

Health Consequences among Subjects Involved in Gulf Oil Spill Clean-up Activities

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ABSTRACT

BACKGROUND: Oil spills are known to affect human health through the exposure of inherent hazardous chemicals such as para-phenols and volatile benzene. This study assessed the adverse health effects of the Gulf oil spill exposure in subjects participating in the clean-up activity along the coast of Louisiana.

METHODS: This retrospective study included subjects that had been exposed and unexposed to the oil spill and dispersant. Using medical charts, clinical data including white blood cell count, platelets count, hemoglobin, hematocrit, blood urea nitrogen, creatinine, alkaline phosphatase (ALP), aspartate amino transferase (AST), alanine amino transferase (ALT), and somatic symptom complaints by the subjects were reviewed and analyzed.

RESULTS: A total of 247 subjects (oil spill exposed, $n = 117$ and unexposed, $n = 130$) were included. Hematologic analysis showed that platelet counts ($\times 10^3$ per μL) were significantly decreased in the exposed group compared with those in the group unexposed to the oil spill (252.1 ± 51.8 vs 269.6 ± 77.3 , $P = .024$). Conversely, the hemoglobin (g per dL) and hematocrit (%) levels were significantly increased among oil spill-exposed subjects compared with the unexposed subjects ($P = .000$). Similarly, oil spill-exposed subjects had significantly higher levels of ALP (76.3 ± 21.3 vs 61.2 ± 26.9 IU/L, $P = .000$), AST (31.0 ± 26.3 vs 22.8 ± 11.8 IU/L, $P = .004$), and ALT (34.8 ± 26.6 vs 29.8 ± 27 IU/L, $P = .054$) compared with the unexposed subjects.

CONCLUSION: The results of this study indicate that clean-up workers exposed to the oil spill and dispersant experienced significantly altered blood profiles, liver enzymes, and somatic symptoms.

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KEYWORDS: Blood disorders; Chemical exposure; Crude oil spill; Dispersants; Health impact; Hematological toxicity; Hepatotoxicity; Somatic symptoms; Urinary phenol

Crude oil spills affect human health through exposure to inherent hazardous chemicals including para-phenols and volatile benzene.¹⁻³ The major health consequences of crude oil spill exposure include the abnormalities in hematologic, hepatic, respiratory, renal, and neurologic functions.^{4,5} In addition, subjects exposed to oil spills often experience frequent asthmatic attacks, headache, diarrhea, dizziness, abdominal pain, back pain, and other symptoms.^{4,6-9}

On April 20, 2010, the British Petroleum (BP) *Deepwater Horizon* offshore drilling rig located 50 miles off the

Louisiana coast exploded and sank in the Gulf of Mexico (**Figure 1**).^{5,10} Consequently over 200 million gallons of oil poured into the Gulf of Mexico,¹¹ thereby contaminating the Gulf coast. During the height of this disaster, BP used nearly 2 million gallons of dispersants such as COREXIT¹² (Nalco Energy Services, L.P., Sugar Land, Tex) to break down the oil slick.¹¹ This oil spill and use of massive amounts of dispersant has the potential to affect human health. It is estimated that up to 170,000 people worked in some capacity to clean up the Gulf oil spill.¹³

Previously, several studies have evaluated the health impact of other oil spills.^{5,6,10,14-16} These studies primarily focused on physical effects and psychological sequelae. In addition, these studies point to potential adverse effects among oil spill clean-up workers. Earlier studies reported that benzene exposure is associated with hematological toxicity and increased cancer risk.¹⁷⁻²² While such rare adverse outcomes may take years to develop, immediate health effects of

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oil spill exposure may be seen in hematological and hepatic parameters, indicating its toxic effects and potential for future health risk.²³

To investigate the adverse health effects of the oil spill in the Gulf of Mexico, a retrospective analysis was performed on subjects participating in the oil spill clean-up activity along the coast of Louisiana. Specifically, we assessed the hematologic and hepatic markers in a cohort of oil spill clean-up workers, and the clinical findings were compared with the unexposed (control) subjects.

MATERIALS AND METHODS

Subjects

This study was approved by an Institutional Review Board. Using medical charts, demographic and clinical data were reviewed for the subjects who underwent clinical and laboratory evaluation between January 2010 and November 2012. The study was conducted according to the principles of the Declaration of Helsinki. The personal information of the subjects was redacted to maintain confidentiality.

Subjects included in this study were referred to the clinic for medical evaluation by the subjects' legal representatives. Written consent was obtained from the subjects' legal representatives. The subjects exposed to the oil spill and dispersant were identified as participants in the oil spill clean-up activities along the coast of Louisiana for a duration of over 3 months.

The unexposed subjects were living geographically at least 100 miles away from the Gulf coast of Louisiana. The unexposed subjects had visited the clinic for a routine wellness check-up. The subjects were selected randomly for the study by their primary care physicians.

Chart Review and Evaluation

Medical charts of exposed and unexposed subjects were reviewed by expert physicians and the data were processed for statistical analysis. Clinical data such as white blood cell (WBC) counts, platelet counts, hemoglobin, hematocrit, blood urea nitrogen (BUN), creatinine, serum beta-2-microglobulin, alkaline phosphatase (ALP), aspartate amino transferase (AST), and alanine amino transferase (ALT) levels were evaluated. Data on urinary phenol also was assessed as a benzene metabolite in oil spill-exposed subjects. Additionally, data on somatic symptoms were collected from the oil spill-exposed subjects and analyzed.

Statistics

Descriptive statistics were used to assess patient demographics and included means and standard deviations for

each group. Variables included were WBC, platelets, hemoglobin, hematocrit, creatinine, BUN, ALP, AST, ALT, beta-2 microglobulin, and urinary phenol. Student's *t* test was used to assess the differences between exposed and unexposed groups. The significance level was predetermined at an alpha level of .05.

CLINICAL SIGNIFICANCE

- Human exposure to oil spill and dispersant use has a potential to alter both hematological and hepatic profiles.
- The hematological and hepatic alterations include decreased platelets and BUN, and increased creatinine, serum levels of ALP, AST, and ALT.
- The most reported somatic symptoms are headache, shortness of breath, skin rash, cough, fatigue, painful joints, and chest pain.

RESULTS

This study included a total of 247 subjects, 117 of which were involved in the clean-up activity of the oil spill. The outcomes of the oil spill-exposed subjects were evaluated and compared with the unexposed subjects ($n = 130$). The subjects' demographics are shown in **Table 1**. Among the oil spill-exposed subjects ($n = 117$), there were 104 (89%) males and 13 (11%) females. The median age of exposed and unexposed subjects was 34.0 (18-63) and 51.0 (15-90) years, respectively. The

demographic characteristics such as sex and age groups differed significantly between the exposed and unexposed groups ($P = .000$).

The data in **Table 2** represents the differences in hematologic and hepatic markers between the subjects exposed to the oil spill and those unexposed. No significant differences were observed in the WBC count ($\times 10^3$ per μL) among those exposed to the oil spill and those unexposed (6.9 ± 1.9 vs 6.5 ± 1.9 , $P = .11$). However, the platelet count ($\times 10^3$ per μL) in subjects exposed to the oil spill was significantly decreased compared with the unexposed subjects (252.1 ± 51.8 vs 269.6 ± 77.3 , $P = .024$).

The hemoglobin (g per dL) levels were significantly increased among oil spill-exposed subjects compared with the unexposed subjects (14.9 ± 1.3 vs 13.6 ± 1.6 , $P = .000$). The hematocrit levels were significantly elevated in oil spill-exposed subjects compared with the unexposed subjects (44.6 ± 3.4 vs 40.8 ± 5.0 , $P = .000$). Similarly, serum creatinine levels (mg/dL) also were significantly higher in the oil spill-exposed subjects compared with the unexposed subjects (1.0 ± 0.2 vs 0.9 ± 0.3 , $P = .000$). Conversely, the BUN (mg/dL) significantly decreased in the oil spill-exposed subjects compared with the unexposed subjects (13.4 ± 3.4 vs 15.4 ± 7.1 , $P = .014$).

Compared with the unexposed subjects, subjects exposed to the oil spill showed significantly elevated levels of ALP (76.3 ± 21.3 vs 61.2 ± 26.9 IU/L, $P = .000$). The AST (IU/L) levels were significantly higher in the oil spill-exposed subjects compared with the unexposed subjects (31.0 ± 26.3 vs 22.8 ± 11.8 , $P = .004$). Similarly, the ALT (IU/L) levels were marginally but significantly increased in the oil spill-exposed group compared with the unexposed group (34.8 ± 26.6 vs 29.8 ± 27 , $P = .054$).

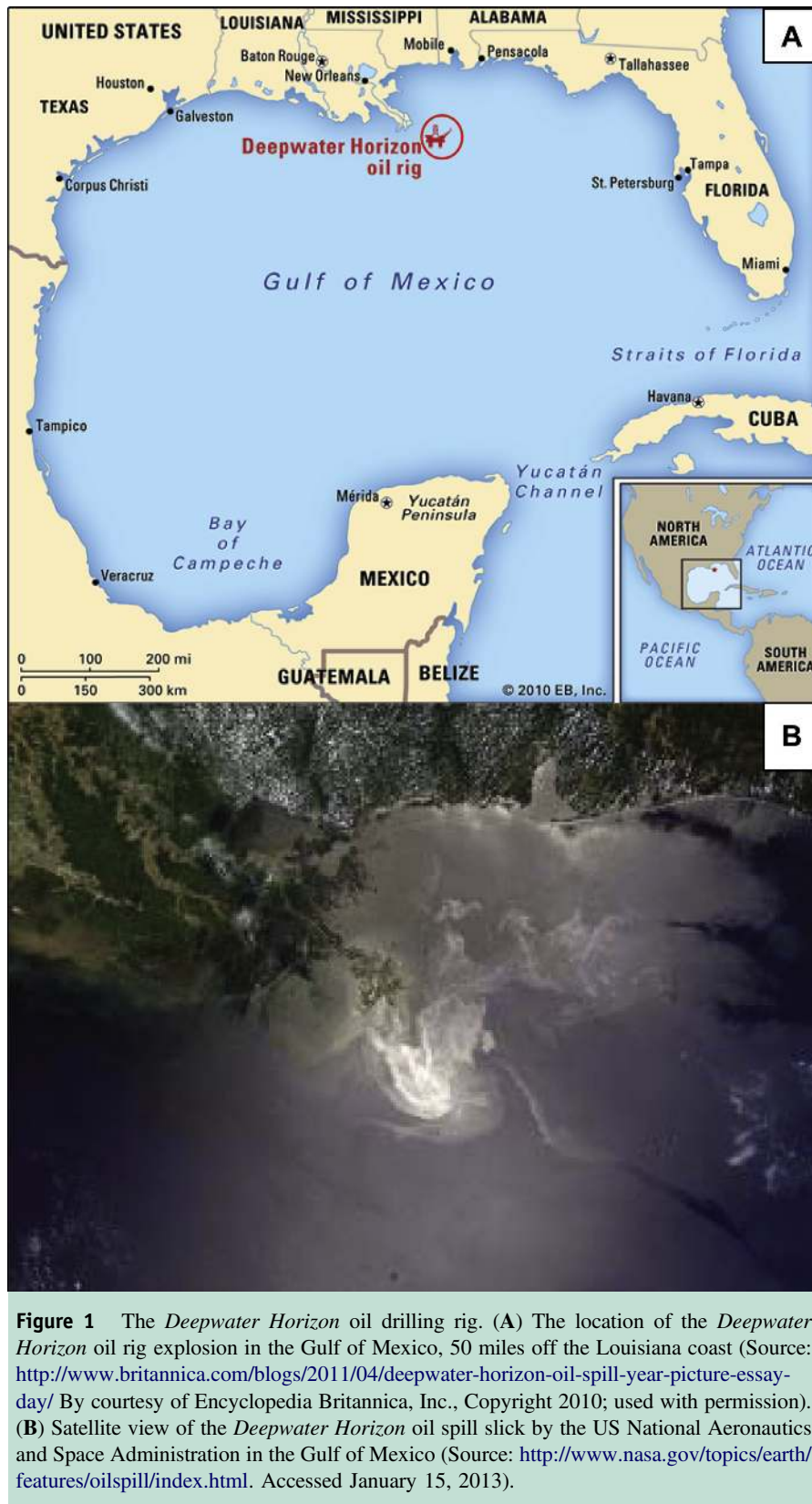


Figure 1 The *Deepwater Horizon* oil drilling rig. (A) The location of the *Deepwater Horizon* oil rig explosion in the Gulf of Mexico, 50 miles off the Louisiana coast (Source: <http://www.britannica.com/blogs/2011/04/deepwater-horizon-oil-spill-year-picture-essay-day/> By courtesy of Encyclopedia Britannica, Inc., Copyright 2010; used with permission). (B) Satellite view of the *Deepwater Horizon* oil spill slick by the US National Aeronautics and Space Administration in the Gulf of Mexico (Source: <http://www.nasa.gov/topics/earth/features/oilspill/index.html>. Accessed January 15, 2013).

The results in **Table 3** show the differences in hematologic and hepatic markers between exposed and unexposed subjects according to their age group. Specifically, subjects were grouped into <40-year and ≥ 40 -year age groups, and

clinical outcomes were compared. No differences were noted in the WBC count ($\times 10^3$ per μL) between unexposed and exposed subjects in the <40-year age group; however, the WBC counts ($\times 10^3$ per μL) were significantly higher in

Table 1 Demographics and Other Characteristics of the Subjects Included in the Study

Demographics	Unexposed	Exposed	P Value
Total subjects	130 (100%)	117 (100%)	-
Sex			.000*
Male	43 (33%)	104 (89%)	
Female	87 (67%)	13 (11%)	
Age, years			.000*
Mean	49.8	35.8	
Median	51	34	
Range	15-90	18-63	
Age group, years			.000*
<20	4 (3%)	0 (0%)	
≥20-<30	13 (10%)	31 (27%)	
≥30-<40	12 (9%)	42 (36%)	
≥40-<50	28 (22%)	35 (30%)	
≥50-<60	41 (32%)	8 (7%)	
≥60-<70	20 (15%)	1 (1%)	
≥70	12 (9%)	0 (0%)	

*Statistically significant at $P = .001$.

oil spill-exposed subjects compared with the unexposed subjects in the ≥ 40 -year age group (7.5 ± 2.0 vs 6.5 ± 1.9 , $P = .007$). Similarly, the platelet counts ($\times 10^3$ per μL) were significantly higher in oil spill-exposed subjects compared with the unexposed subjects in the ≤ 40 -year age group (283.6 ± 67.3 vs 251.8 ± 51.7 , $P = .006$). However, no differences were noted in the platelet counts between exposed and unexposed subjects in the < 40 -year age group. Hemoglobin (g per dL) and creatinine (mg per dL) were increased significantly in oil spill-exposed subjects compared with the unexposed subjects, irrespective of age groups. Similarly, the serum levels of hepatic enzymes (ALP, AST, and ALT) were increased significantly in oil

Table 2 Comparison of Hematologic and Hepatic Indices between the Unexposed and Exposed Subjects to Oil Spill

Variable	Unexposed (n = 130)	Exposed (n = 117)	P Value
WBC ($\times 10^3$ per μL)	6.5 ± 1.9	6.9 ± 1.9	.108†
Platelets ($\times 10^3$ per μL)	269.6 ± 77.3	252.1 ± 51.8	.024*
Hemoglobin (g per dL)	13.6 ± 1.6	14.9 ± 1.3	.000**
Hematocrit (%)	40.8 ± 5.0	44.6 ± 3.4	.000**
BUN (mg per dL)	15.4 ± 7.1	13.4 ± 3.4	.014*
Creatinine (mg per dL)	0.9 ± 0.3	1.0 ± 0.2	.000**
ALP (IU per L)	61.2 ± 26.9	76.3 ± 21.3	.000**
AST (IU per L)	22.8 ± 15.1	31.0 ± 26.3	.004**
ALT (IU per L)	29.8 ± 27.0	34.8 ± 26.6	.054*

ALP = alkaline phosphatase; ALT = alanine amino transferase; AST = aspartate amino transferase; BUN = blood urea nitrogen; WBC = white blood cells.

* $P = 0.05$.

** $P = 0.001$.

†Did not reach statistical significance.

spill-exposed subjects compared with those unexposed subjects, irrespective of age groups (Table 3).

The results in Table 4 indicate the differences in hematologic and hepatic markers between the unexposed subjects and those exposed to the oil spill according to sex. No differences were noted in WBC counts ($\times 10^3$ per μL) between unexposed and exposed male subjects; however, female subjects in the oil spill-exposed group had significantly elevated WBC counts ($\times 10^3$ per μL) compared with the unexposed group (7.5 ± 2.2 vs 6.4 ± 1.7 , $P = .022$). No significant differences were detected by gender in the platelet counts between the unexposed and exposed subjects to the oil spill. Males in the oil spill-exposed group had significantly higher hemoglobin (15.1 ± 1.5 vs 14.6 ± 1.3 g/dL, $P = .022$) and hematocrit (45.1 ± 3.2 vs $42.9 \pm 5.4\%$, $P = .000$) levels compared with the males in the unexposed group. BUN (mg per dL) was significantly decreased in males in the oil spill-exposed group compared with those in the unexposed group (13.4 ± 3.4 vs 17.7 ± 9.3 , $P = .000$).

The serum ALP levels (IU/L) were significantly elevated in male (76.9 ± 21.0 vs 64.3 ± 18.0 , $P = .018$) and female (71.3 ± 23.1 vs 62.2 ± 16.1 , $P = .048$) subjects in the oil spill-exposed group compared with the unexposed group. Similarly, the serum levels of AST and ALT (IU/L) were significantly elevated in both male and female subjects in the oil spill-exposed group compared with the unexposed group (Table 4).

The results in Table 5 represent the hematologic and hepatic changes according to the age group in oil spill-exposed subjects. The WBC ($\times 10^3$ per μL) counts were significantly lower in the 20-29-year age group compared with the 30-39-year (6.2 ± 1.6 vs 6.8 ± 1.8 , $P = .02$) or over-40-year age group (6.2 ± 1.6 vs 7.5 ± 2.0 , $P = .001$). The WBC ($\times 10^3$ per μL) counts were significantly lower in the 30-39-year age group compared with the over-40-year age group (6.8 ± 1.8 vs 7.5 ± 2.0 , $P = .08$). Although no differences were seen in beta-2-microglobulin (mg/L) levels between the 20-29-year and 30-39-year age groups, the 20-29-year age group had significantly lower levels of beta-2-microglobulin compared with the over-40-year age group (1.3 ± 0.4 vs 1.4 ± 0.3 , $P = .02$). Similarly, the 30-39-year age group had significantly lower levels of beta-2-microglobulin compared with the over-40-year age group (1.3 ± 0.4 vs 1.4 ± 0.3 , $P = .05$).

The principal somatic symptoms complained about by the oil spill clean-up workers are illustrated in Figure 2. Headache (77%) was the most frequently reported symptom, followed by shortness of breath (71%), skin rash (59%), cough (51%), dizzy spells (51%), fatigue (50%), painful joints (49%), night sweats (41%), and chest pain (38%).

The findings presented in Table 6 reveal the occurrence of somatic symptoms by sex among oil clean-up workers. Males had a lower frequency of headaches compared with females following exposure to the oil spill (75% vs 92%). Similarly, males experienced a lower frequency of painful joints (46% vs 69%), night sweats (38% vs 62%), ringing in the ears (32% vs 69%), fatigue (48% vs 62%), memory loss (20% vs 46%), and diarrhea (26% vs 38%) compared with

Table 3 Comparison of Hematologic and Hepatic Indices by Age Group between Subjects Unexposed and Exposed to the Oil Spill

Variable	Age Group	Unexposed†	Exposed§	P Value
WBC ($\times 10^3$ per μL)	<40 years	6.5 \pm 1.9	6.6 \pm 1.7	.461†
	≥ 40 years	6.5 \pm 1.9	7.5 \pm 2.0	.007**
Platelets ($\times 10^3$ per μL)	<40 years	283.6 \pm 67.3	251.8 \pm 51.7	.006**
	≥ 40 years	249.5 \pm 60.7	252.5 \pm 52.4	.134†
Hemoglobin (g per dL)	<40 years	13.7 \pm 1.8	14.9 \pm 1.3	.000**
	≤ 40 years	13.6 \pm 1.6	14.9 \pm 1.4	.000**
Hematocrit (%)	≥ 40 years	40.2 \pm 5.9	44.7 \pm 3.4	.000**
	≥ 40 years	41.0 \pm 4.8	44.4 \pm 3.4	.000**
BUN (mg per dL)	<40 years	12.1 \pm 3.8	13.6 \pm 3.3	.039*
	≥ 40 years	15.7 \pm 5.6	14.5 \pm 2.8	.389†
Creatinine (mg per dL)	<40 years	0.8 \pm 0.2	1.1 \pm 0.2	.000**
	≥ 40 years	0.9 \pm 0.3	1.0 \pm 0.2	.044*
ALP (IU per L)	<40 years	65.0 \pm 21.7	74.9 \pm 20.9	.026*
	≥ 40 years	67.5 \pm 20.2	78.6 \pm 22.0	.001**
AST (IU per L)	<40 years	20.9 \pm 6.2	31.6 \pm 9.0	.050*
	≥ 40 years	23.5 \pm 4.7	30.0 \pm 5.8	.048*
ALT (IU per L)	<40 years	22.9 \pm 12.5	33.8 \pm 22.9	.050*
	≥ 40 years	26.0 \pm 13.8	40.1 \pm 20.7	.054*

ALP = alkaline phosphatase; ALT = alanine amino transferase; AST = aspartate amino transferase; BUN = blood urea nitrogen; WBC = white blood cells.

* $P = 0.05$.

** $P = 0.001$.

†Did not reach statistical significance.

‡Unexposed <40 years: $n = 28$; unexposed ≥ 40 years: $n = 102$.

§Exposed <40 years: $n = 73$; exposed ≥ 40 years: $n = 44$.

females, after exposure to the oil spill. Conversely, compared with females, males had a higher frequency of skin rash (61% vs 46%), chronic cough (55% vs 23%), and poor appetite (26% vs 8%).

The findings in **Table 7** reveal the frequency of somatic symptoms according to the age group among oil spill clean-up workers. Subjects in the age group of 20-29 years reported higher frequency of painful joints compared with

Table 4 Comparison of Hematologic and Hepatic Indices by Sex between Unexposed and Exposed Subjects to Oil Spill

Variable	Sex	Unexposed†	Exposed§	P Value
WBC ($\times 10^3$ per μL)	Male	6.7 \pm 2.2	6.8 \pm 1.8	.343†
	Female	6.4 \pm 1.7	7.5 \pm 2.2	.022*
Platelets ($\times 10^3$ per μL)	Male	234.6 \pm 71.5	248.7 \pm 52.0	.094†
	Female	287.0 \pm 74.5	278.7 \pm 42.5	.348†
Hemoglobin (g per dL)	Male	14.6 \pm 1.5	15.1 \pm 1.3	.022*
	Female	13.1 \pm 1.5	13.3 \pm 0.9	.331†
Hematocrit (%)	Male	42.9 \pm 5.4	45.1 \pm 3.2	.000**
	Female	39.8 \pm 4.5	40.5 \pm 2.3	.293†
BUN (mg per dL)	Male	17.7 \pm 9.3	13.4 \pm 3.4	.000**
	Female	14.1 \pm 5.1	13.0 \pm 3.6	.228†
Creatinine (mg per dL)	Male	1.1 \pm 0.3	1.1 \pm 0.2	.381†
	Female	0.8 \pm 0.2	0.8 \pm 0.2	.202†
ALP (IU per L)	Male	64.3 \pm 18.0	76.9 \pm 21.0	.018*
	Female	62.2 \pm 16.1	71.3 \pm 23.1	.048*
AST (IU per L)	Male	21.6 \pm 6.8	32.7 \pm 7.5	.051*
	Female	18.6 \pm 7.3	20.7 \pm 5.0	.046*
ALT (IU per L)	Male	27.4 \pm 13.5	38.2 \pm 27.0	.054*
	Female	17.6 \pm 12.0	26.1 \pm 24.6	.039*

ALP = alkaline phosphatase; ALT = alanine amino transferase; AST = aspartate amino transferase; BUN = blood urea nitrogen; WBC = white blood cells.

* $P = 0.05$.

** $P = 0.001$.

†Did not reach statistical significance.

‡Male unexposed: $n = 43$; female unexposed: $n = 87$.

§Male exposed: $n = 104$; female exposed: $n = 13$.

Table 5 Hematologic and Hepatic Changes by Age Group among Oil Spill Clean-up Workers

Variable	Age Group 20-29 Years (n = 31)	Age Group 30-39 Years (n = 42)	Age Over 40 Years (n = 44)	P Value†	P Value§	P Value¶
WBC ($\times 10^3$ per μ L)	6.2 \pm 1.6	6.8 \pm 1.8	7.5 \pm 2.0	.02*	.001**	.08†
Hemoglobin (g per dL)	15.0 \pm 1.3	14.8 \pm 1.3	14.9 \pm 1.4	.5†	.3†	.3†
Hematocrit (%)	45.0 \pm 3.5	44.4 \pm 3.3	44.4 \pm 3.4	.5†	.2†	.5†
Platelets ($\times 10^3$ per μ L)	245.2 \pm 50.1	256.6 \pm 55.8	252.5 \pm 52.9	.1†	.3†	.4†
BUN (mg per dL)	13.1 \pm 3.2	13.4 \pm 3.7	13.7 \pm 3.3	.1†	.2†	.5†
Creatinine (mg per dL)	1.0 \pm 0.2	1.1 \pm 0.2	0.97 \pm 0.2	.03*	.2†	.00**
β -2-microglobulin (mg per L)	76.1 \pm 21.4	74.1 \pm 20.5	78.6 \pm 22.1	.3†	.3†	.1†
ALP (IU per L)	29.1 \pm 29.0	33.4 \pm 31	30.0 \pm 21.4	.4†	.4†	.4†
AST (IU per L)	32.7 \pm 32.1	34.5 \pm 18.5	37.6 \pm 29.9	.5†	.3†	.2†
ALT (IU per L)	1.3 \pm 0.3	1.3 \pm 0.4	1.4 \pm 0.3	.5†	.02*	.05*
Urinary Phenol (mg per L)	8.7 \pm 9.5	7.6 \pm 9.9	5.3 \pm 5.9	.47†	.03*	.09†

ALP = alkaline phosphatase; ALT = alanine amino transferase; AST = aspartate amino transferase; BUN = blood urea nitrogen; WBC = white blood cells.

*P = 0.05.

**P = 0.001.

†Did not reach statistical significance.

‡Differences between age group of 20-29 years and 30-39 years.

§Differences between age group of 20-29 years and \geq 40 years.

¶Differences between age group of 30-39 years and \geq 40 years.

those in the 30-39-year or over-40-year age groups (65% vs 38% vs 48%). Similarly, the frequency of night sweats was higher in the 20-29-year age group compared with those in the 30-39-year or over-40-year age groups (52% vs 36% vs 39%). Conversely, subjects in the age group of 20-29 years reported the lowest frequency of chronic cough compared with those in the 30-39-year or over-40-year age groups (39% vs 52% vs 59%). The occurrence of other symptoms appeared to be similar among the 3 age groups.

DISCUSSION

Despite numerous oil spills that have affected the coastal populations, there have been very few studies that evaluated the health effects of these oil spills. Therefore, in this retrospective study we evaluated the health effects of the *Deepwater Horizon* oil spill and dispersant use in a cohort of oil spill clean-up workers. To our knowledge, no previous study has explored the effects of the oil spill specifically assessing the hematological and hepatic functions in oil spill clean-up workers.

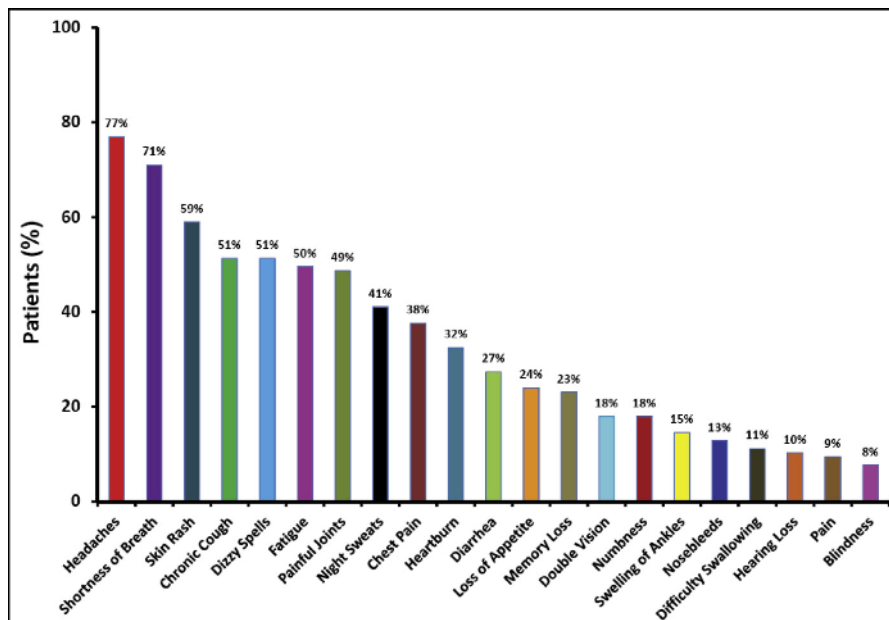


Figure 2 Major somatic symptoms experienced by the oil spill clean-up workers following exposure to oil spill and dispersant.

Table 6 Frequency of Somatic Symptoms by Sex among Oil Spill Clean-up Workers

Variable	Male (n = 104)	Female (n = 13)
Headaches	78 (75%)	12 (92%)
Shortness of breath	74 (71%)	9 (69%)
Skin rash	63 (61%)	6 (46%)
Chronic cough	57 (55%)	3 (23%)
Fatigue	50 (48%)	8 (62%)
Painful joints	48 (46%)	9 (69%)
Chest pain	40 (38%)	4 (31%)
Night sweats	40 (38%)	8 (62%)
Heartburn	33 (32%)	5 (38%)
ringing in ears	33 (32%)	9 (69%)
Diarrhea	27 (26%)	5 (38%)
Poor appetite	27 (26%)	1 (8%)
Memory loss	21 (20%)	6 (46%)

The results of this study indicate that oil spill exposure appears to play a role in the development of hematologic and hepatic toxicity. While WBC levels remained similar, platelet levels were significantly decreased in oil spill-exposed subjects compared with the unexposed subjects. Similarly, the BUN and creatinine levels were significantly decreased in oil spill-exposed subjects compared with the unexposed subjects. Conversely, the hemoglobin and hematocrit levels were significantly increased among oil spill-exposed subjects compared with the unexposed subjects. Although the oil spill-exposed subjects had significant differences in various hematological and hepatic indices, our results indicate that exposure to the oil spill did not uniformly affect subjects who participated in oil spill clean-up activities.

The levels of ALP, AST, and ALT were increased among subjects exposed to the oil spill compared with those of the

Table 7 Frequency of Somatic Symptoms by Age Group among Oil Spill Clean-up Workers

Symptoms	Age Group	Age Group	Age Over
	20-29 Years (n = 31)	30-39 Years (n = 42)	40 Years (n = 44)
Headaches	25 (81%)	33 (79%)	32 (73%)
Painful joints	20 (65%)	16 (38%)	21 (48%)
Skin rash	19 (61%)	23 (55%)	27 (61%)
Shortness of breath	18 (58%)	34 (81%)	31 (70%)
Night sweats	16 (52%)	15 (36%)	17 (39%)
Fatigue	15 (48%)	20 (48%)	23 (52%)
Dizzy spells	15 (48%)	24 (57%)	21 (48%)
Chest pain	13 (42%)	20 (48%)	11 (25%)
Chronic cough	12 (39%)	22 (52%)	26 (59%)
Heartburn	11(35%)	13 (31%)	14 (32%)
ringing in ears	11(35%)	13 (31%)	18 (41%)
Poor appetite	8 (26%)	11 (26%)	9 (20%)
Memory loss	8 (26%)	5 (12%)	14 (32%)
Diarrhea	7 (23%)	13 (31%)	12 (27%)

unexposed subjects. These serum enzyme levels have been considered indicators of hepatic damage. Phosphatases, amino transferases, and dehydrogenases play critical roles in biological processes. Specifically, these enzymes are involved in detoxification, metabolism, and biosynthesis of energetic macromolecules that are important for different essential functions. Alterations in the levels of these enzymes result in biochemical impairment and lesions in the tissue and cellular function. Thus, these enzymes are considered as specific markers for hepatic dysfunction and damage.²⁴ Similar increased levels of these enzymes have been reported in clinical and preclinical studies after benzene exposure.²⁵⁻²⁷ The increase in the levels of these hepatic enzymes in the serum may be due to impairment in the function of hepatic tissues following exposure to toxic chemicals from an oil spill, such as benzene, thereby allowing liberation of these enzymes into the circulation. Furthermore, the serum ALP is often used as a marker to measure hepatic and biliary tract function.^{28,29} The increase in the serum levels of ALP observed in this study suggest the possibility of biliary tract dysfunction in oil spill-exposed subjects.

Traditionally, the excretion of phenol in the urine has been used as an index of benzene exposure, which is a principal component of crude oil.³⁰⁻³² In this study, we found that there were significant amounts of phenol (7.0 mg/L) in the urine of the oil spill-exposed subjects. However, in healthy subjects (not exposed to oil spill), only traceable or an undetectable amount of phenol is expected in the urine. Thus, these findings suggest that the subjects involved in the oil spill clean-up activity were inherently exposed to benzene.

The health complaints reported by those involved in the oil spill clean-up operations are consistent with the previously reported studies of other major oil spills.^{6,7,9,33} However, the prevalence of symptoms appears to be higher in the present study compared with the earlier findings of other investigators. Lyons et al.⁶ found that headache, shortness of breath, fatigue, cough, skin rash, and diarrhea occurred more frequently in subjects exposed to the Sea Empress oil spill. Similarly, Morita et al.³³ observed headache, low back pain, leg pain, and jiggling of vision as major symptoms in subjects engaged in the clean-up activity of the Nakhodka oil spill. In the present study, the major symptoms observed were headache, shortness of breath, skin rash, cough, and fatigue in subjects involved in oil spill clean-up operations.

To understand the health consequences of oil spill exposure, a subgroup analysis was performed in subjects who participated in the oil spill clean-up activity. Specifically, the consequences of oil spill exposure were examined according to sex and age by subgroup analysis. Although the number of female clean-up workers was limited, we found no significant differences in the WBC count, BUN, beta-2-microglobulin, ALP, AST, ALT, and urinary phenol between the male and female subjects exposed to the oil spill. However, the platelet counts were significantly lower

in the male subjects compared with the female subjects exposed to the oil spill. Age appears to have minimal impact on various hematological and hepatic parameters in subjects exposed to the oil spill.

There were certain differences observed in the frequency of somatic symptoms between the male and female subjects involved in the oil spill clean-up activity. Compared with males, females experienced a higher rate of headaches, painful joints, fatigue, night sweats, and diarrhea following the exposure to the oil spill. Similarly, the age of the subjects appears to have a considerable impact on the frequency of somatic symptoms after the oil spill exposure.

Study Limitations

As with any cross-sectional study and most studies of oil spill disasters, our results should be considered in light of the following limitations. Foremost among these limitations is our lack of predisaster health data on the subjects involved in the clean-up activities. In addition, the demographic characteristics such as sex and age were significantly different between the exposed and unexposed groups and may have impacted the observed findings. Specifically, the median age of unexposed subjects was significantly higher when compared with the exposed group. Furthermore, data on somatic symptoms were self-reported by the oil spill-exposed group and may have influenced the findings of the study. Also, because the clinical outcomes were measured at one time point after the oil spill exposure, it was difficult to infer a causality using such a study design. Moreover, the effect of the oil dispersant may have contributed to the current findings. The COREXIT used as a dispersant is currently banned in the United Kingdom because of its potential health risks to clean-up workers.⁵ Nonetheless, this study has shed light on the health effects of oil spill exposure and dispersant use in subjects participating in the clean-up activities.

CONCLUSION

The results of this study indicate that human exposure to the oil spill has a potential to induce both hematological and hepatic toxicity. The hematological alterations include depletion of platelets, decreased BUN, and increased creatinine in subjects exposed to a crude oil spill. In addition, headache, shortness of breath, skin rash, cough, fatigue, painful joints, and chest pain occurred more frequently in oil spill clean-up workers. However, additional long-term follow-up studies are required to understand the clinical significance of the oil spill exposure.

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References

1. Urum K, Grigson S, Pekdemir T, McMenamy S. A comparison of the efficiency of different surfactants for removal of crude oil from contaminated soils. *Chemosphere*. 2006;62:1403-1410.
2. Solomon GM, Janssen S. Health effects of the Gulf oil spill. *JAMA*. 2010;304:1118-1119.
3. Merhi ZO. Gulf Coast oil disaster: impact on human reproduction. *Fertil Steril*. 2010;94:1575-1577.
4. Goldstein BD, Osofsky HJ, Lichtveld MY. The Gulf oil spill. *N Engl J Med*. 2011;364:1334-1348.
5. Diaz JH. The legacy of the Gulf oil spill: analyzing acute public health effects and predicting chronic ones in Louisiana. *Am J Disaster Med*. 2011;6:5-22.
6. Lyons RA, Temple JM, Evans D, Fone DL, Palmer SR. Acute health effects of the Sea Empress oil spill. *J Epidemiol Community Health*. 1999;53:306-310.
7. Cheong HK, Ha M, Lee JS, et al. Hebei spirit oil spill exposure and subjective symptoms in residents participating in clean-up activities. *Environ Health Toxicol*. 2011;26:e2011007.
8. Carrasco JM, Lope V, Perez-Gomez B, et al. Association between health information, use of protective devices and occurrence of acute health problems in the Prestige oil spill clean-up in Asturias and Cantabria (Spain): a cross-sectional study. *BMC Public Health*. 2006;6:1.
9. Janjua NZ, Kadir MM, Lutfi S, Tipre M, Sathiakumar N. Tasman spirit oil spill in Pakistan: Research response and lessons learned. *Am J Ind Med*. 2012;56:124-131.
10. Gill DA, Picou JS, Ritchie LA. The Exxon Valdez and BP oil spills: a comparison of initial social and psychological impacts. *Am Behav Sci*. 2012;56:3-23.
11. Repanich J. The Deepwater Horizon spill by the numbers. Available at: <http://www.popularmechanics.com/science/energy/coal-oil-gas/bp-oil-spill-statistics>. Accessed May 22, 2013.
12. Biello D. Is using dispersants on the BP Gulf oil spill fighting pollution with pollution? *Sci Am*. 2010;June 18:22.
13. Sandler DP, Kwok RK, Engel LS, Parks C, London SJ, Miller AK. GuLF Study: gulf long-term follow-up study. Available at: http://www.niehs.nih.gov/research/programs/gulfspill/gulfstudy/backgrounddocuments/gulf_study_protocol_7092012.pdf. Accessed May 22, 2013.
14. Aguilera F, Mendez J, Pasaro E, Laffon B. Review on the effects of exposure to spilled oils on human health. *J Appl Toxicol*. 2010;30:291-301.
15. Bosch X. Exposure to oil spill has detrimental effect on clean-up workers' health. *Lancet*. 2003;361:147.
16. Murray JS. The effects of the gulf oil spill on children. *J Spec Pediatr Nurs*. 2011;16:70-74.
17. Costantini AS, Benvenuti A, Vineis P, et al. Risk of leukemia and multiple myeloma associated with exposure to benzene and other organic solvents: evidence from the Italian Multicenter Case-control study. *Am J Ind Med*. 2008;51:803-811.
18. Travis LB, Li CY, Zhang ZN, et al. Hematopoietic malignancies and related disorders among benzene-exposed workers in China. *Leuk Lymphoma*. 1994;14:91-102.
19. Hayes RB, Yin S, Rothman N, et al. Benzene and lymphohematopoietic malignancies in China. *J Toxicol Environ Health A*. 2000;61:419-432.
20. Bloemen LJ, Youk A, Bradley TD, Bodner KM, Marsh G. Lymphohematopoietic cancer risk among chemical workers exposed to benzene. *Occup Environ Med*. 2004;61:270-274.
21. Khalade A, Jaakkola MS, Pukkala E, Jaakkola JJ. Exposure to benzene at work and the risk of leukemia: a systematic review and meta-analysis. *Environ Health*. 2010;9:31.
22. McHale CM, Zhang L, Smith MT. Current understanding of the mechanism of benzene-induced leukemia in humans: implications for risk assessment. *Carcinogenesis*. 2012;33:240-252.
23. Reardon S. Gulf oil spill. Ten months after Deepwater Horizon, picking up the remnants of health data. *Science*. 2011;331:1252.
24. Khan IA, Reddy BV, Mahboob M, Rahman MF, Jamil K. Effects of phosphorothionate on the reproductive system of male rats. *J Environ Sci Health Part B*. 2001;36:445-456.

25. Shamy MY, el Gazzar RM, el Sayed MA, Attia AM. Study of some biochemical changes among workers occupationally exposed to phenol, alone or in combination with other organic solvents. *Ind Health*. 1994;32:207-214.
26. Dere E, Ari F. Effect of Benzene on liver functions in rats (*Rattus norvegicus*). *Environ Monit Assess*. 2009;154:23-27.
27. Dede EB, Kagbo HD. A study on the acute toxicological effect of commercial diesel fuel in Nigeria in rats (*Rattus rattus*) using hematological parameters. *J Appl Sci Environ Manage*. 2002;6:84-86.
28. Polin SG, Spellberg MA, Teitelman L, Okumura M. The origin of elevation of serum alkaline phosphatase in hepatic disease. An experimental study. *Gastroenterology*. 1962;42:431-438.
29. Sebesta DG, Bradshaw FJ, Prockop DJ. Source of the elevated serum alkaline phosphatase activity in biliary obstruction: studies utilizing isolated liver perfusion. *Gastroenterology*. 1964;47:166-170.
30. Docter HJ, Zielhuis RL. Phenol excretion as a measure of benzene exposure. *Ann Occup Hyg*. 1967;10:317-326.
31. Inoue O, Seiji K, Kasahara M, et al. Quantitative relation of urinary phenol levels to breathzone benzene concentrations: a factory survey. *Br J Ind Med*. 1986;43:692-697.
32. McDonald TA, Holland NT, Skibola C, Duramad P, Smith MT. Hypothesis: phenol and hydroquinone derived mainly from diet and gastrointestinal flora activity are causal factors in leukemia. *Leukemia*. 2001;15:10-20.
33. Morita A, Kusaka Y, Deguchi Y, et al. Acute health problems among the people engaged in the cleanup of the Nakhodka oil spill. *Environ Res*. 1999;81:185-194.