Effect of Diesel Oil Vapor Inhalation on Liver Function in Male Rat

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Abstract

Background: Studies show that there is association between air pollution and disturbances on normal function of various systems of body. The main aim of this study was to determine the effects of diesel oil vapor inhalation on serum creatine, alkaline phosphatase, SGOT and SGPT levels as indices of liver function in male rats.

Methods: Thirty two rats were randomly divided into 4 groups: control and three treatment groups that exposed to diesel oil vapor for 1 hour/day, for 2 hour/day and for 3 hours/day. After a period of 6 weeks, blood samples were collected and investigated. Serum level of alkaline phosphatase, creatine, glutamate oxaloacetate transaminase (SGOT) and glutamate pyruvate transaminase (SGPT) were measured by spectrophotometry method.

Results: Our findings indicated that there was no significant difference between serum alkaline level of control and experimental rats. Serum creatine level was significantly increased in rats exposed to diesel oil for 1 hour/day, 2 h/day and 3 h/day (P<0.001, P<0.01 and P<0.001, respectively). Also, SGOT was significantly decreased in rats exposed to diesel oil for 2 h/day and 3 h/day (P<0.01 and P<0.05, respectively). SGPT was non-significantly decreased in rats exposed to diesel oil vapor compared with control animals.

Conclusions: We have shown that exposure to diesel oil vapor can bring about enhanced kinase level, decreased SGPT level indicating the health risk caused by exposure to diesel oil inhalation, in particular, to liver.

Keywords: Diesel oil, CK, Alkaline Phosphatase, SGPT.

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Introduction

Diesel oil is a fuel obtained from petroleum distillation that is used in diesel engines. One of the major sources of atmospheric soot is fumes burning diesel that produce air pollution. The infection can lead to damage to the heart and lungs in humans. Diesel fuel consists of toxic chemicals, including benzene, toluene, ethyl benzene, and xylenes (collectively known as benzene “BTEX” compounds: benzene, toluene, ethylbenzene, and xylene). Diesel exhaust also contains nanoparticles.¹

There are reports indicating that truckers, railroad workers, miners, and other workers use diesel-powered equipment in underground mines.² Breathing fumes and evaporative and refueling emissions bring about serious toxic risks.³

The neurotoxic effects of the inhalation of oil fumes have also been established.⁴ Traffic of congested vehicles leads to reduced ambient air quality. Recent studies have shown outbreaks of illness and death in people who live on the margins of highways.⁵ Headache, fatigue, loss of memory, and dizziness were the common signs observed in subjects exposed to vehicle emissions.⁶,⁷

With regard to creatine kinase (CK), it is known that creatine phosphokinase (CPK) is an enzyme that is expressed in different tissues and cells. This enzyme catalyzes the conversion of creatine to creatine phosphate in the presence of ATP. Enhancement of the creatine kinase plasma level is prevalent in various conditions including heart or skeletal muscle damage.⁸

Serum glutamic oxaloacetic transaminase (SGOT) is a hepatic enzyme that is found in various tissues such as heart, skeletal muscle, kidney, brain, and red blood cells and usually is used as a marker for clinically measured liver health check. Serum glutamic pyruvic transaminase (SGPT) is found in serum and in various bodily tissues but is most commonly associated with the liver.⁹,¹⁰,¹¹ This study was designed to evaluate the effects of diesel oil vapor inhalation on serum creatine level, serum alkaline phosphatase level, SGOT, and SGPT levels as indices of liver function in male rats.

Materials and Methods

This experimental study was performed on Wistar rats (mean weight 30±200). These animals were purchased from the Pasteur Institute in Tehran and housed in stainless steel cages in a ventilated animal room. The room temperature was maintained at 23±2°C on a 7-h light/dark cycle. Distilled water and sterilized food for rats were available ad libitum. All animal handling and manipulation procedures were performed according to the guidelines of the Animal Welfare Act and the experimental protocols were approved by the Office of Research Ethics Committee of the University of Sanandaj. In addition, diesel oil was poured into a test tube of 100 cc and the tube was covered with cotton steam so that diesel oil was not high {1.2 [EN Meaning unclear. Please clarify]} and the tube was put into the rats’ cage.

In total, 32 rats were randomly divided into four groups: The control and three treatment groups that were exposed to diesel oil vapor for 1 h/day, for 2 h/day (500-700 ppm), and for 3 h/day. After a period of 6 weeks, after anesthesia, blood samples were collected from the heart (24 h after the last treatment) and maintained in tubes. After collection of blood samples, the tubes
were incubated for 15 min at room temperature to create blood clots. The sample was centrifuged for 15 min (2500 rpm/min), and sera were separated and transferred into smaller tubes, stored at -20 °C, to measure the activity of the enzyme.

The serum levels of creatinine kinase, alkaline phosphatase, SGOT, and SGPT were measured by spectrophotometry method [using a biochemical autoanalyzer (Hitachi Automatic Analyzer 902, Roche, Germany)]. All animal experiments were conducted in accordance with the guidelines of the institutional animal ethics committee.

All data was analyzed by using the statistical package for social sciences (SPSS-19) software and was summarized and expressed as mean and standard error (mean±SEM). In this study, the one-way ANOVA for comparison of averages, at a significance level of 0.05, was used.

Discussion

Our study indicated diesel oil vapor inhalation results in increased serum creatine levels. In line with our finding, other research findings also indicate breathing the evaporative and refueling emissions brings about serious toxic risks, which in turn may give rise to elevated damage in tissues, including muscular tissue, resulting in increased serum creatine levels. Occupational exposure to diesel oil vapor has also been associated with numerous health risks.

Effects of exposure to smoke oil (fat or oil) on the body are several: Effect on Na+, K+-ATPase, superoxide dismutase, acetylcholine esterase, total protein, reducing of glutathione, and lipid peroxidation in the cerebral cortex. In addition, monoamine neurotransmitters dopamine, norepinephrine, and serotonin in the cerebral cortex, hippocampus, cerebellum, and hypothalamus were established. Because fume oil exposure has effects on ATP-dependent pathways at cellular level, the effects of diesel oil vapor inhalation on creatine kinase are explainable.

In addition, our study indicated diesel oil vapor inhalation results in reduced SGOT levels. Consistent with our finding, other research findings also indicate breathing the evaporative and refueling emissions brings about serious toxic risks, which in turn may give rise to damage in tissues, including the liver, to reduce SGOT and SGPT. The reports also indicate ingestion of diesel and biodiesels can cause mild hepatic peroxisomal proliferation. The impacts of fume oil exposure on Na+, K+-ATPase, superoxide dismutase, and monoamine neurotransmitters dopamine, norepinephrine, and serotonin were also established. This indicated diesel oil inhalation imposes cellular and molecular alterations, by which alteration in tissues, including liver tissue and reduction of liver enzymes, is explainable.

We have shown exposure to diesel oil vapor can bring about enhanced kinase levels and decreased SGPT levels, indicating the health risk caused to the liver, in particular, by exposure to diesel oil inhalation.

Acknowledgment

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Conflict of Interest

The authors have no conflicts of interest to disclose.

References


Results

Table 1 shows serum creatine kinase and alkaline phosphates levels in male rats. Our findings indicate there was no significant difference between the serum alkaline levels of control and experimental rats. Serum creatine level was significantly increased in rats exposed to diesel oil for 1 h/day, 2 h/day, and 3 h/day (P<0.001, P<0.01, and P<0.001, respectively).

Table 2 shows SGOT and SGPT levels in control and rats exposed to diesel oil vapor. Our findings indicate SGPT was non-significantly decreased in rats exposed to diesel oil vapor compared to control animals. SGOT was significantly decreased in rats exposed to diesel oil for 2 h/day and 3 h/day (P<0.01 and P<0.05, respectively); however, there was no significant decrease in SGOT between rats exposed to diesel oil for 1 h/day and control animals.

Table 1. Serum creatine kinase and alkaline phosphates in control and experimental groups

<table>
<thead>
<tr>
<th>Groups</th>
<th>Alkaline phosphatase (IU/L)</th>
<th>P</th>
<th>Creatine kinase (IU/L)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>259.1±19.71</td>
<td>-</td>
<td>39.8±4.81</td>
<td>-</td>
</tr>
<tr>
<td>Diesel oil Vapor Receiving (1 h/day)</td>
<td>181.6±13.25</td>
<td>NS</td>
<td>60.8±4.11</td>
<td>&lt;0.001</td>
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<tr>
<td>Diesel oil Vapor Receiving (2 h/day)</td>
<td>303.8±28.92</td>
<td>NS</td>
<td>52.6±7.16</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Diesel oil Vapor Receiving (3 h/day)</td>
<td>241.1±19.75</td>
<td>NS</td>
<td>56.8±5.40</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

All data expressed as mean and standard error (mean±SEM); P value represents the difference between control and experimental groups. NS indicates no significant difference.

Table 2. Sgot and sgpt in control and experimental groups

<table>
<thead>
<tr>
<th>Groups</th>
<th>SGOT (IU/L)</th>
<th>P</th>
<th>SGPT (IU/L)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>227.4±40.8</td>
<td>-</td>
<td>101.6±29.5</td>
<td>-</td>
</tr>
<tr>
<td>Diesel oil Vapor Receiving (1 h/day)</td>
<td>187.2±24.8</td>
<td>NS</td>
<td>(102.4±19.3</td>
<td>NS</td>
</tr>
<tr>
<td>Diesel oil Vapor Receiving (2 h/day)</td>
<td>140.6±20.1</td>
<td>&lt;0.01</td>
<td>86.6±10.4</td>
<td>NS</td>
</tr>
<tr>
<td>Diesel oil Vapor Receiving (3 h/day)</td>
<td>163.2±32.1</td>
<td>&lt;0.05</td>
<td>81.2±6.3</td>
<td>NS</td>
</tr>
</tbody>
</table>

All data expressed as mean and standard error (mean±SEM); P value represents the difference between control and experimental groups. NS indicates no significant difference.


