Biological effects of crude oil vapor. IV. Cardiovascular effects

Introduction

According to the North American Industry Classification System, workers employed in the oil and gas extraction industry are working in one of three areas; 1) extraction, 2) drilling, or 3) processing and refinement (Bureau of Labor Statistics, April 15, 2014). Both workers and people living in communities that are in close proximity to areas where gas and oil extraction and refinement occur have a higher incidence of respiratory, cardiovascular, digestive, reproductive and nervous system disorders,
The goal of these studies was to identify mechanisms underlying the development of cardiovascular disease after inhalation of crude oil vapors (COV). The oil used for this study was a surrogate of the crude oil that leaked into the Gulf of Mexico after the Deepwater Horizon oil spill (McKinney *et al.*, 2021). We tested the hypothesis that changes in cardiovascular function after inhalation of COV are manifested as changes in the responsiveness of the heart and peripheral vascular system to vasoconstricting and vasodilating agonists. Based on previous studies, we also predicted that inhalation of COV would affect the expression of biomarkers of cardiac and kidney function that are associated with blood pressure regulation and disease.

Citations

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Methods

Animals. Male Sprague-Dawley were housed in pairs under controlled light cycle (12 h light/12 h dark) and temperature (22 – 25 °C) conditions and were provided tap water and food *ad libitum*.

Exposure system. The exposure system used to generate COV is described in McKinney *et al.* (2021). In Experiment 1, rats were exposed to a single (acute), 6-h exposure to COV (300 ppm) or filtered air in a whole-body inhalation system and euthanized 1 or 28 d following the exposure. In Experiment 2, rats were exposed to filtered air or 300 ppm COV 6 h/d × 4 d/wk × 4 wk (sub chronic) and euthanized 1, 28 or 90 d following the exposure. After each 6 h exposure, the animals were returned to the colony room. In experiment 1, animals were euthanized 1 or 28 d after exposure and in Experiment 2 they were euthanized 1, 28 or 90 d after exposure.

Measures. A detailed description of the methods can be found in the link below. To measure the effects of the exposures on cardiovascular function, vascular and cardiac responses to adrenergic agonists were analyzed. To determine mechanisms that may underlie change in cardiovascular function, reactive oxygen species were measured using ELISAs for nitrate/nitrite or hydrogen peroxide, or by measuring immunofluorescence in heart and kidney tissues. Quantitative RT-PCR was used to measure transcripts associated with vascular and renal function. Protein arrays were also used to determine if proteins associated with vascular or renal disease were altered in response to the exposure.

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Contact Information: Physical Effects Research Branch, Health Effects Laboratory Division, NIOSH Morgantown. Phone: 304-285-6230.