Health hazard assessment of occupationally di-(2-ethylhexyl)-phthalate-exposed workers in China

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HIGHLIGHTS

• Occupational exposure to ambient DEHP presented a potential health risk to workers.
• Comparatively high levels of DEHP were measured in respirable workplace dust samples.
• A correspondence between ambient DEHP level and biochemical markers was indicated.
• Daily intake of DEHP can be calculated by its ambient level.

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ABSTRACT

Di-(2-ethylhexyl)-phthalate (DEHP) is a potential hazard to human health. The effects of occupational high level DEHP exposure on human health were evaluated by measuring the plasma cholinesterase, residues, renal and hepatic biochemical markers. The study was conducted in three representative polyvinyl chloride manufacturing facilities from large size (S1), medium side (S2) to small size (S3). Total 456 adult males including 352 exposed workers (occupational) and 104 control workers (background) were selected. The average DEHP concentrations in respirable particulate matter were 233, 291, and 707 l/gm³ for S1–S3, respectively, compared with 0.26 l/gm³ in the background atmosphere (labeled by S4). The results showed significant decreases in post exposure plasma cholinesterase (PChE) levels (<30%) from the exposed workers as compared to baseline. These exposed workers had been evaluated for plasma DEHP residues. Regression analyses explored that PChE decreased significantly with increasing plasma DEHP residues. Serum aspartate aminotransferase, alanine aminotransferase, creatinine, urea, gamma glutamyltransferase, malondialdehyde, total antioxidant and C-reactive protein were significantly raised as compared to the controls. Of the 352 exposed workers, 116 (33.0%) had a daily DEHP intake 22.7 l/gk gb w⁻¹ d⁻¹, which is more than 20 l/gk gb w⁻¹ d⁻¹ specified by the US Environmental Protection Agency. The study demonstrated that occupational phthalate exposure produces health hazards.

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1. Introduction

Phthalate esters (PAEs) are abundant synthetic chemicals in the environment (Liu et al., 2011). Their health hazards are a major concern especially in developing countries, where PAEs are commonly used as plasticizer in the production of polyvinyl chloride (PVC). PVC plastics may contain up to 50% PAEs by weight (Meeker et al., 2009). As a plasticizer, PAEs are not chemically bound to the product and may leak out to the public environment (Staples et al., 1997). Among commercial PAEs, di(2-ethylhexyl)phthalate (DEHP) is the major phthalate plasticizer for PVC, and the content of DEHP is 50–60% (Grande et al., 2007). More than two million tons of DEHP were used each year worldwide. Its potential environmental and health hazards cannot be ignored due to its low degradability (Vermeulen et al., 2005).

Although PAEs furnish some benefits for plastics, building materials, and personal care products, they entail a number of human health hazards (Wittassek et al., 2009). Multiple sources, including the workspace, diet, personal care products and other activities, can contribute to human phthalate exposure, which
has led to the potentially serious hazardous effects to human health and environmental quality (Matsumoto et al., 2008). Humans can be exposed to PAEs carried by the airborne dust via ingestion and dermal absorption. The skin and respiratory tract provide efficient surfaces for the absorption of PAEs. The airborne dust ingestion and inhalation rates are 0.11 g d⁻¹ and 20 m³ d⁻¹ for adults. The dermal exposure area including face, forearms, hands and lower legs is 3300 cm², and the dermal adherence factor is 0.2 mg cm⁻² (Kang et al., 2012). Common exposure to PAEs is often considered to originate from food packaging materials with PAEs plasticizer (Kolarik et al., 2008). Furthermore, special attention has also been directed towards medical treatment, i.e. via use of medical devices containing DEHP, due to the fact that numerous bags and tubes used in hospitals are made of PVC and these PVC polymers leach phthalate plasticizer DEHP (Koch et al., 2005). PAEs hazards on professional workers in PVC industries are the most concern, DEHP is released to the atmosphere as respirable workplace dust (PM₁₀, particulate matter with aerodynamic diameter <10 μm) contaminated with high PAE levels, which cause bronchial obstruction and asthma if inhaled by the professional workers (Gaudin et al., 2011). This workplace dust can penetrate effectively into the respiratory system and deposit deeply in the bronchioles and alveoli of the lungs. The association of workplace dust with these chemicals may contribute to adverse health effects or potentially result in long-term health hazards (Wang et al., 2008).

Adverse human effects of PAEs depend upon their potential toxicity, as well as levels and exposure duration. Toxicological evaluation of these chemicals has shown that long-term exposure to high PAE levels can affect multiple organs including liver and kidney functions (Heudorf et al., 2007). DEHP is the most commonly used phthalate plasticizer in the production of PVC. It is suspected to be endocrine disrupting chemicals exhibiting carcinogenic action (Calafat and McKee, 2006). As a result, DEHP has been added to the list of “chemicals of concern” by the US Environmental Protection Agency (EPA). Although many studies were carried out to investigate the possible toxicity of PAEs including DEHP, their health hazards were observed in laboratory animals, mainly in rats (Andrade et al., 2006; Grande et al., 2007; Christiansen et al., 2010), there remain questions about human health effects of these chemicals (Latini et al., 2003; Gaudin et al., 2011).

 Clinically, occupationally exposed chemical workers are more likely to represent with potential adverse symptoms (David and Gans, 2003). Biomarkers may be used to detect the hazardous effects of chemicals before clinical adverse events occur (Manno et al., 2010). In the late 20th century, several epidemiological studies have shown an association between high DEHP exposure and adverse effects on human endocrine or reproductive system (Andrade et al., 2006; Benson, 2009; Eveillard et al., 2009; Gaudin et al., 2011). At present, DEHP has widely spread in our environment. Does occupationally exposure to high dose DEHP cause adverse poison symptoms? There is a concern for estimating the association between high DEHP exposure and human health risks. Information about DEHP pollution in the occupational environment and workforces is limited. In addition, the human exposure to DEHP considering as a health hazard is not well reported. In this paper, we reported the hazardous health effects of occupationally DEHP-exposed workers, by measuring plasma cholinesterase (PChE), DEHP residues, and biochemical markers, with the aims of establishing baseline levels needed for assessing DEHP safety in exposure among populations.

2. Materials and methods

A longitudinal comparative study was conducted to investigate the DEHP health effects on workers from three PVC factories ranging in size, S1 being a large factory (9100 workers), S2 being medium size (4645 workers) and S3 being small (888 workers).

2.1. Subjects

Sampling sites and selected workers are summarized in Table 1. 456 workers from the three PVC factories were identified, 352 workers from the production department normally exposed to airborne dusts were selected as exposed workers and 104 office workers were as control workers. The exposed workers ranged between 20 and 45 years of age: 125 of them from S1, 111 from S2 and 116 from S3. They were full time workers actively involved in PVC manufacturing. The 104 control workers have the comparable socio-economic status as the exposed workers but were seldom exposed to DEHP. Each participant was interviewed with questions about their general status, exposure history, smoking habits, previous medical records, and present symptoms. Medical history and physical examination of the subjects were also carried out before tests. Those with diabetes mellitus, hypertension, and viral hepatitis were excluded. A record of their exposure history, smoking habits and general health conditions were maintained.

2.2. Sample collection

A trained nurse from the factory’s Occupational Health Service was assigned to perform blood sampling. Each participant took two blood tests: one when the worker was newly employed and one after the worker had worked in the same place for 3 years. The blood samples were kept in cold chamber while being transported to the analytical laboratory. Blood plasma was separated by blood centrifugation at 1500 × g for 15 min. Samples were stored at −20 °C in clean glass vials for biochemical and DEHP analysis.

The studies were conducted in the different period of time. Airborne dust samples were collected from the three factories: 5 samples each from their worksites and 2 samples each from their office areas during the months of January, April, July, and November. Each sampling period was 6 consecutive days every three
months. A total of 1512 air samples were collected over a 3-year period from 2009 to 2012. Particle-bound DEHP was captured by flowing a total of 24 m³ collected air samples at a rate of 16.7 L min⁻¹ through a 47 mm diameter Whatman quartz fiber filter with a sampling inlet head less than 10.0 μm. Prior to use, the quartz filters were baked at 550 °C for 12 h to remove organic impurities. After use, they were kept in individual Petri dishes in a −20 °C freezer until analysis.

2.3. Analytical methods

Plasma biochemical analysis was carried out with a Selectra E auto analyzer (Vita Lab, Netherlands) following standard procedures of Pioneer Diagnostic Kits (New York, USA) at the medical laboratory of the General Hospital of Nanjing Military Command, China. PChE levels were estimated by Ellman’s method with butyrylthiocholine (Ellman et al., 1961). Serum alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP) were measured using the International Federation of Clinical Chemistry and Laboratory Medicine method (Bergmeyer et al., 1998). Serum total bilirubin (TBIL) was measured from Jendrassik and Grot method (Perry et al., 1983), plasma albumin (ALB) by bromocresol green (Doumas et al., 1971), and total protein (TP) by Biuret method (Kingsley, 1942). Gamma glutamyltransferase (GGT) was determined by an International Federation of Clinical Chemistry reference measurement procedure (Schumann et al., 2002). Serum creatinine (Cr) was determined by a Waters Empower Chromatography Manager System (Waters Inc., USA). The eluant used was the mixture of 90% methanol and 10% water at a flow rate of 1.0 mL min⁻¹. Detection was set at a wavelength of 228 nm. An ultrasonic cleaner (The Second Medical Instrument Plant, Beijing, China) was used for sonication of sample solution and mobile phase. All organic solvents used were tested for possible contaminants. Blanks were extracted and examined to provide an assessment of the blank contamination introduced by laboratory procedures, and field blanks were prepared and handled by exactly the same procedures as those used for the source samples for contaminants. DEHP in the samples was identified on the basis of retention times and quantified on the basis of peak area by using external standard method. The retention time for DEHP was 12.5 min. The recovery of DEHP was from standard spikes treated and processed like samples. It varied between 86.7% and 104.3%. Results presented in this study were blank and recovery corrected. The overall deviation of the HPLC was <±7% for DEHP.

2.6. Statistical analysis

The data were entered into SPSS version-15 (SPSS Inc, Chicago, USA) for statistical analysis. Post-exposure PChE in the participating workers was compared to baseline levels by Wilcoxon rank sum test. Biochemical, oxidative, and inflammatory markers in the exposed workers were analyzed by Kruskal–Wallis test. Mann–Whitney U test was applied for pairwise group comparisons. Association of reduction PChE (%) with change in biochemical markers in the workers was established by Spearman’s coefficient correlations. The probability value of p < 0.05 was set as the level for statistical significance.

3. Results and Discussion

3.1. Ambient level of DEHP

On the basis of the investigated level of ambient DEHP, we selected high and low DEHP intensity locations in the different industrial workplaces. To examine the site variation of DEHP exposure, we simultaneously measured the ambient DEHP concentrations at the three worksites and their office areas, respectively. The exposed profile of ambient DEHP had been displayed in Fig. 1A. Collectively, S3 had highest ambient DEHP level of 707 μg m⁻³ (n = 360). The median concentration (291 μg m⁻³ (n = 360)) of DEHP at S2 was slightly higher than at S1 (233 μg m⁻³, n = 360). The background level at S4 was 0.26 μg m⁻³ (n = 432). The ambient DEHP level shows the trends as S3 > S2 > S1 > S4, which was consistent with that air monitor located in a heavily
exposed workplace with production release gas a potential prime source for atmospheric DEHP (Hines et al., 2009). In S3, backward production lines and unregularly operating processes were likely the contributors to high DEHP exposure. The high temperature attained in the worksites increased the level of DEHP in the air.

Release of DEHP to indoor worksites could occur during PVC production and also during the incorporation of the phthalate into the final products. The occupationally exposed DEHP concentration depended not only on the PVC production procedure but also on the functions of the workspace in which the manufacturing process occurs.

3.2. Clinical symptoms

Total 352 surveyed workers were males with age ranging from 20 to 45 years. The average duration of occupational exposure was 3 years. The workers of small-sized PVC industrial unit experienced more clinical symptoms of central nervous, gastrointestinal, and respiratory systems than the other factories. Frequent headaches followed by dizziness, fatigue, nausea, muscle weakness, vomiting, skin rash, and shortness of breath happened significantly among these workers. Only some workers complained of shortness of breath as depicted in Fig. 1B.

In our study, the surveyed workers were constantly exposed to toxic DEHP for 9 h per work day (8 h of work plus 30 min to 1 h of lunch or break time). Unconformable medical symptoms occurred significantly higher among the workers than in the general population. Extensive exposure to high dose DEHP caused adverse health effects in the workers ranging from minor respiratory problems and allergy symptoms up to neurotoxic diseases. As can be seen from Fig. 1B, the workers may have some clinical symptoms like headache, dizziness, fatigue, nausea, muscle weakness, vomiting, skin rash, and shortness of breath. Distribution of clinical symptoms from the participants at different worksites increases in the order of: S3 > S2 > S1 > S4. Clearly, occupational exposure to environmental DEHP presented a potential health risk to workers.

3.3. Cholinesterase activity

Fig. 2A presented a decrease in plasma level of post exposure PChE in the workers as compared to baseline. The participants of
small-sized and medium-sized industrial units had significantly lower median post-phthalates exposure vs. baseline PChE 5343 vs. 6937 U L\(^{-1}\) and 5735 vs. 6731 U L\(^{-1}\), respectively. The participants of large-sized industry had similar trend of median 6578 vs. 6915 U L\(^{-1}\), but decreases in post exposure PChE levels were not significant for the control workers.

Serum PChE was routinely measured as a function test and is recognized as an important test for diagnosis of substantial liver disorder. Depression in PChE levels >30% as compared to baseline was considered adverse phthalate exposure. PChE measurement is used widely to investigate incidents of organophosphorus pesticides (OP) over-exposure or as part of health surveillance in workers at risk of OP exposure (Khan et al., 2010). Similar evaluation of the PChE depression was also applied to DEHP exposure. The results indicated that high occupational exposure could cause substantial depression in human PChE activity, as seen in Fig. 1A and Fig. 2A. Since PAE-induced PChE depression was used as an indicator of over-exposure, the PChE depression may well reflect the levels of anticholinergic toxicity in the adverse occupational exposure to DEHP. The exposed workers had depression of PChE more than 30% in small-sized (n = 25), medium-sized (n = 10), and large-sized PVC factories (n = 7) (Fig. 2B).

Certain synthetic chemicals like DEHP inhibited the PChE, which was helpful for detecting the early poison symptoms, as shown in Fig. 1B. In order to estimate the chemical-induced toxicity, the level of PChE activity in chemical-exposed workers was a good biological monitoring factor for several years (Manno et al., 2010). PChE levels were more reliable indicators than the visible clinical symptoms for risk assessment and monitoring of phthalate exposure. This result was also consistent with a study by Hernández et al. (2004), both directly and indirectly exposed workers presented significant decreased levels of PChE when exposed to chemicals extensively.

3.4. Biochemical markers

3.4.1. Hepatic injury

Since other synthetic chemicals also inhibit PChE and affect other biomarkers of hepatic injury. The measurements of PChE and hepatic injury may not be an effective indicator to assess toxic effects of DEHP exposure. Here, the group of 104 office workers does not expose to other chemicals. This idea extends to the other biomarkers of hepatic injury that we measured. Table 2 illustrated that these exposed workers had slight hepatic injury. The enzyme activities of ALT, GGT, and MDA were significantly raised among the exposed workers in comparison of the control workers. Other biological parameters, such as plasma TBIL, ALB, TP, urea, and Cr were not sensitive to the exposures.

3.4.2. Oxidative stress markers

Table 2 shown that the small-sized factory workers (S3) had increased oxidative stress with raised plasma GGT, MDA, CRP levels and reduced total antioxidant status, as compared to the other factories. Plasma CRP levels were also increased due to nonspecific inflammatory changes in the exposed workers. Conversely, the plasma total antioxidants of workers were significantly decreased, which proved that their human defense system had been potentially weakened. A possible explanation for this finding was that the oxygen radicals (O\(_2\)) could not be cleared and then caused large accumulation of radicals, which enhanced the destruction of liver cells. The oxidative radical resistant system in human body, similar to the immune system, is complicated and well functioned unit. Liver consumes oxygen and balances the production/elimination of oxygen radicals dynamically during metabolism in the body. If the balance were broken with less oxygen radicals, the human being would be difficult to survive. In contrast, more oxygen radicals would relatively weaken the capability to remove

### Table 2

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Exposed (n = 352)</th>
<th>S4 (control, n = 104)</th>
<th>Reference value</th>
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<tbody>
<tr>
<td></td>
<td>S3</td>
<td>S2</td>
<td>S1</td>
</tr>
<tr>
<td>Hemoglobin (HGB) (g dL(^{-1}))</td>
<td>12.1 ± 1.1(^a)</td>
<td>12.8 ± 1.2</td>
<td>13.3 ± 1.1</td>
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<td>(11.2–14.7)</td>
<td>(11.1–14.3)</td>
<td>(11.2–14.3)</td>
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<tr>
<td>TBIL (µM)</td>
<td>11 ± 1.0</td>
<td>10 ± 0.7</td>
<td>9.0 ± 1.1</td>
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<tr>
<td></td>
<td>(8.0–13)</td>
<td>(7.0–12)</td>
<td>(7.0–12)</td>
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<tr>
<td>ALT (IU L(^{-1}))</td>
<td>43 ± 3.3</td>
<td>37 ± 3.0</td>
<td>35 ± 4.7</td>
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<td>AST (IU L(^{-1}))</td>
<td>26 ± 3.5</td>
<td>27 ± 2.0</td>
<td>24 ± 2.8</td>
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<td>ALP (IU L(^{-1}))</td>
<td>191 ± 16.7</td>
<td>193 ± 12.8</td>
<td>188 ± 15.4</td>
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<td>ALB (g L(^{-1}))</td>
<td>44 ± 2.8</td>
<td>44 ± 1.5</td>
<td>43 ± 1.7</td>
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<td>Urea (mM)</td>
<td>4.51 ± 0.38</td>
<td>4.34 ± 0.21</td>
<td>4.24 ± 0.31</td>
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<td>(4.05–5.11)</td>
<td>(4.10–5.05)</td>
<td>(3.70–4.77)</td>
</tr>
<tr>
<td>Cr (µM)</td>
<td>68.3 ± 5.3</td>
<td>69.7 ± 6.8</td>
<td>69.0 ± 4.2</td>
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<tr>
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<td>(49.5–93.3)</td>
<td>(49.2–96.3)</td>
<td>(47.2–99.4)</td>
</tr>
<tr>
<td>GGT (U L(^{-1}))</td>
<td>36 ± 1.6</td>
<td>31 ± 1.8</td>
<td>30 ± 1.2</td>
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<td></td>
<td>(22–60)</td>
<td>(18–56)</td>
<td>(15–52)</td>
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<tr>
<td>MDA (µM)</td>
<td>4.54 ± 0.52</td>
<td>4.42 ± 0.45</td>
<td>4.35 ± 0.58</td>
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<td>(3.72–5.30)</td>
<td>(3.51–4.76)</td>
<td>(3.55–4.91)</td>
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<tr>
<td>CRP (mg L(^{-1}))</td>
<td>1.55 ± 0.23</td>
<td>0.90 ± 0.11</td>
<td>0.74 ± 0.10</td>
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<td></td>
<td>(0.90–2.65)</td>
<td>(0.30–1.30)</td>
<td>(0.30–1.20)</td>
</tr>
<tr>
<td>TP (g L(^{-1}))</td>
<td>65 ± 3.8</td>
<td>66 ± 3.2</td>
<td>68 ± 4.5</td>
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<td></td>
<td>(60.2–69.4)</td>
<td>(62.3–71.7)</td>
<td>(63.2–73.8)</td>
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<tr>
<td>Total antioxidant (mM)</td>
<td>1.40 ± 0.21</td>
<td>1.46 ± 0.13</td>
<td>1.48 ± 0.17</td>
</tr>
</tbody>
</table>

\(^a\) Median ± standard deviation.
or radicals and damage the body (Deneke and Fanburg, 1989). Additionally, the radicals could be decomposed in the extracellular fluid matrix of hyaluronic acid, so that the skeleton of membrane phospholipid polyunsaturated fatty acid peroxidation damage to cell membranes affected the structure and function of ribonucleic acid, deoxyribonucleic acid, protein oxidation and crosslinking. The oxygen radicals have oxidation properties and can decompose to more harmful species. These harmful species may damage cell membrane, thus affecting the body's normal metabolism, induced by a variety of clinical symptoms (Koch et al., 2011).

Our study had revealed significant association between reduction PChE (%) with change in biochemical indicators of oxidative stress and inflammation in the occupational exposure. Interestingly, the negative correlations of PChE levels with ALT (R² = 0.931, p < 0.0001), GGT (R² = 0.931, p < 0.0001), MDA (R² = 0.826, p < 0.0001) and CRP (R² = 0.922, p < 0.0001) had been observed among the exposed workers (see Supplementary Fig. S1 online). Consistently, the correlation of post exposure PChE reduction (%) with total antioxidant status (R² = 0.759, p < 0.0001) was exhibited in Fig. 3A. Calibration equation was calculated as Y = 1.72 – 0.00861X.

These exposed workers were fully equipped to meet any emergency situation under current safety regulations. The workers' phthalate exposure was monitored by measuring PChE levels. We found that long term occupational exposure with increased ambient DEHP level accumulatively decreases PChE levels and causes derangement of hepatic renal function. These symptoms were found from the oxidative stress and inflammatory markers. The experimental data regarding health risks caused by the occupational DEHP exposure will provide the evidence for government and policy makers in reviewing occupationally exposed health and environmental policies.

3.5. DEHP residue in plasma

As shown in Fig. 2C, the surveyed workers had detectable DEHP residue in their plasma, with maximum value of 160 ng L⁻¹. High exposure to DEHP associated with small-sized PVC manufacturing factory had significant high plasma DEHP residue. It was predicted that the high levels of plasma DEHP residues were caused primarily by heavy air contamination (Latini et al., 2003). Plasma is a suitable matrix for health-related monitoring with controlled comparison.

As shown in Fig. 3B, DEHP could be detected in highest amounts in the plasma collected from the exposed workers of the small-sized factory. The profile of plasma DEHP concentrations varied among the sampled areas. Coincidentally, a relationship between the plasma DEHP residue and occupational DEHP air level was observed, yielding a linear curve: Y = 194 + 3.12X (R² = 0.999, p < 0.0001). This finding clearly indicated that higher DEHP air concentration in workforce the workers inhaled would have higher concentration of the DEHP residues in the workers' serum.

3.6. Estimation of DEHP daily intake

Daily intake information is essential to adequately evaluate exposure/response relationships in field study. We can use these relationships to generate credible health risk assessments (Wittassek et al., 2007). Exposure in occupational settings can occur through skin contact and by inhalation of gases and dust. PVC products were manufactured in indoor workspaces. High ambient exposure to DEHP could occur during the incorporation of DEHP into the final product during the operation, especially at an elevated temperature.

Long-term exposure to high dose DEHP may increase human health damage. The respiration is an effective way for DEHP to enter human body, accounting for more than 95% of DEHP daily intake. The inhalation of 90% DEHP was assumed to be absorbed, solely based on the ambient DEHP level. Such an extrapolation does not need to include different elimination factors and elimination half-times depending on the possible significance of the dermal route. Daily intake (DI) can be obtained by Eq. (1).

\[
DI = \frac{C \times V \times 0.9}{BW}
\]

where DI is the daily intake of DEHP (\(\mu g \text{ kg}^{-1} \text{bw} \cdot \text{d}^{-1}\)), C is the ambient DEHP concentration (\(\mu g \text{ m}^{-3}\)), V is the air volume of human daily inhalation (\(m^3 \cdot \text{d}^{-1}\)). For an adult, the average inhalation volume is 4.5 L min⁻¹ or 0.27 m³ h⁻¹. Here, the value for V is reported to be 0.27 × 9 = 2.43 (m³ d⁻¹) when occupationally exposed time is set for 9 h. BW is the body weight of participants, for an adult male, the value for BW is estimated to be 65 kg.

The estimated daily exposures to DEHP by the participants are summarized in Table 3. 9 h-Average inhalation volume of occupational air is 2.43 m³ for adults per workday. We calculated daily DEHP intakes for the investigated workers. The median of

![Fig. 3. Correlation of PChE depression with total antioxidant level in the exposed workers (A); correlation of occupational DEHP air levels with plasma DEHP residues in the exposed workers (B). In each figure, a total of 30 data points has been used, 10 data points for each worksite.](image-url)
estimated DI of DEHP in the exposed group was 13.4 μg kg bw^−1 d^−1 (22.7–9.8–7.8 = 13.4), which was significantly higher than that of the unexposed group (0.0232 μg kg bw^−1 d^−1). Of the 352 exposed workers, only 116 participants (33.0%, 116 = 33.0) at S3 had a maximum DI (22.7 μg kg bw^−1 d^−1), which exceeds the reference dose (RfD) of 20 μg kg bw^−1 d^−1 from DEHP exposure (US EPA, 1981). A possible explanation for this phenomenon is the long-term ingestion of occupational air containing higher dose DEHP.

4. Conclusion

In terms of long-term occupational exposure assessment, a specific polluted episode was investigated. The main conclusions in this study are as follows:

(1) For DEHP, the occupationally exposed values from participated workers exhibit higher doses in workplaces where the workers spend most of the day time. Occupational exposure to DEHP associated with PVC manufacturing was substantially higher than control background exposure.

(2) Using PChE, residues, renal and hepatic biochemical markers, we found strong evidence that high dose DEHP exposure was hazardous to human health. High exposure to DEHP caused significant decrease in PChE levels, elevated oxidative stress, and derangement of hepatic function. The slight high DEHP residue in plasma had been observed among the workers. PChE was significantly and negatively correlated with plasma DEHP residue. The plasma DEHP residue also correlated well with the DEHP levels measured in the air of workplaces.

(3) The workers from small PVC industrial unit experienced more clinical symptoms of central nervous, gastrointestinal, and respiratory systems than the other factories. Headache followed by dizziness, fatigue, nausea, muscle weakness, vomiting, skin rash, and shortness of breath were obvious among these workers. The results clearly indicated that high exposure to environmental DEHP presented a potential health risk to PVC manufacturing workers.

(4) Based on the occupational air values, we simply calculated the daily intakes of DEHP. A median DI, 13.4 μg kg bw^−1 d^−1, was estimated in the occupationally exposed workers. Their maximum average DI for the small industrial workers (33.0% of the whole group) was up to 22.7 μg kg bw^−1 d^−1, which exceeded the RfD of 20 μg kg bw^−1 d^−1.

Based on our findings, we hope to identify key factors likely influencing exposures (e.g. DEHP vapor pressure, work practices and processes, ventilation, enclosures, protective equipment, etc.). More detailed investigation is needed to correspond different exposure conditions with different workers.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.chemosphere.2014.05.053.

References


