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Environmental stress in the Gulf of Mexico and its potential impact on public health



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ABSTRACT

The Deepwater Horizon (DWH) oil spill in the Gulf of Mexico was the largest maritime oil spill in history resulting in the accumulation of genotoxic substances in the air, soil, and water. This has potential far-reaching health impacts on cleanup field workers and on the populations living in the contaminated coastal areas. We have employed portable airborne particulate matter samplers (SKC Biosampler Impinger) and a genetically engineered bacterial reporter system (umu-ChromoTest from EBPI) to determine levels of genotoxicity of air samples collected from highly contaminated areas of coastal Louisiana including Grand Isle, Port Fourchon, and Elmer's Island in the spring, summer and fall of 2011, 2012, 2013 and 2014. Air samples collected from a non-contaminated area, Sea Rim State Park, Texas, served as a control for background airborne genotoxic particles. In comparison to controls, air samples from the contaminated areas demonstrated highly significant increases in genotoxicity with the highest values registered during the month of July in 2011, 2013, and 2014, in all three locations. This seasonal trend was disrupted in 2012, when the highest genotoxicity values were detected in October, which correlated with hurricane Isaac landfall in late August of 2012, about five weeks before a routine collection of fall air samples. Our data demonstrate: (i) high levels of air genotoxicity in the monitored areas over last four years *post* DWH oil spill; (ii) airborne particulate genotoxicity peaks in summers and correlates with high temperatures and high humidity; and (iii) this seasonal trend was disrupted by the hurricane Isaac landfall, which further supports the concept of a continuous negative impact of the oil spill in this region.

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1. Introduction

The 2010 Deepwater Horizon (DWH) oil spill in the Gulf of Mexico is the largest environmental disaster and remediation effort in the U.S. history. Approximately 800 million liters of crude oil were spilled and about 7 million liters of chemical dispersant were applied along the northern Gulf of Mexico, resulting in over 1600 km of the shoreline contaminated with weathered oil (Barron, 2012; Kleindienst et al., 2015). The closing of almost 50 million acres to fishing, hunting, and tourism, displacement of residents, and the arrival of workers recruited to participate in

mitigation efforts, all contributed to the overall impact of the spill (Barron, 2012; Fisher et al., 2014). A large number of governmental and non-governmental agencies were involved in environmental relief efforts and interventions to alleviate both the environmental and economic impacts of the spill. Since the beginning of the spill, cleanup workers have been reporting acute symptoms of sickness including skin and eye irritation, respiratory problems, and headaches (D'Andrea and Reddy, 2013; King and Gibbins, 2011; Rotkin-Ellman et al., 2010). In addition, long-term health problems are also anticipated, however a direct cause and effect relationship between chronic exposure to weathered oil and health require further investigation (Diaz, 2011). Multiple chemicals and physical factors associated with the oil spill are known to cause both acute and chronic health problems. These chemicals include volatile organic compounds (VOCs): benzene, toluene, xylene, and ethyl

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benzene; semi-volatile compounds: polycyclic aromatic hydrocarbons (PAHs), higher molecular weight alkanes, and particulate matter (PM 2.5 and PM 10.0). In addition oil mist, carbon monoxide, gasoline and diesel engine exhausts were all present during the cleanup efforts (McCoy and Salerno, 2010).

Importantly, there was an increase of over 10-times in the concentration of the relatively low molecular weight, volatile components, including naphthalene and C-1-naphthalenes, and in higher molecular weight analytes between September 2010 and October 2012 in heavily oiled marsh in Louisiana (Turner et al., 2014). Although these volatile components are well known to trigger both immune responses, and cause DNA damage in affected organisms including humans (Alpha et al., 2015; Tagiyeva and Sheikh, 2014; Wang et al., 2013; Wang et al., 2014), air sampling and the evaluation of airborne particulates have not been used in the assessment of health risks associated with oil spills. Our research team collected and analyzed airborne particulate matter (PM) samples from three areas of coastal Louisiana, which according to the U.S. Environmental Protection Agency (EPA) demonstrated heavy contamination with the natural crude oil post DWH oil spill. These include Grand Isle, Port Fourchon, and Elmer's Island. The PM samples were collected in the spring, summer and fall of 2011, 2012, 2013 and 2014. Our data demonstrate: (i) persistent air genotoxicity in the monitored areas of coastal Louisiana over the last four years; (ii) air genotoxicity peaks in the summers, which correlated with high temperatures and high air humidity; and (iii) this seasonal trend was disrupted by the hurricane Isaac landfall, which caused a further increase of air genotoxicity in the fall of 2012. In conclusion, our data indicate a continuous negative health impact of DWH oil spill, which persists in the monitored regions of the Gulf, and supports the need for a continuous monitoring of air quality in this region.

2. Materials and methods

2.1. Air sample collection and storage

BioSampler Impinger (SKC, Inc.) with sonic flow sample pumps were used according to manufacturer recommendations. Briefly, particulate matter (PM) recovered from size-segregated air samples were collected from oil-impacted beaches at Port Fourchon, Elmer's Island, and Grand Isle, Louisiana. These air samples were collected during 8-h sampling campaigns, which occurred on consecutive days over weekends in March, July, and October of 2011, 2012, 2013 and 2014. Airborne PM samples collected from a non-contaminated area (Sea Rim State Park, TX) were used as controls. The samples were then stored at -80°C until further analyses. The selection of oil impacted sites was made by using air quality data gathered through the U.S. Environmental Protection Agency (EPA) in response to the DWH oil spill to identify the most heavily impacted regions in the Gulf (2011 United States Environmental Protection Agency (EPA, 2011)). Permits were acquired from the Louisiana State Park Service to collect samples in Grand Isle State Park, one of the most adversely affected coastal shorelines of Louisiana.

2.2. Genotoxicity of air samples

A standard bacterial reporter assay (umu-ChromoTest from EBPI) was used to determine the genotoxicity of the collected PM samples. In this assay, DNA damage induces activity of the reporter gene, umuC, as a part of genotoxic response. In this system, the umuC gene is fused to lacZ, thus its activation results in the production of β -galactosidase, measured using a standard colorimetric reaction and UV-vis plate reader (FilterMax F5, Molecular

Devices). The results are expressed as Induction Ratios, which were calculated using the formula recommended by the manufacturer in which the positive control is the response to the known genotoxic compound [4-nitroquinoline 1-oxide (4NQO)], and the negative control is the response to the elution buffer. Accordingly, the Induction Ratio of 1.5 was determined as the baseline for the genotoxicity. In particular, a series of dilutions of the air samples were tested to select a dilution, which is not cytotoxic to bacteria, and still triggers high genotoxic response.

2.3. Meteorological data

Meteorological data were logged (ACU > RITE Professional Weather Center-5-in-1 wireless sensor) at the collection sites during PM sampling campaigns. The data include: temperature (F), humidity (%), wind direction, wind speed (mph), heat index (F), dew point (F), and wind chill (F).

2.4. Statistical analysis

Statistical evaluation was based on one-way ANOVA with Satterthwaite correction for unequal variances, followed with multiple comparisons of mean Induction Ratios with p-values adjusted via simulation to keep an overall alpha of ≥ 0.05 .

3. Results

Large-scale oil spills increase the overall concentration of toxic chemicals in affected environments. Some of these substances, such as volatile organic compounds (VOCs) and semi-volatile polycyclic aromatic hydrocarbons (PAHs), can be released from water and sediments affecting the quality of air in areas heavily contaminated with the natural crude oil. To address the potential health risk associated with chronic exposure to natural crude oil, we collected air samples from coastal areas polluted by the 2010 DWH oil spill in the Gulf of Mexico. Fig. 1 shows the locations of three collection sites, which are superimposed on the map of relative oiling of the Louisiana coastline as of January 23, 2011 (Jan 23 2011 Environmental Response Management Application[®] (ERMA)) derived as part of the Shoreline Cleanup Assessment Technique (SCAT) Program (ERMA, 2011). The samples were collected from Grand Isle (A), Port Fourchon (B), and Elmer's Island (C) in spring, summer and fall of 2011, 2012, 2013 and 2014 using portable PM samplers (SKC Biosampler Impinger). The collected samples were stored in -80°C , and were subsequently tested for the ability to induce a genotoxic response in genetically engineered bacterial reporter system (umu-ChromoTest from EBPI). PM samples collected from a non-contaminated area (Sea Rim State Park, TX) served as a control for background genotoxicity. The results in Fig. 2 demonstrate that samples collected from Grand Isle in March, July and October of 2011 demonstrated 443%, 570%, and 315% increases in genotoxic response (expressed as Induction Ratio) in comparison to the corresponding control samples. Significant increases in genotoxicity were also detected in samples from Elmer's Island and Port Fourchon, LA. Genotoxicity analysis of samples from Elmer's Island in 2011 demonstrated a 356% increase in March, 440% increase in July, and 264% increase in October. Analysis of samples collected at Port Fourchon demonstrated a 198% increase in March; 397% increase in July; and 223% increase in October (all statistically significant).

Additionally, we also observed significantly higher genotoxicity values in July 2011 in comparison to March and October of the same year in all three locations. The results in Fig. 3A demonstrate that combined genotoxicity levels (from all three Louisiana locations) were 24% higher in July in comparison to March ($p=0.048$);

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