

Diesel Oil Vapor Inhalation: Risk Factor for Heart Attack

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Abstract—The studies show that breathing diesel oil vapor impose serious health damage on body systems.. The main aim of this study was to determine the effects of diesel oil vapor inhalation on serum levels of cardiac troponin in male rats. In our study male Wistar rats were randomly divided into control and groups exposed to diesel oil vapor for 1, 4 or 8h/day of 5 rats in each group. After 8 weeks, blood samples were obtained using cardiac puncture method. Following serum preparation, level of cardiac troponin was measured using immunoassay method. Data were statistically analyzed and compared between groups using ANOVA. The results indicated that serum level of cardiac troponin increased in rats exposed to diesel oil vapor for 4 and 8h/day compared to control rats ($P < 0.001$ and $P < 0.05$, respectively). Our findings show that exposure to diesel vapor for long period of time enhances serum cardiac troponin level which may indicate damage to heart.

Keywords—Diesel oil vapor, Cardiac Troponin, Male Rat.

I. INTRODUCTION

GASOLINE is a liquid fuel intended for use in spark-ignition, internal combustion engines and the U.S., gasoline production in 2013 was the highest on record (API, 2013)[1] and it is a blended product (i.e., mixture), which is not listed on the Toxic Substances Control Act (TSCA) Chemical Inventory. It is typically composed of hundreds of paraffinic, olefinic, naphthenic and aromatic hydrocarbons (generally referred to as PONA) refined from petroleum (crude oil) in the C4–C12 carbon-chain length range (API, 2008)[2] and boiling points in the range of 30–220 °C. In addition to the hydrocarbon base, gasoline also can contain a variety of blending components, such as oxygenates (e.g., alcohols, ethers). During gasoline manufacture, crude oil is fractionated, the fractions are chemically modified, and resulting refinery process “streams” are blended to meet specific physical and chemical property requirements (e.g., octane rating, sulfur limits, oxygen content, etc.) [3]. The 1990 amendments to the Clean Air Act (CAA) mandated the use of oxygenates in motor gasoline. In 1994, the U.S. Environmental Protection Agency (EPA) issued a final rule under the Act which added new health effects information and testing requirements to the Agency’s existing registration requirements. As described in more detail in a companion paper (Henley et al., 2014) [4], requirements

include inhalation exposures to evaporative emissions of the gasoline or additive in question. The health endpoints include assessments for standard subchronic toxicity, neurotoxicity, genotoxicity, immunotoxicity, developmental and reproductive toxicity, and chronic toxicity/carcinogenicity. The results of chronic toxicity testing of gasoline and gasoline combined with MTBE have already been reported[5] and reported elsewhere in this issue are the findings for genotoxicity [6], neurotoxicity [7], immunotoxicity [8], reproductive toxicity [9], and developmental toxicity testing in mice and rats [10],[11]. Key differences between whole gasoline and the vaporized gasoline are the significantly greater concentration of C4 and C5 constituents and depletion of C7–C12 aromatic constituents in the vapor condensate. The equilibrium vapor and vapor condensate are also less complex and have a lower average molecular weight and specific gravity [4]. Human studies of both short- and long-term exposures to combustion emissions and ambient fine particulate air pollution have been associated with measures of genetic damage. Long-term epidemiologic studies have reported an increased risk of all causes of mortality, cardiopulmonary mortality, and lung cancer mortality associated with increasing exposures to air pollution. Adverse reproductive effects (e.g., risk for low birth weight) have also recently been reported in Eastern Europe and North America [12].

In vertebrate striated skeletal and cardiac muscle, contraction is regulated by changes in the Ca^{2+} concentration inside the cell. These changes are sensed by the troponin (Tn) complex and transmitted to the other components of the contractile unit. The troponin complex is formed by three subunits, troponin C (TnC), troponin I (TnI), and troponin T (TnT). TnC is an EF-hand protein that can bind four Ca^{2+} ions and is responsible for sensing the increase in calcium concentration. Two of its EF-hand sites, located in the C-domain, are high-affinity sites, capable of binding both Ca^{2+} and Mg^{2+} . These sites are occupied both during contraction and in the relaxed state. The other two EF-hand sites, located in the N-domain, are Ca^{2+} -specific and have lower affinity, being occupied when the intracellular Ca^{2+} levels rise during contraction, and empty in the relaxed state. This change in occupancy changes the interactions between TnC and TnI, and these conformational changes are transmitted to the rest of the thin filament, regulating muscle contraction. The only interaction between TnC and TnI is the structural interaction between the C-domain of TnC and the N-terminal region of TnI. When Ca^{2+} levels inside the cell rise, the N-domain of TnC interacts with the “switch” region of TnI (residues 116–131 in skeletal TnI), removing the interactions between

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TnI and actin and, consequently, the inhibition [13]. The cardiac isoforms of TnC and TnI differ significantly from skeletal isoforms. In the cardiac isoform of TnC, Ca²⁺ binding site I is naturally inactive and was found to undergo chemical exchange consistent with an equilibrium between 'closed' and 'open' forms. Calcium binding to the isolated cTnC regulatory domain was shown not to induce a structural opening similar to that seen in the skeletal isoform [13]-[14].

II. MATERIAL AND METHODS

A. Animals

Adult Wistar rats weighting 200±30g were purchased and raised in our colony from an original stock of Pasteur institute (Tehran, Iran). The temperature was at 23±2 °C and animals kept under a schedule of 12h light:12h darkness (light on at: 08: 00 a.m.) with free access to water and standard laboratory chow. This study was performed according to ethical guidelines relating to working with laboratory animals.

B. Protocol of Study

Male Wistar rats were randomly divided into control and groups exposed to diesel oil vapor for 1, 4 or 8h/day of 5 rats in each group. After 8 weeks, blood samples were obtained using cardiac puncture method. Following serum preparation, level of cardiac troponin was measured using immunoassay method.

C. Statistical Analysis

All values are presented as mean ± S.E.M. Statistical significance was evaluated by one-way analysis of variance (ANOVA) using SPSS 19. Differences with P<0.05 were considered significant.

III. RESULTS

Table I and Figure I show the serum levels of Troponin C in male rats. The results indicated that serum level of cardiac troponin increased in rats exposed to diesel oil vapor for 4 and 8h/day compared to control rats (P<0.001 and P<0.05, respectively).

TABLE I

SERUM LEVEL OF TROPONIN C IN CONTROL ANIMALS AND RATS EXPOSED TO DIESEL OIL VAPOR FOR 1, 4 AND 8H/DAY. P VALUES ARE VERSUS CONTROL GROUP. NS INDICATES NONSIGNIFICANT DIFFERENCE COMPARED TO CONTROL GROUP.

Group	Serum Troponin (ng/dl)	P
Control	1.92±0.40	-
1h/day	2.06±0.32	NS
4h/day	3.53±0.18	<0.001
8h/day	3.00±0.10	<0.05

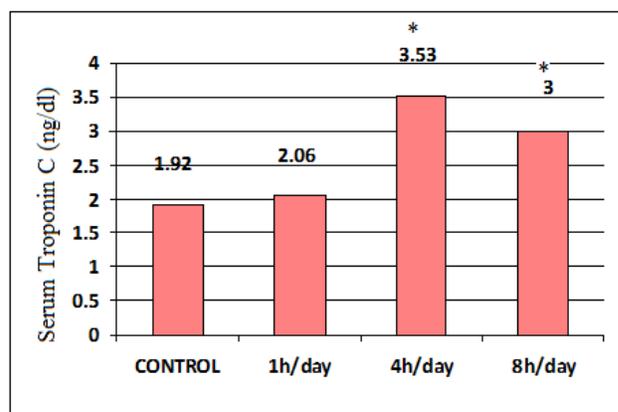


Fig. 1 Serum level of troponin C in control animals and rats exposed to diesel oil vapor for 1, 4 and 8h/day. * represents significant difference compared to control rats.

IV. DISCUSSION

Our study indicated that diesel oil vapor inhalation results in increased serum levels of troponin C.

Other studies have shown an association between air pollution and diseases. It has been suggested a possible association between chronic low level benzene exposure and increased risk of leukemia. It has also been shown that air pollution is associated with significant public health impacts, with cardiovascular disease being a prominent outcome [15]-[16]. Studies also suggest that indoor air pollution from use of solid fuel is an important cause of acute coronary syndrome [17]. It has been reported that incorporation of L48Q cTnC significantly increased contractility of cardiomyocytes from healthy and MI hearts without adversely affecting Ca²⁺ transient properties or relaxation. However, some studies have shown that unleaded gasoline vapors did not produce evidence of developmental toxicity [19].

V. CONCLUSION

Our findings show that exposure to diesel vapor for long period of time enhances serum cardiac troponin level which may indicate damage to heart.

ACKNOWLEDGMENT

This research has been done with the support of Islamic Azad University, Hamedan Branch, Hamedan, Iran. We appreciate all who helped us to exert the present study.

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