36 P= 0.001*	t = 16.99 P = 0.001*	t = 0.539 P= 0.133
FEV1 (L/min.) Range Mean ± SD	 45 – 67 52.72 ± 5.08	 80-90 82.05 ± 2.70	 80-88 83.3 ± 3.14	t = 19.58 P = 0.001*	t = 20.71 P = 0.001*	t = 0.867 P = 0.474
FEV1/FVC Range Mean ± SD	 64 – 95 74.84 ± 6.51	 89-97 94 ± 2.07	 90-96 93 ± 2.45	t = 14.30 P = 0.001*	t = 12.28 P = 0.001*	t = 0.836 P = 0.250
PEF (L/min.) Range Mean ± SD	 56 – 71 65.94 ± 3.91	 81-89 86.3 ± 1.97	 85-91 88.1 ± 1.72	t = 23.58 P = 0.001*	t = 21.96 P = 0.001*	t = 2.325 P = 0.057

**Significant at P<0.05.*

P1: Comparison between exposed smokers & control smokers.
P2: Comparison between exposed smokers & control non-smokers.
P3: Comparison between control smokers & control non-smokers.

Table (3): Serum levels of CC16 in exposed and control subjects.

	Exposed smokers	Control smokers	Control non-smokers	P1	P2	P3
CC16 (ng/ml) Range Mean ± SD	 6.20 – 12.6 9.18 ± 1.89	 12.1 – 16.7 14.55 ± 1.25	 17.3-21.8 19.8 ± 1.36	t =14.25 P = 0.001*	t =12.55 P = 0.001*	t =12.16 P = 0.048*

**Significant at P<0.05.*

P1: Comparison between exposed smokers & control smokers.
P2: Comparison between exposed smokers & control non-smokers.
P3: Comparison between control smokers & control non-smokers.

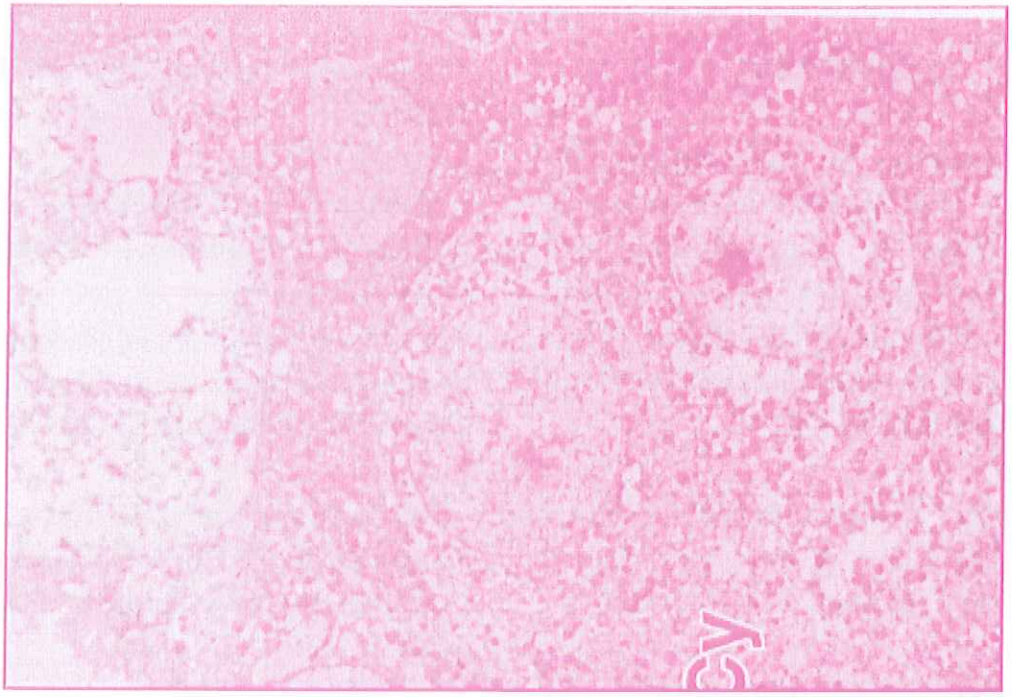

Fig. (1) : A photomicrograph of broncho-alveolar lavage of control subject showing some desquamated bronchial epithelial cells (two black arrows), foam cells (red arrow) and some normally appearing Clara cells (black arrow). *(X400)*

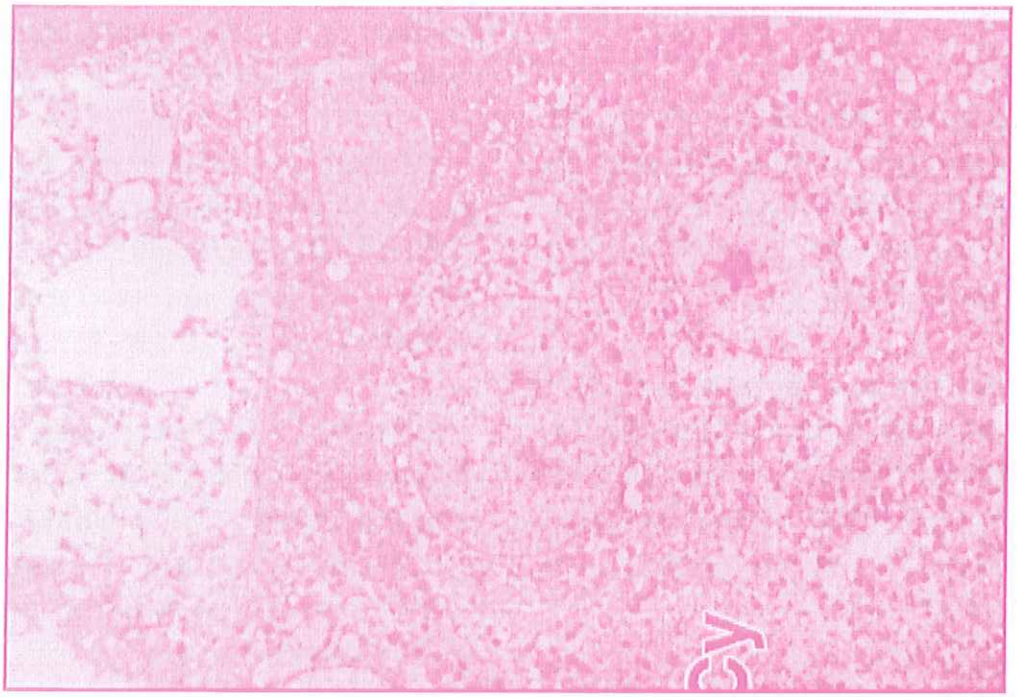

Fig. (2) : A photomicrograph of broncho-alveolar lavage of exposed subject showing excessive desquamation of bronchial epithelial cells (red arrows), degenerated Clara cells with foamy cytoplasm and fragmented nuclei (black arrows). *(X400)*

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التسمم الرئوى بين العاملين بدهان السيارات

المشتركون فى البحث

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إن العلاقة بين التعرض لمركبات الأيزوسيانات والأمراض التنفسية قد لاقت إهتماماً بسيطاً من الباحثين بالرغم من إتساع دائرة إستخدام هذه المواد، ويعتبر العاملون بدهان السيارات من أكثر الفئات تعرضاً للأيزوسيانات نظراً لوجودها فى معظم أنواع الدهانات، ولاتكفى وظائف الرئة لتشخيص ومتابعة مثل هؤلاء العمال، لذا فقد تم إختيار بروتين خلايا الكلارا باعتباره واحداً من المؤشرات الهامة التى تعكس تأثر الجهاز التنفسى عند التعرض للأيزوسيانات، وقد شملت هذه الدراسة خمسين عاملاً مدخناً يتعرضون للأيزوسيانات أثناء دهان السيارات، وكذلك ثلاثين فرداً من الأصحاء (بينهم عشرون مدخناً وعشرة من غير المدخنين) كمجموعة ضابطة، ولقد تم عمل وظائف الرئة ومنظار رئوى بالإضافة إلى قياس نسبة بروتين الكلارا فى الدم لكل منهم. وقد أوضحت الدراسة حدوث تلف بخلايا الكلارا وكذلك إنخفاض كل من وظائف الرئة ونسبة بروتين الكلارا فى العمال المتعرضين للأيزوسيانات مما يعكس إرتباطاً واضحاً بين التعرض للأيزوسيانات فى دهان السيارات وانخفاض كل من وظائف الرئة ونسبة بروتين الكلارا، الأمر الذى يؤكد أهمية حماية هؤلاء العاملين، كما يوجه عناية الباحثين إلى إستخدام بروتين الكلارا كطريقة بسيطة وفعالة لتشخيص ومتابعة حالة الجهاز التنفسى للعاملين فى دهان السيارات.